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Coronary Steal Syndrome After Coronary Artery Bypass for Anomalous Aortic Origin of a Coronary Artery
Benjamin A. Youdelman, Glenn J. Pelletier, C. Igor Mesia and Marshall L. Jacobs

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the patient was brought to the intensive care unit. The postoperative creatine kinase-MB fraction was 11.04 ng/mL, and the patient was discharged from the hospital on postoperative day 4. A follow-up computed tomographic scan with contrast at 1 month confirmed that there was no flow into the aneurysm and that it was stable in size. At 1-year follow-up the patient is doing well and has no complaints.

Comment

Several possible treatments were considered for this patient including coil embolization, median sternotomy to ligate the graft, a small lateral thoracotomy to dissect the vein graft from the sternum, followed by a median sternotomy, as well as our innovative approach to patch the ostia of the graft from the sternum, followed by a median sternotomy, the graft, a small lateral thoracotomy to dissect the vein graft aneurysm while preserving the other grafts.

Aneurysms of the vein grafts are known to occur, but are usually treated conservatively. We believe that replacement of patent vein grafts greater than 5 mm in diameter should be considered an indication for surgical intervention. Aneurysms larger than 1 cm are considered to be at increased risk for rupture and require surgical intervention 

References


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Anomalous aortic origin of a coronary artery found in a symptomatic 9-year-old boy was initially treated with coronary artery bypass grafting using a left internal mammary artery anastomoses to the left anterior descending coronary artery, but resulted in coronary ischemia, likely from a steal phenomenon. Subsequent transection of the proximal left internal mammary artery with anastomosis to the ascending aorta, and coronary ostial enlargement, resulted in a durable treatment. We recommend caution in choosing coronary artery bypass grafting using a left internal mammary artery pedicle graft for the treatment of anomalous aortic origin of a coronary artery.

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Anomalous aortic origin of a coronary artery (AAOCA) has been associated with signs and symptoms of myocardial ischemia, and may be a cause for sudden death in children and young adults [1, 2].

Anatomic variations include the left main coronary artery arising from the right sinus of Valsalva, and the right coronary artery arising from the left sinus. Theories to explain the mechanism for myocardial ischemia are varied. They include stenosis or distortion of the coronary ostium, compression of an intramural coronary segment subjected to high aortic wall tension, kinking at the acute angle of the proximal anomalous coronary during hyperdynamic conditions, or compression of the coronary artery between the aortic and pulmonary roots during effort-related expansion of these vessels [3]. With the pathophysiology imprecise, it follows that the surgical approach to AAOCA has been varied.

Numerous operations have been proposed to treat this condition including coronary artery bypass surgery (CABG) with arterial or venous conduits, reimplantation of the coronary artery into the appropriate sinus of Valsalva [4], and coronary ostial enlargement with or without unroofing of an intramural segment [5]. Currently there is no consensus for surgical treatment.

Risk of sudden death in a patient with AAOCA is believed to be reduced by creating durable, unobstructed coronary artery flow. The recommendation for surgery in adolescents or young adults is generally made when signs or symptoms of myocardial ischemia are present, but are less well-defined when the coronary anomaly is discovered incidentally in asymptomatic infants or young children.

We report a 9-year-old boy who presented with syncope preceded by palpitations, dizziness, and diaphoresis. Similar symptoms occurred 4 years earlier, and palpitations are reported with exercise.

His electocardiogram showed sinus arrhythmia, a prolonged Q-T interval of 461 msec, but no ischemia. He underwent echocardiography that revealed anomalous origin of the left coronary artery from the right coronary sinus of Valsalva. Computerized tomographic angiography and cardiac catheterization demonstrated the left coronary artery coursing between the aorta and pulmonary trunk. No intramural course was identified.

Coronary artery bypass grafting surgery using the left internal mammary artery (LIMA) pedicle graft connected to the left anterior descending coronary artery was performed. After separation from cardiopulmonary bypass, the hemodynamics were good. However, when the chest retractor was removed, diffuse ST segment elevation occurred and the patient had ventricular tachycardia. The graft was inspected and found to have a palpable pulse. Echocardiography showed normal cardiac function without segmental wall motion abnormalities. The patient was transported to the cardiac catheterization laboratory for emergent angiography.

Initial injection into the left subclavian artery showed poor filling of the LIMA graft; however, direct injection into the LIMA filled the entire coronary circulation. Injection into the right sinus of Valsalva uniformly filled both right and left coronary systems, but retrograde flow from the left anterior descending coronary artery through the LIMA and into left subclavian artery demonstrated a steal circuit diverting blood from the left anterior descending coronary artery territory (Fig 1; video 1 viewable at http://ats.ctsnetjournals.org/content/vol87/issue4/images/data/1292/DC1/youdelman1.mpg). The patient returned to the operating room for surgical revision.

Opening the chest widely again reduced the ischemic changes on electrocardiogram, but occlusion of the LIMA did not resolve them. Therefore, the attention was directed to the coronary artery origin.

The aortic root was explored and a single coronary ostium was identified. The right coronary artery arose from the ostium and traveled in its usual course. The left coronary artery exited from within the single ostium in an oblique fashion. No intramural course was identified. The obliquity of the left coronary origin seemed to create a point of coronary stenosis, and this opening was enlarged by incising it into the aortic media and then repairing it.

The patient was weaned from circulatory support with good hemodynamics and no ischemic changes on electrocardiogram. However, again with removal of the chest retractor, the ischemic phenomenon recurred even with the LIMA graft occluded.

Two videos of this procedure can be viewed on the Internet at: http://ats.ctsnetjournals.org/content/vol87/issue4/images/data/1292/DC1/youdelman1.mpg and http://ats.ctsnetjournals.org/content/vol87/issue4/images/data/1292/DC1/youdelman2.mpg.
The internal mammary artery has been favored for CABG in children because of superior patency rates as compared with saphenous vein grafts and its ability to grow with the child [7].

Internal mammary artery steal syndrome after coronary bypass operations is rare in adults and is usually associated with subclavian artery stenosis [8]. Steal syndrome associate with internal mammary artery grafting in children has not previously been reported.

In this case, we hypothesize that ischemia after CABG using a pedicle LIMA graft occurred when impingement on the left subclavian artery or proximal LIMA graft was relieved by removal of the chest retractor. Without the obstruction to flow, runoff through the LIMA into the left subclavian artery, away from the coronary circulation occurred, producing a steal phenomenon. That occlusion of the LIMA graft on return to the operating room did not completely resolve the ischemic changes on electrocardiogram and may be a consequence of the myocardium having been ischemic for several hours and not having had adequate recovery time.

Although CABG for surgical treatment of AAOCA has been advocated by some, the experience with this patient demonstrates a shortfall of this approach. Internal mammary artery steal for a LIMA pedicle graft is a real phenomenon that can be created when two patent vessels supply blood flow to a common end artery. Relative resistances of the vascular beds at either end of the LIMA will determine flow through this graft. It would be unlikely to predict a case of steal syndrome using a LIMA graft without the presence of proximal left subclavian artery stenosis, which has been seen in adults [8]. Based on this experience, we recommend caution in choosing CABG using a LIMA pedicle graft for the treatment of AAOC.

Many thanks to Jill Kaiser for preparing the images for this report.

References
Atrioventricular Septal Defect in a Patient With Heterotaxy Syndrome

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A rare combination of aortopulmonary window and complete atrioventricular septal defect diagnosed in a 2-month-old infant with heterotaxy syndrome is presented. Being aware of this combination of cardiac anomalies before surgical intervention is crucial for perioperative anesthetic technique and preservation of the myocardium.

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Aortopulmonary (AP) window and complete atrioventricular septal defect (AVSD) are cardiovascular defects that may occur alone, although they are associated with other cardiac anomalies. The combination of both defects was reported in a 3-month-old infant described by McElhinney and colleagues in 2000 [1]. Here we describe a 2-month-old infant with coexisting AP window and complete AVSD in association with heterotaxia, a rare combination of congenital cardiovascular defects.

A nondysmorphic 2-month-old boy (3.5 kg) was referred to us for surgical intervention because of little improvement in his growth and intractable cardiac failure despite optimal medical management. At age 3 weeks he was evaluated for cardiac anomaly because of tachypnea and poor feeding. The echocardiography demonstrated a complete AVSD (Rastelli type C) with unbalanced ventricles (the right ventricular type), a left-sided superior caval vein draining through an enlarged coronary sinus, and a right aortic arch. Although a retrograde review of echocardiographic video images confirmed an AP window, it was not diagnosed preoperatively. The infant also had a midline liver with equally sized lobes.

The operation was initiated through a midsternotomy, but cardiac arrest suddenly occurred while the sternotomy was being performed. We rapidly completed the sternotomy, and open cardiac massage was begun. The external cardiac anatomy was evaluated, and a type I AP window (Richardson classification) of about 5 mm in diameter was noted. To have the highest cardiac output during cardiopulmonary resuscitation, we clamped the right pulmonary artery.

Cardiopulmonary resuscitative measures were not successful, and cardiopulmonary bypass was initiated. The patient was cooled to 20°C. The aorta and pulmonary arteries were separated, and the resultant aortic and pulmonary arterial defects were each closed with two separate pericardial patches. The pulmonary artery was banded.

Unfortunately, the patient could not be weaned from bypass by either a tight or loose band because of very poor contractility. The autopsy confirmed all echocardiographic findings and also bilateral trilobed lungs with eparterial bronchi, asplenia, and a symmetrical liver.

Comment

The embryogenesis of AP window is related to incomplete fusion or malalignment of the right and left conotruncal ridges, which normally completely septate the truncus arteriosus between the fifth and eighth weeks of intrauterine life. The division of the truncus arteriosus into separate aortic and pulmonary channels is influenced by cells that migrate from the neural crest [2]. This influence may explain the association of AP window with various arterial abnormalities, including transposition of the great arteries and aortic interruption. Other associated congenital cardiac anomalies with AP window are anomalous origin of a coronary artery, ventricular septal defect, atrial septal defect, patent ductus arteriosus, pulmonary or aortic atresia, tricuspid atresia, right aortic arch, pulmonary venous stenosis, and persistent left superior caval vein to coronary sinus [2]. However, an AP window is not seen when the neural crest is removed [3]. In addition, AP window has not been reported in association with DiGeorge syndrome [4, 5].

Partitioning of the AV canal begins about the middle of the fourth week of gestation and is essentially complete by the end of the fifth week. Concurrent with atrial septation, thickenings of subendocardial tissue, called endocardial cushions, develop in the dorsal and ventral walls of the heart in the region of the AV canal. Therefore, abnormal growth in this region produces a deficiency in the lowermost part of the atrial septum and also a VSD.

Because the dextrodorsal conus cushion contributes to the development of the right AV valve and the outflow tracts lie adjacent to their respective inflow tracts, AVSDs may be associated with conotruncal anomalies, such as tetralogy of Fallot and double-outlet right ventricle. Like in patients with AP window, neural crest ablation in embryonic avian models almost never results in anomalies of the AV junction [6].

AVSDs are also associated with other cardiac defects, including heterotaxy syndromes, total anomalous pulmonary venous return, transposition of great arteries, patent...
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