Transmitral myectomy and how to deal with systolic anterior motion (SAM) in hypertrophic obstructive cardiomyopathy

Sabine Meier[1], Thilo Noack[1], Friedrich W. Mohr[1], Joerg Seeburger[1], Jurgen Passage[2,3]

[1]Department of Cardiac Surgery, Heart Center, University of Leipzig, Leipzig, Germany; [2]Department of Cardiothoracic Surgery, Sir Charles Gairdner Hospital, Perth, Western Australia, Australia; [3]The Baird Institute, Sydney, NSW, Australia and School of Medicine Fremantle, University of Notre Dame, Western Australia, Australia

Correspondence to: Jurgen Passage. Department of Cardiothoracic Surgery, Sir Charles Gairdner Hospital, Perth, Western Australia, Australia. Email: jurgen.passage@gmail.com.

Submitted Mar 14, 2017. Accepted for publication Apr 20, 2017.
doi: 10.21037/acs.2017.06.01
View this article at: http://dx.doi.org/10.21037/acs.2017.06.01

Introduction

Since Brock first described hypertrophic cardiomyopathy (HCM) in 1957, much has been learnt about the underlying aetiology and the different patterns of hypertrophy. In the majority of cases, an autosomal-dominant inherited mutation of sarcomere proteins is the cause for hypertrophy of the left, sometimes also of the right, ventricle, leading to several patterns of wall-thickening, including asymmetrical septal, midventricular, apical and concentric hypertrophy (1). Due to septal bulging into the left ventricular outflow tract (LVOT) and anterior malposition of the anterior papillary muscle, systolic anterior motion (SAM) of the anterior mitral leaflet (AML) can occur. This is the result of the Venturi effect leading to a relevant LVOT gradient and mitral regurgitation caused by delayed leaflet closure. Several studies have demonstrated that SAM can occur even in the absence of septal hypertrophy due to the concomitant abnormality of the mitral apparatus in HOCM (2,3). In addition, decreased AML mobility and abnormal positioning of the line of coaptation towards the LV outflow tract increases the SAM and MR (*Video 1*). Symptoms vary from dyspnea, angina, presyncope, syncope and sudden cardiac death (1).

Several surgical techniques have been described to address these problems, including septal myectomy as a stand-alone procedure (Morrow), myectomy in combination with mitral valve replacement (Cooley), myectomy combined with partial resection and mobilization of the papillary muscles (Schoendube), anterior papillary muscle resection and resuspension of the AML (Rankin), and AML patch-extension (Kofflard). We herein describe a modified technique for treatment of MR and SAM in HOCM using a minimally invasive, endoscopic supported approach for a combination of trans-mitral LV myectomy and valve repair with the implantation of neochordae and MV annuloplasty.

Clinical vignette

A 45-year-old male patient was referred for mitral regurgitation. The patient experienced NYHA class III dyspnoea. The past medical history of this patient was unremarkable except for mild arterial hypertension. Echocardiographic examination revealed severe MR due to SAM caused by restricted AML mobility and HOCM. Coronary artery disease was excluded by angiography. Additional investigations included MRI for detailed anatomic information of the mitral valve and the papillary muscle distribution, the pattern of hypertrophy and confirmation of the pathomechanism of mitral regurgitation.

Surgical technique

General anaesthesia is induced with single lumen endotracheal intubation. Arterial and central venous lines are placed at preferred sites. The patient is placed in a supine position with slight elevation of the right chest with the right arm slightly abducted. Cardiopulmonary bypass (CPB) is instituted via cannulation of the right femoral vessels. A multi-stage femoral venous cannula is positioned

with its tip in the superior vena cava in Seldinger technique under transesophageal echo cardiographic (TOE) guidance. The common femoral artery is then cannulated with an appropriate size (16–20 Fr) cannula in Seldinger technique under TOE guidance. Mild hypothermia (34 ℃) is used. A right antero-lateral mini-thoracotomy is then performed and the chest entered through the 4th intercostal space (ICS). Additional 5 mm incisions are then made (1), in the lateral clavicular line in the second ICS for placement of the videoscope and carbon dioxide insufflation and (2) in the anterior axillary line in the second ICS for later placement of the Chitwood cross clamp. The heart is arrested with Custodiol cardioplegia delivered via the aortic root. Sondergaard's groove is then dissected and the left atrium opened. A left atrial retractor is inserted.

After inspection of the MV, all native chords connected to the AML, including primary, secondary and tertiary chordae, are resected (*Video 1*) from both papillary muscles and the AML. The AML is pulled up to the interatrial septum to allow exposure of the interventricular septum and an extensive subaortic myectomy is performed (*Video 1*). In case of suboptimal exposure, a 30° scope can be helpful. The AML is then resuspended using the "Loop-technique" with pre-manufactured Gore-Tex loops at a definite length (average 8 loops of 26 mm length). The required length of the neochordae is measured using a caliper measuring the distance from the tip of the corresponding papillary muscle to the "neo-coaptation" zone projected the annular plane. The neochordae need to be significantly longer than the native chords to allow for adequate AML mobilisation to move the coaptation plane posteriorly, away from the LVOT. After securing one set of loops to the anterolateral and posteromedial papillary muscles respectively, the free ends of the loops are attached to the corresponding free margin of the AML using Gore-Tex sutures. It is important to distribute the loops equally on all segments of the AML and not to cross the midline of the A2 segment to allow for natural and free leaflet mobility without distortion. An annuloplasty is then performed if necessary due to significant annular dilation. A partial posterior, flexible band rather than a complete ring is used in order to not decrease the antero-posterior diameter of the annulus, allowing the line of coaptation to move away from the LVOT. After testing the competence of the valve with a sealing probe, the left atrium is closed in a standard fashion (4,5). De-airing is achieved by filling the left atrium prior to tying the suture line and through suction on the aortic root vent. A ventricular temporary pacing wire is placed and the cross clamp removed. After a brief period of re-perfusion, the patient is then ventilated and temporarily weaned off CPB. After confirmation of satisfactory MV function, absence of SAM and intra-cardiac air, and satisfactory left and right ventricular function by TOE, bypass is recommenced and the aortic root vent removed. Haemostasis is confirmed and the pericardium re-approximated. A single intercostal chest tube is placed and the thoracotomy and port sites closed. The patient is then weaned off CPB and protamine is given. The femoral cannulas are removed and the groin wound closed in layers.

Comments

The concept of the combination of a trans-mitral LV myectomy and re-suspension of the AML with a partial, flexible annuloplasty is valuable for several reasons: (I) by extensive subaortic myectomy, enlargement of the LVOT is accomplished; (II) resection of relatively restricted native chords to the AML and resuspension with longer neochordae moves the line of coaptation back into the mitral annular plane, away from the LVOT; (III) the use of a partial flexible, posterior annuloplasty band does not reduce the anterior-posterior mitral annular diameter and thus allows the line of coaptation to remain posteriorly located. Our herein described technique is an effective approach for correction of MR and SAM in HOCM.

Acknowledgements

None.

Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

References

1. Fifer MA, Vlahakes GJ. Management of symptoms in hypertrophic cardiomyopathy. Circulation 2008;117:429-39.
2. Levine RA, Vlahakes GJ, Lefebvre X, et al. Papillary muscle displacement causes systolic anterior motion of the mitral valve. Experimental validation and insights into the mechanism of subaortic obstruction. Circulation 1995;91:1189-95.
3. Jiang L, Levine RA, King ME, et al. An integrated

mechanism for systolic anterior motion of the mitral valve in hypertrophic cardiomyopathy based on echocardiographic observations. Am Heart J 1987;113:633-44.
4. Mohr FW, Seeburger J, Misfeld M. Keynote Lecture-Transmitral hypertrophic obstructive cardiomyopathy (HOCM) repair. Ann Cardiothorac Surg 2013;2:729-32.
5. Seeburger J, Borger MA, Falk V, et al. Gore-Tex Loop Implantation for Mitral Valve Prolapse: The Leipzig Loop Technique. Oper Tech Thorac Cardiovasc Surg 2008;13:83-90.

Cite this article as: Meier S, Noack T, Mohr FW, Seeburger J, Passage J. Transmitral myectomy and how to deal with systolic anterior motion (SAM) in hypertrophic obstructive cardiomyopathy. Ann Cardiothorac Surg 2017;6(4):416-418. doi: 10.21037/acs.2017.06.01

