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Benjamin A. Youdelman

Thomas Jefferson University, benjamin.youdelman@jefferson.edu

Glenn J. Pelletier

Department of Cardiothoracic Surgery, Drexel University College of Medicine

C. Igor Mesia

Department of Pediatric Cardiology, Drexel University College of Medicine

Marshall L. Jacobs

Department of Cardiothoracic Surgery, Drexel University College of Medicine

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Title: Coronary steal syndrome after coronary artery bypass for anomalous aortic origin of a coronary artery

Authors: Benjamin A. Youdelman, M.D.¹, Glenn J. Pelletier, M.D.², C. Igor Mesia, M.D.³,
Marshall L. Jacobs, M.D.²

¹Division of Cardiothoracic Surgery, Department of Surgery, Thomas Jefferson
University

²Department of Cardiothoracic Surgery, Drexel University College of Medicine

³Department of Pediatric Cardiology, Drexel University College of Medicine

From: Division of Cardiothoracic Surgery, Department of Surgery, Thomas Jefferson
University

Corresponding Author: Benjamin A. Youdelman, M.D.
Division of Cardiothoracic Surgery, Department of Surgery
Thomas Jefferson University
1025 Walnut Street, Suite 607
Philadelphia, PA 19107

Tel: 215-955-6996

Fax: 215-955-6010

E-mail: benjamin.youdelman@jefferson.edu

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Abstract

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

Case Report

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 (ALMCA), and the right coronary artery arising from the left sinus (ARCA). 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 conditons, 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 approached 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 thought 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 nine-year-old boy who presented with syncope preceded by palpitations, dizziness, and diaphoresis. Similar symptoms occurred four years earlier, and palpitations are reported with exercise.

His ECG showed sinus arrhythmia, a prolonged QT of 461 milliseconds, 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.

CABG using the left internal mammary artery (LIMA) pedicle graft connected to the left anterior descending (LAD) coronary artery was done. Following 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 (LSCA) 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 LAD through the LIMA and into LSCA demonstrated a steal circuit diverting blood from LAD territory (Video 1). The patient returned to the operating room for surgical revision.

Opening the chest widely again reduced the ischemic changes on ECG, 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 appeared to create a point of coronary stenosis and this opening was enlarged by incising into the aortic media and then repaired.

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

We hypothesized that transferring the LIMA to the ascending aorta would increase the perfusion pressure enough to improve flow through the LIMA graft and relieve the steal syndrome. Following completion of the LIMA to aorta proximal anastomosis, when the chest retractor was removed this time, there was less pronounced ST elevation in the lateral ECG leads and only rare premature ventricular complexes (PVC). The ECG remained normal sinus rhythm and the mild ST elevations improved over time.

The day following surgery, transthoracic echocardiography was normal. A repeat angiogram showed the left coronary ostial repair to be patent with unobstructed antegrade flow. Flow in the distal LIMA graft was biphasic. Injection in the ascending

aorta showed antegrade flow into the LAD from the LIMA graft (Video 2). The coronary steal was no longer present.

A troponin level on postoperative day one was 12.2 ng/ml, but decreased to 3.6 ng/ml by day four. One week after surgery he underwent a stress test using a modified Bruce protocol. No ischemic changes or arrhythmias were observed. In follow up, he is well and free of signs and symptoms of coronary artery insufficiency.

Patients who have anomalous left coronary arteries which come from a single ostium and course between the great arteries are at risk for sudden death (1). The first reported bypass grafting for an aberrant left coronary artery was done in 1977 using a saphenous vein bypass from the aorta to the left main coronary artery (6). The IMA has been favored for CABG in children because of superior patency rates as compared to 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 associated with IMA grafting in children has not previously been reported.

In this case we hypothesize that ischemia following CABG using a pedicle LIMA graft occurred when impingement on the LSCA or proximal LIMA graft was relieved by removal of the chest retractor. Without the obstruction to flow, runoff through the LIMA into the LSCA, 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 ECG 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 AAOCA.

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