

There are a few additional case reports that have described idiopathic fetal ductus constriction/closure without evidence of maternal use of a PDA constricting agent or structural cardiac defect (7, 8).

In this case, we excluded all known secondary causes of premature ductus constriction by history, including NSAIDs, teas, and herbal remedies as well as structural cardiac and ductus abnormalities. We believe that our case represents idiopathic premature restriction of the DA. We have no clear explanation or theory as to why this ductus was restricted.

In the healthy fetus, ductal flow velocity increases gradually with advancing gestational age. In previous reports, ductal restriction has been defined as a peak systolic velocity of greater than 1.4 m/s, a persistent diastolic peak flow velocity greater than 0.35 m/s and a pulsatility index less than 1.9 (9, 10). Each of these criteria was found in our patient. The four-chamber view is a key to the detection of ductal constriction or closure. Although a dilated right ventricle and significant tricuspid regurgitation may be the first indication of DA restriction on this view, when the constriction is initially occurring, the four-chamber view may appear to be normal. Thus, the diagnosis should be made based on the velocity of the DA upon Doppler examination in the sagittal view. Thus, the determination of DA velocity should be a routine part of fetal echocardiographic examination.

Conclusion

The outcomes associated with premature ductus constriction include right heart failure, fetal hydrops, persistent pulmonary hypertension and death. The wide spectrum of clinical presentations is dependent on the duration and severity of flow obstruction. If ductal arteriosus flow is not checked using color and pulsed Doppler, the diagnosis of this serious condition could be missed.

Kadir Babaoğlu, Yiğit Çakıroğlu*, Gürkan Altun, Emek Doğer*, Demet Oğuz**

Departments of Pediatric Cardiology, *Obstetrics and Gynecology and **Neonatology, Faculty of Medicine, Kocaeli University, Kocaeli-Turkey

Video 1. A four-chamber view showing marked right ventricular enlargement and hypertrophy with tricuspid regurgitation

Video 2. A Sagittal views showing that the ductus arteriosus is severely constricted. Color flow imaging of the ductus arteriosus shows a narrowed jet with increased velocity, indicating ductus arteriosus constriction

References

1. Becker AE, Becker MJ, Wagenvoort CA. Premature contraction of the ductus arteriosus: a cause of foetal death. J Pathol 1977;121:187-91. [CrossRef]
2. Harlass FE, Duff P, Brady K, Read J. Hydrops fetalis and premature closure of the ductus arteriosus: a review. Obstet Gynecol Surv 1989;44:541-3. [CrossRef]
3. Hofstadler G, Tulzer G, Altmann R, Schmitt K, Danford D, Huhta JC. Spontaneous closure of the human fetal ductus arteriosus--A cause of fetal congestive heart failure. Am J Obstet Gynecol 1996;174:879-83. [CrossRef]
4. Koren G, Florescu A, Costei AM, Boskovic R, Moretti ME. Nonsteroidal antiinflammatory drugs during third trimester and the risk of premature closure of the ductus arteriosus: a meta-analysis. Ann Pharmacother 2006;40:824-9. [CrossRef]
5. Azancot-Benisty A, Benifla JL, Matias A, De Crépy A, Madelenat P. Constriction of the fetal ductus arteriosus during prenatal betamethasone therapy. Obstet Gynecol 1995;85:874-6. [CrossRef]
6. Zielinsky P, Piccoli AL Jr, Manica JL, Nicoloso LH, Menezes H, Busato A, et

- al. Maternal consumption of polyphenol-rich foods in late pregnancy and fetal ductus arteriosus flow dynamics. J Perinatol 2010;30:17-21. [CrossRef]
7. Trevett TN Jr, Cotton J. Idiopathic constriction of the fetal ductus arteriosus. Ultrasound Obstet Gynecol 2004;23:517-9. [CrossRef]
8. Downing GJ, Thibeault DW. Pulmonary vasculature changes associated with idiopathic closure of the ductus arteriosus and hydrops fetalis. Pediatr Cardiol 1994;15:71-5.
9. Huhta JC, Moise KJ, Fisher DJ, Sharif DS, Wasserstrum N, Martin C. Detection and quantitation of constriction of the fetal ductus arteriosus by Doppler echocardiography. Circulation 1987;75:406-12. [CrossRef]
10. Tulzer G, Gudmundsson S, Sharkey AM, Wood DC, Cohen AW, Huhta JC. Doppler echocardiography of fetal ductus arteriosus constriction versus increased right ventricular output. J Am Coll Cardiol 1991;18:532-6. [CrossRef]

Address for Correspondence/Yazışma Adresi: Dr. Kadir Babaoğlu
Kocaeli Üniversitesi Tıp Fakültesi, Pediyatrik Kardiyoloji Bilim Dalı,
Kocaeli- Türkiye
Phone: +90 262 303 80 35
E-mail: babaoğluk@yahoo.com

Available Online Date/Çevrimiçi Yayın Tarihi: 27.05.2013

©Telif Hakkı 2013 AVES Yayıncılık Ltd. Şti. - Makale metnine www.anakarder.com web sayfasından ulaşılabilir.

©Copyright 2013 by AVES Yayıncılık Ltd. - Available online at www.anakarder.com doi:10.5152/akd.2013.150



Subclavian artery stenosis in a patient undergoing coronary bypass using composite t-grafting technique: is it subclavian artery stenosis or more?

Kompozit T-graft tekniği kullanılan koroner baypass bir hastada subklavian arter stenozu: Subklavian arter stenozu ya da fazlası?

Introduction

Occlusive arterial disease of the upper extremity occurs at a much lower frequency than disease of the lower extremity. They are mostly seen in the subclavian and the innominate artery. The common cause is atherosclerosis (1). With an increase in the use of internal mammary artery in heart surgery, detection of subclavian artery stenosis (SAS) has gained importance. In a patient with history of coronary bypass surgery (CABG) 6 months ago, we determined SAS. What makes this case interesting is that left internal mammary artery (LIMA) was anastomosed to the left anterior descending artery (LAD); furthermore, the proximal anastomoses of the arterial grafts belonging to the circumflex (CX) and the right coronary arteries (RCA) were anastomosed to LIMA using the composite T-grafting technique. Hence, perfusion of the heart was rendered totally dependent on LIMA and consequently on the left subclavian artery.

Case Report

A 65-year-old woman was admitted to our clinic with pulmonary edema. Her medical history revealed that she had undergone coronary artery bypass grafting with a LIMA conduit to the LAD and with a composite T-grafting technique applied to the RCA and CX six months ago. On

examination, the blood pressure was 60/40 mmHg in the right arm and there was no pulse in the left arm. The electrocardiogram showed normal sinus rhythm. Transthoracic echocardiography revealed severe left ventricular dysfunction. Oxygen inhalation, dopamine infusion and IV furosemide was administered. Pulmonary edema regressed and the patient's condition stabilized. Coronary angiography revealed cut-off in all the major coronary arteries. In addition, in the selective aortic arch vessel angiography, the left subclavian artery had a stenosis of 90% at 2 cm distal to its origin (Fig. 1). The LIMA-LAD graft was patent. The radial artery graft to the CX and RCA was anastomosed to LIMA from its proximal (Fig. 2, 3). Cardiac perfusion was totally dependent on LIMA and consequently on the left subclavian artery. Angioplasty was not appropriate because of the severe lesion angle (approximately 90°). The patient was referred to vascular surgery. She underwent left carotid-left subclavian bypass with a Dacron graft. She was discharged on the postoperative 5th day and has remained symptom-free for 1 year since surgery.

Discussion

SAS is a very rare disorder of the arterial tree and has gained clinical importance with an increase in the use of LIMA as a graft in coronary bypass surgery. It is generally caused by atherosclerotic disease. Other etiological factors include arteritis, neurofibromatosis, fibromuscular dysplasia, radiation, posttraumatic scarring and compression syndromes (2-5). The incidence is reported to be up to 3.4% after CABG (6). SAS can be the cause of various phenomena, namely the subclavian steal syndrome, coronary-subclavian steal syndrome (CSS) and ischemic symptoms without steal due to diminished blood flow (1).

Although the patients are usually asymptomatic, the symptoms are usually due to either distal ischemia due to stenosis or embolic events, particularly ischemia occurring during upper extremity exercise because of diminished blood flow to the left arm, to the cerebrovascular circulation. Symptoms include angina, lightheadedness, left arm numbness or weakness, silent ischemia, heart failure, and myocardial infarction (7). CSS should be suspected when there are symptoms of vertebrobasilar insufficiency, arm claudication, and differential upper arm systolic blood pressure greater than or equal to 20 mmHg (8). Definitive

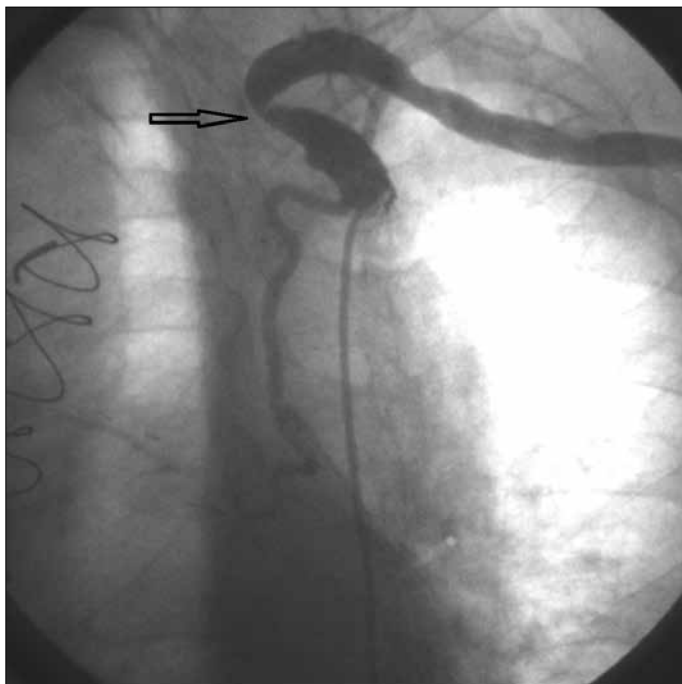


Figure 1. Proximal subclavian artery stenosis

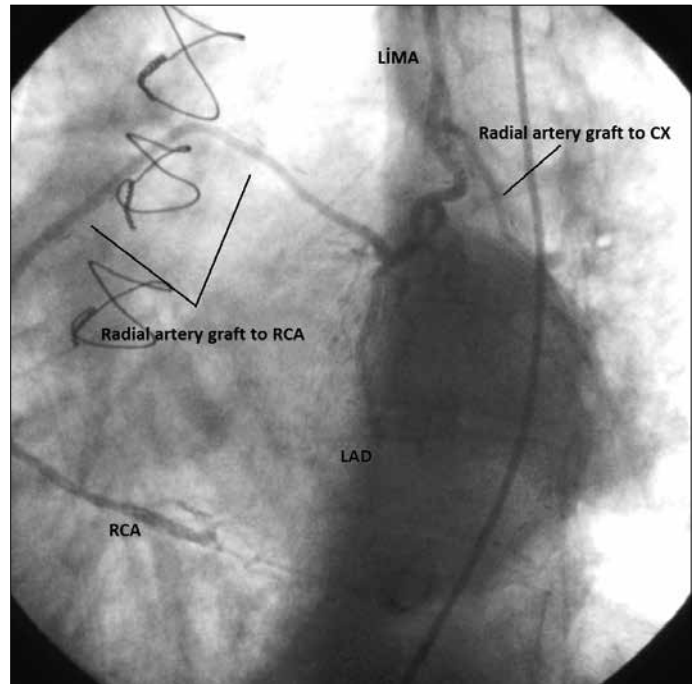


Figure 2. LIMA-LAD graft was patent, radial artery graft to CX and RCA was anastomosed to LIMA

CX - circumflex artery, LAD - left anterior descending artery, LIMA - left internal mammary artery, RCA - right coronary artery

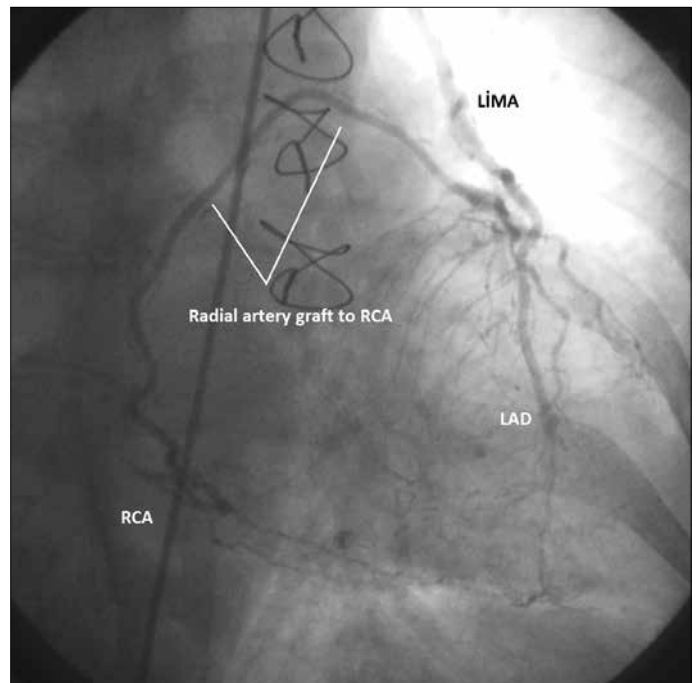


Figure 3. LIMA-LAD graft was patent, radial artery graft to CX and RCA was anastomosed to LIMA

CX - circumflex artery, LAD - left anterior descending artery, LIMA - left internal mammary artery, RCA - right coronary artery

diagnosis requires angiographic evaluation of the aortic arch. Hemodynamically significant stenosis can be ruled out by simple bilateral brachial artery BP measurement rather than routine angiography. In addition, computed tomography, magnetic resonance angiography and duplex ultrasonography are also available for the diagnosis (9).

The treatment options for SAS are surgery or percutaneous transluminal

angioplasty and stenting. Percutaneous intervention has recently gained popularity because it is less invasive. A review by Ribichini et al. (10) summarized the success and safety of percutaneous treatment of these lesions.

In our case, there was no steal, but there was severe myocardial ischemia due to diminished blood flow to LIMA in which there was distal subclavian stenosis. A careful evaluation before the operation for preventing of this syndrome is essential.

Conclusion

Although SAS is very rare and the patients are usually asymptomatic, it has clinical importance in patients with CABG, especially those with composite T-grafting technique. Patients with angina undergoing CABG using LIMA grafting should be considered in terms of left SAS as a possible cause of myocardial ischemia.

Arif Arısoy, Selim Topçu, Hüseyin Karal, Serdar Sevimli
From Department of Cardiology, Faculty of Medicine, Atatürk University, Erzurum-Turkey

References

1. Hacıbayramoğlu M, Werba T, Schmidt A, Klepzig H. Angina pectoris in consequence of subtotal subclavian artery stenosis 2 years after CABG. *Thorac Cardiovasc Surg* 2010; 58: 47-9. [CrossRef]
2. Andros G, Schneider PA, Harris RW, Dulawa LB, Oblath RW, Salles-Cunha SX. Management of arterial occlusive disease following radiation therapy. *Cardiovasc Surg* 1996; 4: 135-42. [CrossRef]
3. Smith TP, Halbach VV, Fraser KW, Teitelbaum GP, Dowd CF, Higashida RT. Percutaneous transluminal angioplasty of subclavian stenosis from neurofibromatosis. *AJNR Am J Neuroradiol* 1995; 16(4 Suppl): 872-4.
4. Tyagi S, Verma PK, Gambhir DS, Kaul UA, Saha R, Arora R. Early and long-term results of subclavian angioplasty in aortoarteritis (Takayasu disease): comparison with atherosclerosis. *Cardiovasc Intervent Radiol* 1998; 21: 219-24. [CrossRef]
5. Cingöz F, Bingöl H, Özal E, Tatar H. Coronary subclavian steal syndrome in a patient with Behçet's disease. *Thorac Cardiovasc Surg* 2010; 58: 244-6. [CrossRef]
6. Lobato EB, Kern KB, Bauder-Heit J, Hughes L, Sulek CA. Incidence of coronary-subclavian steal syndrome in patients undergoing noncardiac surgery. *J Cardiothorac Vasc Anesth* 2001; 15: 689-92. [CrossRef]
7. Sadek MM, Ravindran A, Marcuzzi DW, Chisholm RJ. Complete occlusion of the proximal subclavian artery post-CABG: presentation and treatment. *Can J Cardiol* 2008; 24: 591-2. [CrossRef]
8. Marshall WG Jr, Miller EC, Kouchoukos NT. The coronary-subclavian steal syndrome: report of a case and recommendations for prevention and management. *Ann Thorac Surg* 1988; 46: 93-6. [CrossRef]
9. Zebebe C, Özdemir HI, Hamad MA. Coronary ischemia due to subclavian stenosis after arterial revascularization. *Asian Cardiovasc Thorac Ann* 2011; 19: 169-71. [CrossRef]
10. Ribichini F, Maffè S, Ferrero V, Cotroneo A, Vassanelli C. Percutaneous angioplasty of the subclavian artery in patients with mammary-coronary bypass grafts. *J Interv Cardiol* 2005; 18: 39-44. [CrossRef]

Address for Correspondence/Yazışma Adresi: Dr. Arif Arısoy, Atatürk Üniversitesi Tıp Fakültesi, Kardiyoloji Anabilim Dalı, Erzurum-Türkiye
Phone: +90 442 316 63 33
E-mail: arfarsy@hotmail.com



Available Online Date/Çevrimiçi Yayın Tarihi: 27.05.2013

© Telif Hakkı 2013 AVES Yayıncılık Ltd. Şti. - Makale metnine www.anakarder.com web sayfasından ulaşılabilir.

© Copyright 2013 by AVES Yayıncılık Ltd. - Available online at www.anakarder.com

doi:10.5152/akd.2013.151

Dilated cardiomyopathy due to miliary tuberculosis

Miliyer tüberküloza bağlı dilate kardiyomiyopat

Introduction

Miliary tuberculosis (TB) is a potentially lethal form of tuberculosis resulting from massive lymphohaematogeneous dissemination of *Mycobacterium tuberculosis* bacilli. Involvement of heart in tuberculosis occurs in one to two percent of patients with tuberculosis (1-6). We as well introduced a case with dilated cardiomyopathy (DCM) secondary to miliary TB, which was improved with antituberculosis therapy.

Case Report

A 15-year-old girl was presented with weakness, fatigue, weight loss, and swelling on the neck. Productive cough, night fever and night sweat particularly for the last three week. Her personal and family medical history was remarkable revealing a history of TB in the children of her aunt and uncle. She had a single bacille Calmette-Guerin (BCG) scar. Auscultation of the lungs revealed bilateral rales. On cardiac auscultation, she had tachycardia and grade 2-3/6 regurgitant systolic murmur on the mesocardiac focus. Purified protein derivative was negative, gastric aspirates was negative, and TB screening performed via polymerase chain reaction (PCR) method was negative as well.

The neck and abdominal ultrasonographies revealed multiple lymphadenopathies (LAP) on cervical chains and submandibular region. Postero-anterior chest radiograph showed miliar appearance (Fig. 1A). Her electrocardiogram was considered unremarkable. Transthoracic echocardiography (Fig. 2A) showed enlarged left ventricle end-diastolic dimension of the left ventricle was 6.21 cm, end-systolic dimension of the left ventricle was 5.6 cm and decreased systolic function. Systolic ejection fraction and shortening fraction were found 21% and 10% respectively. There was grade 1-2 mitral regurgitation secondary to the dilated annulus. Other causes of DCM were excluded. Cervical lymph node biopsy showed signs consistent with caseified granulomatous lymphadenitis. On thoracic computed tomography (Fig. 2B), calcified-noncalcified lymph nodes of pathological size were detected in bilateral axillary, paratracheal, perivascular, carinal, subcarinal, bilateral hilar and paracardiac regions. Cavitation was observed in a nodule in the left lung. It was thought that these findings might be consistent with miliary TB.

After making the diagnosis of miliary TB, four-drug combination antituberculosis therapy with anticongestive medications was commenced. The patient, whose symptoms relieved on the 2nd-3rd week of antituberculosis therapy, was discharged to be followed in the outpatient clinic. Control echocardiography exam performed on the 2nd month of polyclinic follow-up (Fig. 2C), end-diastolic dimension of the left ventricle was 5.06 cm and the systolic ejection has been increased up to 66%. Within two months, 6 kg increase in body weight and the improvement in anemia of chronic disease and disappearance of LAP's in the neck was determined. Control chest radiograph (Fig. 1B) and tomography (Fig. 2D) was considered unremarkable.

Discussion

Tuberculosis myocarditis is extremely rare and may occur usually secondary to hematogenous or direct spread, as well as retrograde spread from the lymph nodes, of TB of another focus to the myocardium (1-5). TB myocarditis may present itself with rhythm disorders, as well as different pictures such as sudden cardiac death (1, 4, 6).