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Case Report

Use Of ADO II Device In A Large Aorto-Pulmonary Collateral-An Interesting Case

N. Awasthy^{a,*}, R. Garg^a, P. Khurana^b, S. Radhakrishnan^a^a Department of Pediatric Cardiology, Fortis Escorts Heart Institute, New Delhi, India^b Department of Radiology, Fortis Escorts Heart Institute, New Delhi, India

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ABSTRACT

A 11month old girl was diagnosed to have a large aorto pulmonary collateral during evaluation for respiratory distress and seizures. Echocardiographic evaluation showed a large collateral from the descending aorta to left lung with ventricular dysfunction (left ventricular ejection fraction of 40%). This was confirmed on computed tomography angiogram (CT) scan. The collateral was closed with Amplatzer Ductal Occluder II device under fluoroscopic guidance. To the best of our knowledge, this is the first reported case of large aorto-pulmonary collateral closed successfully with an Amplatzer Ductal Occluder II device.

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1. Introduction

Aorto-pulmonary collaterals are a source of alternative blood supply to the lungs in congenital heart disease with decreased pulmonary blood flow e.g., Tetralogy of Fallot (TOF), ventricular septal defect (VSD) with pulmonary atresia. These are also noted in premature neonates with bronchopulmonary disorders.^{1,2} These collaterals are usually small and rarely cause symptoms. They usually regress on their own after treatment of the primary disorder and rarely need intervention.³ We describe a case of large unusual aortopulmonary collateral presenting as bronchopneumonia that was closed successfully using Amplatzer Ductal Occluder II device.

2. Case report

Baby J is a 11 month old female, weighing 3.3 kg, delivered at term, without any perinatal complications with a birth weight of 2.25 kg. She had feeding difficulty with failure to thrive until 11 months of age when she developed respiratory distress and seizures. She was treated as a case of bronchopneumonia, meningitis with dilated cardiomyopathy. Her clinical examination was fairly unremarkable. Chest X-ray showed consolidation. Initial echocardiogram showed mild left ventricular dysfunction, dilated left ventricle, Left ventricular ejection fraction (LVEF) of 40% and abnormal channel from descending aorta to lungs. CT confirmed collateral malformation in left

* Corresponding author. Department of Pediatric Cardiology, Fortis Escorts Heart Institute, New Delhi, India.

E-mail address: n_awasthy@yahoo.com (N. Awasthy).<http://dx.doi.org/10.1016/j.ihj.2015.06.008>

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Fig. 1 – CT angiogram coronal view, maximum intensity projection shows a side branch from descending thoracic aorta forming a loop and supplying left lower lobe.

lower lobe with arterial supply from the descending thoracic aorta and venous drainage in left atrium (Fig. 1). Her work-up showed that she had hypocalcaemia, ionized calcium of 0.3 ml/l with decreased serum calcium of 4gm/dl. Her alkaline phosphate was increased. Her vitamin D levels were low with increased serum Parathyroid (PTH) levels. In view of hypocalcaemia she received vitamin D 6 lac IU IM and was started on IV calcium 150 mg/kg/day which was overlapped by oral calcium when the serum ionized calcium levels normalized.

At cardiac cath child was observed to have no significant step-up from superior vena cava (SVC) to pulmonary artery (PA). A large collateral arising from descending aorta at sixth thoracic vertebral level and coursing towards left, supplied left lung (lower lobe) (Fig. 2). The collateral was



Fig. 2 – Aortogram done in AP view showing the collateral (marked by arrow) from the descending aorta supplying the left lung field with cardiomegaly. Dao.: Descending aorta.



Fig. 3 – Aortogram done in AP view showing the collateral from the descending aorta supplying the left lung field occluded by the Amplatzer ductal occluder 2 with no residual flow. Dao.: Descending aorta.

closed with ADO II device (4/4 mm) deployed under fluoroscopic guidance. Post device deployment fluoroscopy revealed device in situ with tiny residual flow across (Fig. 3). Immediately after the procedure her left ventricular ejection fraction decreased to 25%. The child was discharged on oral supplementation of calcium. At 6 months follow-up, there was adequate weight gain. Echocardiogram showed normal LVEF (LVEF = 65%), no residual flow though the collateral and normal left ventricle end diastolic volumes.

3. Discussion

Aorto-pulmonary collaterals are considered innocuous lesions which rarely cause symptoms in normal heart. These may represent persistent, abnormally dilated arteries that connect the bronchial arteries to the pulmonary circulation secondary to external factors such as hypoxia, trauma, and inflammation.³ Our child was a term child without any history of asphyxia, so it is difficult to say as to what factors were operative in the present case.

The clinical course of our case was similar to that of a large post-tricuspid left-right shunt, presenting as congestive heart failure after decrease in neonatal pulmonary hypertension. There was no step-up from SVC to PA as shunt was beyond major pulmonary arteries. Since the aorto-pulmonary collateral was symptomatic, it needed closure. The options available were either simultaneous delivery of multiple coils or use of a vascular plug. For simultaneous delivery of multiple coils, a 7 French sheath was needed. However, in a small child with a weight of 3.3 kg, it would have been difficult without causing much damage to femoral artery so a ADO II device, was used as it could be delivered through a 4 French sheath and has high thrombogenic potential. Surgical option remain another possibility. Perrot et al reported a case of fistula between an

artery arising from the aorta and a pulmonary vein associated with absence of a branch of the pulmonary artery, who underwent left lobectomy.⁴ To the best of our knowledge ADOII device has never been deployed in a patient with isolated aorto-pulmonary collateral in the literature.

Ventricular function was 40% before the delivery of the device. With a large shunt the increased left ventricular volume (preload) masked true ventricular dysfunction in our patient. Once the collateral was closed the ventricular dysfunction was unmasked (LVEF = 25%). The cause of ventricular dysfunction in the present case was also associated hypocalcemia. With the treatment of hypocalcaemia, ventricular function recovered over a period of 6 months.

4. Conclusion

The present case highlights a rare case of large aortopulmonary collateral. This is the first case of ADOII device deployed in a patient with isolated aorto-pulmonary collateral. The case also re-emphasizes to look at associated causes of ventricular dysfunction, such as hypocalcaemia. Successful treatment of hypocalcaemia resulted in complete recovery in our case.

Conflicts of interest

The authors have none to declare.

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