Potential causes of flail TV in the neonate include congenital endocarditis, ischemia induced by premature ductal closure, maternal autoimmune disease, birth asphyxia, a thromboembolic event, or traumatic rupture during the birth process [1, 2, 5]. The anterior papillary muscle is typically injured because of its location within a watershed area and its requirement for perfusion during diastole when systemic right ventricular pressures are reached [2]. No signs or history of maternal autoimmune disease, sepsis, or the use of prostaglandin synthase inhibitors were noted in any of the neonates in our series. There was no obvious indication of prenatal ischemia or inhibitors were noted in any of the neonates in our series. There was no obvious indication of prenatal ischemia or report of significant trauma or asphyxia at birth. Given that the etiology of flail TV is often unknown, an understanding of the early signs of flail TV, recognition of echocardiographic findings, and knowledge of stabilization methods is extremely important in the management of this rare but potentially fatal condition.

References


One-Stage Midline Unifocalization and Anatomic Correction of Corrected Transposition With Pulmonary Atresia and Absence of Central Pulmonary Arteries

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A concomitant double-switch procedure and unifocalization were performed in a child with congenital corrected transposition of the great arteries, ventricular septal defect, pulmonary atresia, absence of central pulmonary arteries and major aortopulmonary collateral arteries. Predisharge echocardiography showed no residual shunts with laminar flow in baffles and outflow tracts. Follow-up computed tomographic angiography revealed good neo—main pulmonary artery confluence with satisfactory pulmonary blood flow.

The advantages of anatomic repair of congenitally corrected transposition of the great arteries (CCTGA) over conventional repair are well described in the literature, as are also those of single-stage unifocalization of the major aortopulmonary collateral arteries (MAPCAS) in patients with absence of central pulmonary arteries [1, 2]. The operation described here brings together both these sets of advantages and, in our opinion, despite its complexity, provides the ideal result in this complicated association.

To the best of our knowledge, there has been no report so far of single-stage repair of CCTGA, ventricular septal defect (VSD), and pulmonary atresia (PA) with MAPCAS.

A 1-year-old boy with a history of recurrent respiratory tract infections and failure to thrive was referred to us for further evaluation and management. Transthoracic echocardiography revealed dextrocardia, atioventricular discordance, and aorta arising from the right ventricle (RV). A large inlet VSD was present, which seemed nonroutable to an anteriorly placed aorta. Single-outlet pulmonary atresia was present. The child had MAPCAS, which were further profiled on computed tomographic (CT) pulmonary angiography. CT confirmed atresia of the main pulmonary artery and absence of the central branch pulmonary arteries (Figs 1, 2). The child was scheduled for elective complete intracardiac repair.

A median sternotomy was used for access. The thymus was excised, and a pericardial patch was obtained and treated with 6% gluteraldehyde. The collaterals were dissected up to their lobar branches and looped. Cannulation of the aorta, inferior vena cava, and right atrium was done, and cardiopulmonary bypass (CPB) was commenced. The patient was cooled to 26°C. The collateral flow was restricted by the application of occlusive clips. The collaterals were sequentially ligated at their aortic ends by use of a transfixation Prolene suture in addition to silk ties. The stenosed collateral supplying the right lower lobe was patched with untreated autologous pericardium. The superior and inferior collaterals draining the left lung were anastomosed to each other, as were the collaterals on the right side to create a neo—left pulmonary artery and a neo—right pulmonary artery. The right and left neo—pulmonary arteries so created were joined across the midline to create neo—pulmonary artery confluence by suturing their posterior walls together.

The heart was fibrillated at this stage, and the left atrium was vented through the right superior pulmonary

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An 18-mm handmade bovine pericardial valved conduit was sutured distally to the neo-pulmonary artery confluence (Fig 3) and then displaced in the left pleural cavity.

At this stage, the right atrial cannula was removed, and another cannula was inserted in the superior vena cava. The patient was further cooled to 16°C. The aorta was cross-clamped and cardioplegia was given (Del Nido’s solution, 20 mL/kg). The inferior vena cava and the superior vena cava were snared. The right atrium was opened. As viewed through the retracted mitral valve leaflets, the VSD was assessed to be routable to the aorta. A transverse right ventriculotomy was done, and the VSD and aortic valve anatomy were further examined. The VSD was baffled to the aorta with a bovine pericardial patch. Right ventricular muscle resection was done to avoid potential right ventricular outflow tract obstruction. This was followed by Senning’s procedure for atrial switch by use of the in-situ pericardial modification technique [3]. A short duration of total circulatory arrest was used during the creation of the pulmonary venous baffle. Rewarming was started, deairing was done, and the cross-clamp was released. The aorta was vented through the cardioplegia cannula site for left-sided deairing. The right atrium was closed. The proximal end of the right ventricular–pulmonary artery conduit was completed on the beating heart. The patient was weaned from CPB at 34°C. The total CPB time was 209 minutes, and the cross-clamp time was 110 minutes.

Chest closure was done on the fourth postoperative day. Owing to failed extubation, a tracheostomy was done on the 18th postoperative day. The child was successfully decannulated on the 46th postoperative day. Echocardiography showed the VSD patch in situ and no residual shunts. Laminar flow was visualized in the pulmonary and systemic venous baffles. The flow in the left ventricular and right ventricular outflow tracts was laminar.
There was trace tricuspid regurgitation (maximum gradient 16) and mild MR. There was no aortic regurgitation or pulmonary regurgitation. Biventricular function was normal. Because the child was still oxygen dependent, a CT pulmonary angiogram was done. It confirmed these findings and additionally revealed residual stenosis in the middle part of the unifocalized collateral supplying the right lower lobe. Other collaterals were found to be normal in course and in caliber (Fig 4). The right innominate artery was seen to be causing a mild indentation of the trachea anteriorly. Left main bronchial compression between the proximal left pulmonary artery and the descending thoracic aorta was also seen, thus revealing the cause of the prolonged ventilation (Fig 5). However, we found the lumen to be adequate, and given that the child recovered, we decided against stenting the patient.

Comment

This case presents a few firsts in the management of this surgical pathologic condition. Double switch along with unifocalization is a technically demanding procedure. Longer cross-clamp times necessitate careful attention to myocardial protection. Periods of deep hypothermic circulatory arrest have been used by surgeons to supplement myocardial protection in addition to cardioplegia. We used ventricular fibrillatory arrest with continuous coronary perfusion for the unifocalization and a distal right ventricle–pulmonary artery conduit anastomosis to the neo–pulmonary artery; the ventricles were decompressed by use of inferior vena cava and right atrium cannulas during this time (32 minutes). We used Del Nido’s cardioplegia solution, which gives a relatively longer window time of 45 to 60 minutes. Our total cross-clamp time was 110 minutes, during which two doses of Del Nido’s cardioplegia solution were used. Also, the proximal end of the right ventricle–pulmonary artery conduit was anastomosed to the right ventricle on the beating heart, which actively helped in deairing the right side of the heart as well.

Conventionally, an inlet VSD is found to be nonroutable to the aorta and may preclude a Rastelli repair. However, in this case, because of the absence of the main pulmonary artery and central pulmonary arteries, and the presence of a malaligned septum, we deemed it routable. The alternate option of univentricular repair was a poor option because of the hypertensive aortopulmonary collaterals and was unlikely to be successful; hence, a long tunnel was created to route the left ventricle to the aorta. Dextrocardia may cause underdevelopment of the right atrium and increase the difficulty of achieving a satisfactory Senning baffle. Dextrocardia can also increase the risk of sternal compression of the right ventricle–pulmonary artery conduit by placing it in an immediate retrosternal location. We opened the left pleura completely and displaced the conduit in the left pleural cavity to avert this complication.

Postoperative echocardiography and CT scans revealed that the baffles were satisfactory and that there was no compression of the conduit. We are unaware of any case report so far wherein double switch and unifocalization for a patient with absence of the central pulmonary arteries have been done in a single stage. Our decision was based on the child’s age, the absence of central pulmonary arteries, and an adequate left ventricle. The size and mass dimensions of the left ventricle on preoperative echocardiography indicated that the left ventricle did not need retraining, and because the right ventricular (systemic) pressures were similar to the left ventricular...
pressure, we decided to perform a double-switch procedure at the same setting [4].

In view of heart failure, the risk of pulmonary hypertension, and progression of pulmonary vascular disease, a single-stage double-switch procedure with unifocalization is a satisfactory surgical treatment option in a patient with CCTGA, VSD, PA, and MAPCAs. It can result in a balanced circulation without heart failure or cyanosis, which could potentially occur if staged repairs were to be undertaken, resulting in inoperability toward complete repair [5].

References

Life-Threatening Hemorrhage During Removal of a Nuss Bar Associated With Sternal Erosion

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We present a case of life-threatening hemorrhage occurring during Nuss bar removal without obvious cardiac or major vascular injury. A 19-year-old woman with marfanoid features had undergone a Nuss procedure 3 years earlier for a pectus index of 7.2. A lateral chest radiograph revealed erosion of the upper bar into the sternum. During surgery, a 3.5-L blood loss occurred after removal of the eroded bar. This case provided many opportunities to improve preparedness for bar removal.


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Minimally invasive repair of pectus excavatum requires placement of one or more subternal bars to elevate the sternum to the normal anterior position. Removal of the bars is recommended 2 to 3 years after implantation. Bleeding during bar removal secondary to aortic, pulmonary, or cardiac injury has been previously reported [1–3]. We present a case of life-threatening hemorrhage during removal of a bar associated with sternal erosion.

A 19-year-old woman presented for elective removal of her Nuss pectus bars. She had undergone repair of pectus excavatum (Haller index, 7.5; Fig 1) 3 years earlier. Preoperatively she had symptoms of lightheadedness and exercise intolerance documented on treadmill testing. There was no family history of Marfan syndrome. Although not meeting the diagnostic criteria for Marfan syndrome, the patient exhibited some marfanoid features, including minor skin stria on the back, arachnodactyly, and positive wrist and thumb signs as well as mild scoliosis. Her arm span was less than her height. She had no hypermobile joints. The remainder of her examination was unremarkable.

Minimally invasive repair with complete thoracoscopic transmediastinal dissection and placement of two 11-inch pectus bars (Biomet, Jacksonville, FL) was performed. The bars were secured with FiberWire (Arthrex, Inc, Naples, FL). She had an uneventful recovery. At her 3-year follow-up, she had excellent correction with resolution of all preoperative symptoms. A preoperative chest radiograph confirmed good bar position; however, the superior bar was noted to have migrated anteriorly through the posterior sternal table on the lateral view (Fig 2).

Bar removal was scheduled in the main operating suite. She was positioned supine with arms out. The prior incisions were opened bilaterally. Both bars had moderate heterotopic ossification as well as fibrin sheath encasement. Both bars were freed of all bony fragments and completely straightened. The lower bar was removed first without difficulty. The upper bar initially showed significant resistance to removal. With an additional effort, the straightened bar was removed. Immediately after the bar’s exit, a rapid flow of nonpulsatile bright red...