In addition to our first case, we have noticed four more cases of acute swelling of the scrotum with subsequent abandonment of the EVH technique to avoid the complications as previously described. The patients were followed-up postoperatively on weeks 6 and 12, and no complications were noted.

In conclusion, patients with a previous history of indirect inguinal hernia or repaired hernia warrant careful assessment. The increased possibility of intraoperative testicular vascular impediment, necrosis, and subsequent testicular loss may warrant the use of minimally invasive rather than endoscopic vein harvesting during CABG. Further work is required in this area to prevent this potentially serious complication.

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Anomalous Left Coronary Artery From Pulmonary Artery With Mitral Stenosis

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The usual presentation of anomalous left coronary artery from pulmonary artery is severe left-sided heart failure and mitral valve insufficiency presenting during the first months of life. The manifestations of left heart failure may be masked if pulmonary artery pressure remains high. We believe this is a rarest of rare case of anomalous left coronary artery from pulmonary artery with severe mitral stenosis and pulmonary hypertension in which pulmonary hypertension, along with good collateral circulation helped to preserve left ventricular function.

(Ann Thorac Surg 2011;92:735–7)

Origin of anomalous left main coronary artery from pulmonary artery (ALCAPA) occurs once in every 300,000 live births accounting for 0.25% to 0.5% of congenital heart disease cases [1]. The majority of these cases die in infancy, if not corrected in time. Most of the untreated patients who reach adulthood develop left ventricular dysfunction, mitral regurgitation, and sometimes myocardial infarction. We present a rare case of an adolescent girl with ALCAPA and mitral stenosis with preserved left ventricular function.

A 20-year-old woman was referred to our institute with a history of dyspnea for 4 months. There was pallor with raised jugular venous pressure and hepatomegaly. Apart from findings of severe mitral stenosis and pulmonary hypertension, the patient had a continuous murmur in the left third and fourth intercostal spaces in the parasternal area. There was history of episodic migrating joint pain with fever that started at 5 years of age, suggestive of rheumatic fever. With this background, our provisional diagnosis was rheumatic mitral stenosis with pulmonary hypertension, but there was no obvious explanation for continuous murmur.

Her electrocardiogram showed sinus rhythm with right axis deviation; there were no ST-segment elevation changes or abnormal Q waves in any leads. Transthoracic echocardiography showed severe mitral valve stenosis (orifice area, 0.8 cm^2; mean gradient, 18 mm Hg) with mild subvalvular fusion, pliable mitral valve leaflets without any calcification. The echocardiographic score of the mitral valve was 7/16 (mobility, 2; subvalvular thickening, 2; leaflets thickening, 3; calcification, 0; total score, 7/16), suggesting suitability for an open mitral valvotomy at surgery [2]. There was no mitral regurgitation. The tricuspid valve was morphologically normal. There was moderate tricuspid valve regurgitation with a gradient of 48 mm Hg most probably due to pulmonary artery hypertension. Surprisingly, the right coronary artery was aneurysmally dilated. Her left ventricular ejection fraction was 65%.

Because there was high suspicion of ALCAPA, a coronary angiogram and right heart catheterization was done. Selective right coronary injection showed hugely dilated right coronary artery filling left coronary artery through collaterals (Fig 1). Left coronary artery was eventually draining into pulmonary artery. Aortic root injection did not show left coronary artery arising from the left aortic sinus. Direct pulmonary artery pressure was 52/36 mm Hg with a mean of 41 mm Hg. Thus, the diagnosis of ALCAPA was confirmed.

Accepted for publication Feb 8, 2011.

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0003-4975/$36.00
doi:10.1016/j.athoracsur.2011.02.022

Ann Thorac Surg
2011;92:735–7

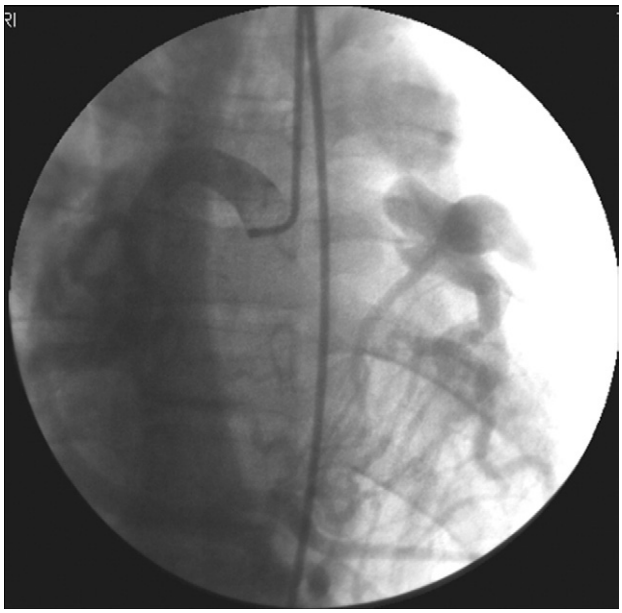

Fig 1. Angiogram shows dilated right coronary system and filling of left coronary system and pulmonary artery.

During surgery, a median sternotomy was performed. The right coronary artery was hugely dilated (Fig 2). Left main coronary artery was arising from the main pulmonary artery. Cardiopulmonary bypass was established using aorto-bicaval cannulation. The first dose of cardioplegia was delivered through aortic root after occluding both branches of pulmonary artery to prevent steal through ALCAPA. Subsequent dose of cardioplegia was delivered through the coronary sinus. There was rheumatic mitral valve stenosis with thickened leaflets and fused commissures (Fig 3). For mitral stenosis, a standard open commissurotomy was performed with splitting of the commissural chordae. Because the tricuspid valve was morphologically

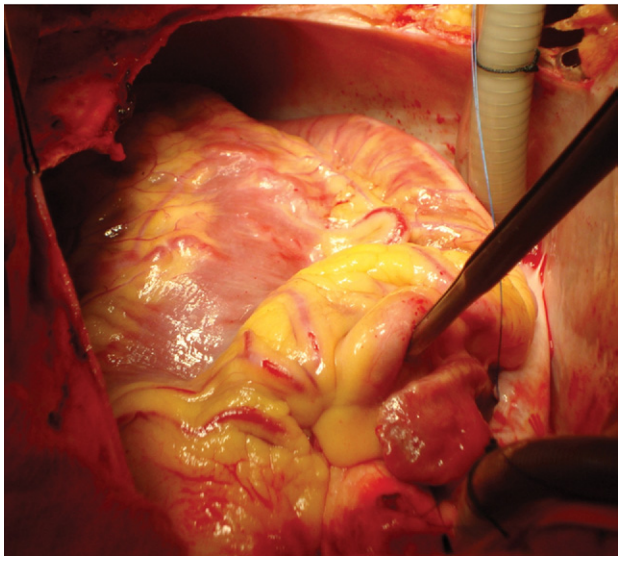

Fig 2. Intraoperative picture shows dilated right coronary artery.

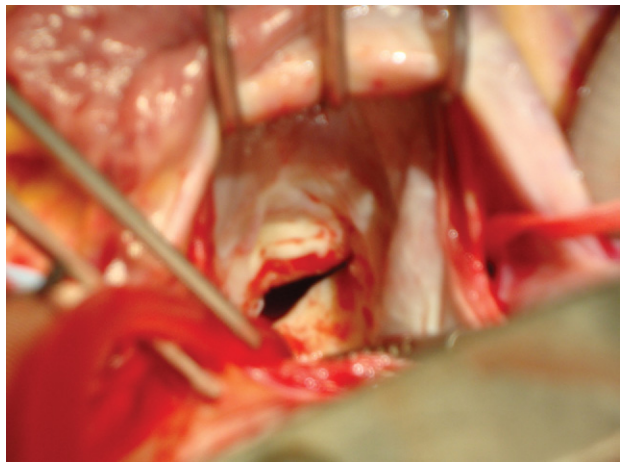

Fig 3. Intraoperative picture shows the stenosed mitral valve.

normal and the regurgitation was functional, we decided not to intervene on the tricuspid valve. A button of the aorta was removed adjacent to the pulmonary artery to create an aortopulmonary window. An aortopulmonary tunnel was created using a free segment of the anterior wall of the main pulmonary artery, which was stitched to the back wall of the main pulmonary artery diverting aortic blood into the left coronary artery. An anterior wall defect of the pulmonary artery was covered with a pericardial patch using the Takeuchi repair. The patient was weaned off cardiopulmonary bypass with minimal support. An open mitral valvotomy in a young girl with pliable valve leaflets obviated the need for anticoagulation and associated complications of valve replacement during future possible pregnancy [3].

Postoperatively, the patient made an uneventful recovery. Echocardiography on day 10 showed normal left ventricular function without any mitral regurgitation. Her mean mitral valve gradient was 5 mm Hg with valve area of 1.8 cm^2. There was mild tricuspid regurgitation and the gradient went from 48 mm Hg preoperatively down to 28 mm Hg postoperatively, most likely as an effect of reduction in right ventricular systolic pressures. The estimated postoperative mean pulmonary artery pressure was 24 mm Hg by Mahan's equation [4]. Good flow was noted in the left coronary artery from the aortopulmonary tunnel with no leak in the aortopulmonary baffle.

Comment

The clinical expression of ALCAPA results from morphologic functional alterations in pulmonary circulation that occurs after birth. In the common form of ALCAPA, a child presents at the age of 1 to 2 months. Because the collateral circulation is poorly developed in infants, as soon as the pulmonary pressure drops in the neonatal period, the left-to-right shunt across the coronary circulation increases, and coronary steal and myocardial ischemia supervene. This causes congestive heart failure, which is worsened by mitral regurgitation. Mitral regurgitation occurs secondary to papillary muscle ischemia or permanent fibrosis and left ventricular dilatation [5].

In the adult type of ALCAPA, collateral circulation between the right and left coronary systems ensues, and the left coronary artery flow reverses and enters the pulmonary trunk due to the low pulmonary arterial pressure. Consequently, the myocardium remains inadequately perfused. There is an estimated 80% to 90% incidence of sudden death at a mean age of 35 years in this group [6].

If by some reason the pulmonary hypertension does not come down, as in infants with large ventricular septal defects or large patent ductus arteriosus, pulmonary artery perfuses the anomalous coronary artery and myocardial ischemia does not manifest. The importance of such findings lies in the fact that ventricular septal defect closure or patent ductus arteriosus ligation in such cases would reduce the flow in the left coronary artery from thee pulmonary artery, and unmask ALCAPA [7]. Some of these patients have an origin stenosis of the left coronary artery, limiting the steal and increasing myocardial perfusion pressure, and thus maintaining left ventricular pressure [8].

Association of ALCAPA with mitral regurgitation is a well-documented entity, but ALCAPA presenting with mitral stenosis is a rarest of rare associations. Although the patient had severe mitral stenosis with high pulmonary arterial pressure, along with good collaterals between the right and left coronary arteries, her ventricular function was preserved. Her presentation was mainly symptoms of pulmonary venous hypertension. Had we missed the ALCAPA and her mitral stenosis had been relieved, then myocardial ischemia or infarction would have manifested. The clue to the diagnosis was a good clinical examination and high index of suspicion.

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Giant Tumor of the Right Atrium in Infancy

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Cardiac hemangiomas can occur at any age, but they are extremely rare when they occur early after birth. We describe the case of a 5-month-old infant who had a giant right atrial capillary hemangioma associated with massive pericardial effusion. The tumor was incidentally diagnosed during routine clinical follow-up. The hemangioma was removed successfully under cardiopulmonary bypass, and the patient's postoperative course was uneventful. The occurrence of giant capillary hemangioma in infancy represents an unusual event in the relevant literature. Herein, we discuss the clinical features and surgical management of this rare primary tumor of infancy.

(Ann Thorac Surg 2011;92:737–40)

FEATURE ARTICLES

Primary cardiac tumors are extremely rare, with an incidence of 0.001% to 0.03% in clinical postmortem series. Hemangiomas account for less than 3.5% of all cardiac tumors, and the reports of these in infancy have been very rare [1]. The availability of diagnostic modalities and close perinatal follow-up has facilitated the diagnosis and surgical treatment of such lesions in the perinatal period. Clinical presentation and associated symptoms depend on the size and location of the mass, but the tumor may have an unpredictable course and can be fatal if undiagnosed. A huge intracardiac hemangioma associated with massive pericardial effusion is an unusual situation in infancy.

A 5-month-old female infant presented to the outpatient clinic with a history of infantile colic and tachypnea. She was suffering from an intractable colic for 2 months, and her previous treatment was unsuccessful to relieve the symptoms. The patient was afebrile, had no visible cutaneousvascular lesions, or any signs of congestive heart failure. On admission, her blood pressure and heart rate were 80/52 mm Hg and 156 beats per minute, respectively. Cardiac auscultation revealed diminished heart sounds. An electrocardiogram showed sinus tachycardia and excluded cardiac ischemia or rhythm disturbance. The hematocrit was 32.8% and the C-reactive protein was 104 mg/dL. The other levels of blood chemistry and urine

Accepted for publication Feb 7, 2011.

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0003-4975/$36.00
doi:10.1016/j.athoracsur.2011.02.026