The Heart Surgery Forum #2007-1020
10 (4), 2007 [Epub May 2007]
doi: 10.1532/HSF98.20071020

Online address: http://cardenjennings.metapress.com

Discrete Membranous Subaortic Stenosis Complicated by Infective Endocarditis: A Case Report

Emre Gurel, MD,[1] Tansu Karaahmet, MD,[1] Ali Cevat Tanalp, MD,[2] Bulent Mutlu, MD,[1] Yelda Basaran, MD[1]

[1]Department of Cardiology, Kartal Kosuyolu Heart Education and Research Hospital, Istanbul, Turkey;
[2]Department of Cardiology, Yuzuncuyil Hospital, Ankara, Turkey

ABSTRACT

Discrete membraneous subaortic stenosis is an uncommon cause of left ventricular outflow tract obstruction. Although its relationship to infective endocarditis is well defined, the expected site of vegetation is over the aortic valve. We report on a 46-year-old man who had a discrete membranous subaortic stenosis, complicated with infective endocarditis, in which the vegetation was over the subaortic membrane and the aortic valve was spared. To our knowledge, this is the first reported case of that entity.

INTRODUCTION

Aortic valve abnormalities and a number of congenital heart diseases, primarily ventricular septal defect, patent ductus arteriosus, and tetralogy of Fallot are reported to be the common underlying conditions of infective endocarditis. We report here a case of discrete membraneous subaortic stenosis, complicated by a vegetation on the discrete membrane.

CASE REPORT

A 46-year-old man was referred to our center for evaluation of a cardiac murmur that was discovered during physical examination. A detailed anamnesis revealed that the patient had undergone a tooth extraction 3 months ago and for the past 2 months had suffered from progressive fatigue, dyspnea, palpitation, sweating, and an intermittant fever that did not exceed 38.5 C; he had been receiving nonspecific antibiotic therapy for several days. On physical examination, the blood pressure was 110/75 mmHg and the body temperature was 37.2 C. A systolic thrill was palpable at the apex and left sternal border and a grade 4/6 harsh systolic ejection murmur was audible at the left sternal border, radiating to the right carotid area. The remaining physical examination was normal. The surface electrocardiogram revealed signs of left ventricular hypertrophy, and a chest x-ray also showed concentric hypertrophy of the left ventricle and slight enlargement of the left atrium. Laboratory examinations demonstrated leukocytosis, increased acute phase reactants, and normal blood urea nitrogen, creatinine, and urine parameters. Three blood cultures failed to grow any microorganisms. Transthoracic echocardiography revealed narrowing of the left ventricular outflow tract (LVOT) due to both mild hypertrophy in the interventricular septum and a subaortic discrete membrane that was associated with the base of the anterior mitral leaflet. A 120 mmHg maximal systolic pressure gradient was demonstrated across the LVOT. There was also an 8-mm long, mobile, hyperechogenic mass on the membrane, consistent with vegetation (Figure 1). There was also mild aortic regurgitation, systolic anterior motion of the mitral anterior leaflet, and mild mitral regurgitation. Aortic cusps were clear and there was no vegetation on the aortic cusps. The patient was diagnosed with subacute infective endocarditis and penicillin G and gentamycin combination therapy was initiated. The clinical symptoms of endocarditis diminished and the size of the vegetation reduced to 6 mm during 2 weeks of therapy. The patient was free from fever during this period. Because of LVOT obstruction and accompanying vegetation, surgical therapy was planned. On the 14th day of the antibiotic therapy, the patient was operated and the obstructive discrete membrane and the vegetation were excised without myectomy (Figures 2 and 3). The procedure did not include a myectomy as the patient did not have marked septal hypertrophy. The intraoperative appearance of the aortic valve was normal and free from vegetation. The patient recovered without complication. The histopathologic examination of the resected material revealed inflammation, small vessel proliferation, granulation tissue, and subacute endocarditis of the discrete membrane (Figure 4). Antibiotherapy was reinitiated and continued for 6 weeks, during which the patient did not have fever. A control echocardiographic examination, which was performed one week after the operation, revealed slightly reduced residual pressure gradient across the LVOT (maximal 90 mmHg), probably due to inflammatory responses of operated, unhealed tissues and edema. However, a markedly reduced pressure gradient (maximal 50 mmHg) was determined one month after the operation. The patient is under echocardiographic follow-up with recommendation of infective endocarditis prophylaxis.

Received January 17, 2007; received in revised form April 9, 2007; accepted April 23, 2007.

Correspondence: Emre Gurel, MD, Çinar Mah. Yilmaz Sok. Arzum Apt. No:6 Daire:7 Küçükyali, Istanbul, Turkey; 90-533-4232151, 90-212-5875825; fax: 90-216-3390441 (e-mail: emregurelctf@yahoo.com).

© 2007 Forum Multimedia Publishing, LLC

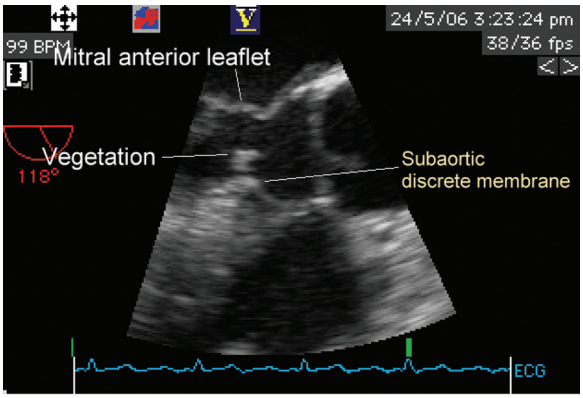

Figure 1. Transesophageal echocardiographic image at the aortic level demonstrating the subaortic discrete membrane and accompanying vegetation.

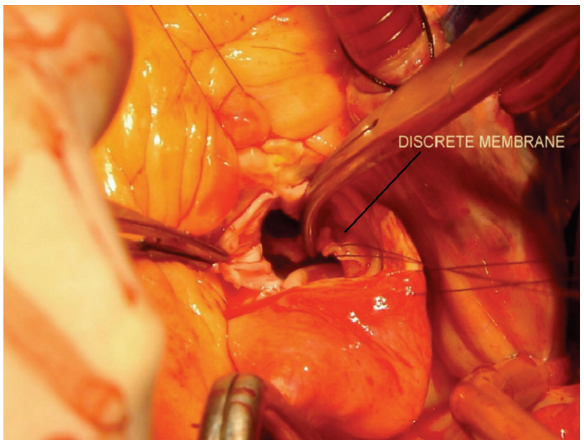

Figure 3. Intraoperative view of the subaortic discrete membrane.

DISCUSSION

Discrete membranous subaortic stenosis is a rare cause of LVOT obstruction, which may coexist with various congenital lesions such as ventricular septal defect, patent ductus arteriosus, coarctation of the aorta, and bicuspid aortic valve. Subaortic stenosis can be divided into discrete and fibromuscular tunnel forms. Discrete type is more common and consists of a fibrous ridge alone or associated with a muscular base located below the aortic valve in the LVOT or even in cystic form [Oliver 2001; Ciliers 2002; Izgi 2004]. A majority of patients have abnormal aortic valve. Aortic insufficiency results from trauma to the leaflets by the abnormal jet-flow pattern [Oliver 2001; Ciliers 2002]. The jet originating from the narrowed subaortic tract damages the aortic cusps and causes regurgitation, which makes the aortic valve prone to infective endocarditis [Darcin 2003]. Surgical repair is advised to reduce the damage to the aortic valve. Adequate resection of subaortic stenosis reduces the risk of endocarditis. Another criterion for operation is new-onset aortic regurgitation regardless of the gradient [Erentug 2005]. However, in this case, the discrete subaortic membrane was complicated by a vegetation resulting from the regurgitant aortic jet and there was no vegetation on aortic cusps. The patient was operated on because of the critical pressure gradient and the discrete membrane that was causing aortic regurgitation. In the operation, the discrete membrane and the accompanying vegetation was resected and septal myectomy was not performed (Figures 2 and 3).

For the surgical treatment of discrete subaortic stenosis, there are several options for the surgeon. These include resection of localized subaortic stenosis with or without myectomy, the Ross-Konno procedure, which is used in patients who require aortic valve replacement, the Konno procedure, and the modified Konno procedure, which is preferred for tunnel type subaortic stenosis [Kouchoukos 2003; Marasini 2003]. Although membranectomy with or without septal myotomy or myectomy has been the treatment of choice, there are still controversies concerning the operative methods and uncertainties about the recurrence of subaortic obstruction and development of aortic regurgitation after repair [Welton 2001; Darcin 2003;

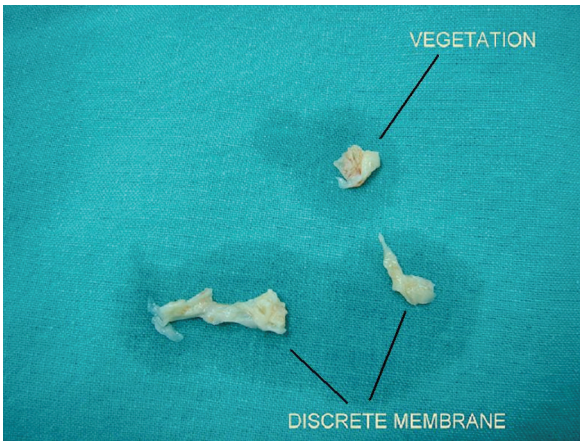

Figure 2. Gross pathologic examination of the resected vegetation and the discrete membrane.

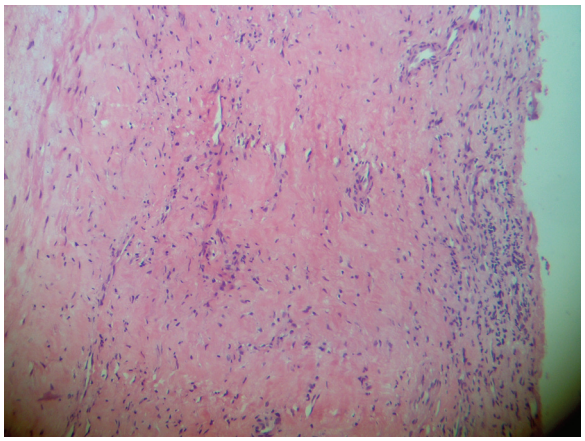

Figure 4. Histopathologic examination of the discrete membrane revealing active inflammatory granulation tissue formed by small vessel proliferation accompanying mixed-type inflammation.

Niva 2005]. In many authors' series, myectomy or myotomy with membrane resection is suggested because of good results and low recurrence rates; however, some authors suggest that resection without myectomy is expected to relieve the obstruction with higher recurrence rates and the surgeon can decide to perform myectomy or not, according to the degree of left ventricular hypertrophy. Thus in this case myectomy was not preferred by the surgeon as there was no marked hypertrophy.

Recurrence rates of 6% to 30% have been reported in various series. The risk of recurrence may be caused by inadequate resection, regrowth of tissue from the region of the septum, and scar formation in the subvalvular area during healing [Darcin 2003].

In the postoperative patients, close follow-up with serial transthoracic echocardiographic examinations is helpful in early detection of subaortic reobstruction or recurrence of aortic valve pathology after surgery. Our patient has been under echocardiographic follow-up for 6 months after surgery and no recurrence has yet been observed.

REFERENCES

Ciliers AM, Gewillig M. 2002. Rheology of discrete subaortic stenosis. Heart 88:335-6.

Darcin OT, Yagdi T, Atay Y, et al. 2003. Discrete subaortic stenosis: surgical outcomes and follow-up results. Tex Heart Inst J 30:286-92.

Erentug V, Bozbusa N, Kirali K, et al. 2005. The surgical treatment of subaortic obstruction in adolescents and adults: long-term follow up. J Card Surg 20:1-6.

Izgi A, Kirma C, Tanalp AC, Mansuroglu D, Ipek G. 2004. Cystic subaortic discrete membrane in an elderly woman. J Heart Valve Dis 13:925-6.

Kouchoukos NT, Blackstone EH, Doty DB, Hanley FL, Karp RB, eds. 2003. *Congenital Aortic Stenosis in Kirklin/Barrat-Boyes Cardiac Surgery.* Vol. 2, 3 ed. Philadelphia, PA; 1265-313.

Marasini M, Zannini L, Ussia GP, et al. 2003. Discrete subaortic stenosis: incidence, morphology and surgical impact of associated subaortic anomalies. Ann Thorac Surg 75:1763-8.

Niva K, Nakazava M, Tateno S, Yoshinaga M, Terai M. 2005. Infective endocarditis in congenital heart disease: Japanese national collaboration study. Heart 91:795-800.

Oliver JM, Gonzalez A, Gallego P, Recalde AS, Benito F, Mesa JM. 2001. Discrete subaortic stenosis in adults: increased prevalence and slow rate of progression of the obstruction and aortic regurgitation. J Am Coll Cardiol 38:835-42.

Welton M. 2001. Natural history of discrete subvalvular aortic stenosis: management implications. J Am Coll Cardiol 38:843-5.

© 2007 Forum Multimedia Publishing, LLC