Heart, Lung and Circulation (2019) **28**, 477–485
1443-9506/04/$36.00
https://doi.org/10.1016/j.hlc.2018.02.006

ORIGINAL ARTICLE

Pathoanatomic Findings and Treatment During Hypertrophic Obstructive Cardiomyopathy Surgery: The Role of Mitral Valve in LVOT Obstruction

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Received 7 September 2017; accepted 5 February 2018; online published-ahead-of-print 14 February 2018

Background To assess the role of the mitral valve apparatus (leaflets, chordae and papillary muscles, (PM)) in left ventricle outflow tract (LVOT) obstruction, and results of the surgical treatment for hypertrophic obstructive cardiomyopathy (HOCM).

Methods Twenty-eight consecutive patients (58 ± 11 years, 53% female) undergoing HOCM surgery from 2007 to 2016 at our institute were retrospectively reviewed. Endpoints included the involvement of the mitral valve in LVOT obstruction, mortality, and changes in clinical and echocardiographic characteristics after HOCM surgery.

Results Secondary chordae tendineae tractioning the anterior mitral leaflet to the interventricular septum, and systolic anterior motion were detected in 78% of the patients. Anomalous, hypertrophied, and fused PM with muscularis trabeculae hypertrophy were found in 50%, 25%, and 35% of the patients, respectively. Four patients had posterior leaflet redundancy. Secondary chordae (92%), PM, and muscularis trabeculae resection (71%), and PM splitting and elongation (28%) were added variably to septal myectomy (100%). Nine procedures (32%) on mitral valve leaflets were performed, involving six posterior and three anterior mitral leaflets. Long-term follow-up was 4 ± 2.8 years. There was no hospital mortality, and NYHA was reduced from 3 ± 0.5 to 1 ± 0.7 ($p < 0.0001$), the LVOT gradient from 88 ± 35 to 20 ± 18 mmHg ($p < 0.0001$), mitral valve regurgitation from grade 3 ± 1 to 1 ± 0.7 ($p < 0.0001$), and septum thickness from 18 ± 3 to 14 ± 2 mm ($p < 0.0001$).

Conclusions The mitral valve apparatus contributes with all its components variably to LVOT dynamic obstruction thus surgical correction in addition to extended myectomy is recommended to achieve the best outcome.

Keywords Hypertrophic obstructive cardiomyopathy • Mitral valve repair • Aortic approach • Mitral approach • Systolic anterior motion • Extensive myectomy

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Introduction

Transaortic septal myectomy [1] is the most commonly used surgical technique to treat hypertrophic obstructive cardiomyopathy (HOCM), and is associated with reduction of the outflow gradients and low operative morbidity and mortality [2,3]. In the modern era, the role of the mitral valve [4] in the pathophysiology of HOCM has been addressed [5–8]. Initially, left ventricle outflow tract (LVOT) obstruction was thought to be the result of a Venturi effect produced by septal hypertrophy involving the mitral valve [9]. More recently, a ''drag'' [10] rather than ''suck'' [9] mechanism was proposed: the flow acceleration around the hypertrophied septum pushes the anterior mitral leaflet (AML) into the LVOT, causing systolic anterior motion (SAM). Mitral valve apparatus malformations causing SAM–related LVOT obstruction can occur even in patients with no or moderate septal hypertrophy [8]. These include leaflets' elongation and a wide array of malformations of the papillary muscles (PM) and chordae [10], which can be detected by echocardiography and cardiac magnetic resonance [11,12]

In this study we sought to assess the role of the mitral valve in LVOT obstruction, and the operative results of its surgical treatment in a retrospective observational study.

Material and Methods

Study Design and Statistical Methods

The study was approved by our Institutional Research Review Board. All demographic, preoperative, intraoperative and postoperative variables were analysed. Surgical techniques were recorded. Follow-up information (survival, New York Heart Association (NYHA), LVOT gradient, major adverse cardiac and cerebrovascular events) was gathered routinely by the outpatient clinic, telephone calls or the treating general physician. Preoperative and follow-up image findings (transthoracic, transoesophageal and stress echocardiograms and magnetic resonance) were recorded. Patients' consents were obtained before the operation. Endpoints included the involvement of the mitral valve in the LVOT obstruction and the effectiveness of HOC surgery (mortality, changes in clinical and echocardiographic characteristics). In order to assess the endpoints, mitral valve surgery (any procedures involving the mitral valve and subvalvular apparatus), 30-day and hospital survival, reduction in NYHA, LVOT gradient, septum thickness and residual mitral regurgitation before and after surgery were evaluated. Long-term survival was evaluated with the survival at the last follow-up. Continuous variables are expressed as mean, standard deviation, and range; categorical variables are expressed as absolute value and percentage. The comparison between NYHA, LVOT gradient, septum thickness, and residual mitral regurgitation (MR) before and after surgery were assessed by paired T-test. Data management and analysis were done with SAS version 9.4 (SAS Institute Inc., Cary, NC, USA). All p-values of <0.05 were considered statistically significant.

Surgical Technique

All operations were performed by a single surgeon (MP), through a full sternotomy, with aorta and bicaval cannulation and intermittent antegrade cold blood cardioplegia, and with mild hypothermia (32 °C). All patients underwent a septal resection through the aortic valve. Myectomy was performed starting at nadir of the right coronary sinus, and extended apically to achieve the exposure of the PM bases. All anomalous muscular trabeculae were resected. Muscular resection was extended toward the lateral ventricular wall up to the left trigone. Transaortic resection of fibrotic, thickened and agglutinated secondary chordae tendineae was performed from the tip of the PM to the ventricular surface of the anterior mitral leaflet. The PM were carefully inspected to detect any hypertrophy, fusion, displacement, anomalies or aberrances (e.g., bifurcation and fibrosis). Hypertrophied and thick PM were split to increase mobility. Accessory PM were resected. In case of mitral leaflet repair, a left or right atrial approach was used. The repair was done after myectomy and the release of the AML achieved by the resection of secondary chordae (Video 1 in the online version at doi:10.1016/j.hlc.2018.02.006). Further procedures on cardiopulmonary bypass were performed if necessary. After weaning from cardiopulmonary bypass and before protamine administration, a pharmacological stress test (inotropes) was performed in all patients to assess the residual LVOT gradient, SAM, and residual MR by transoesophageal echocardiography. Intracardiac pressures were measured with needles placed directly into the aorta and the LV, and provocative manoeuvers were used to elicit outflow tract gradients as necessary. The mean cardiopulmonary bypass and aortic cross-clamp times were 86 ± 27 minutes and 62 ± 22 minutes, respectively.

Results

Patients' Characteristics

From March 2007 to November 2016, 28 consecutive patients with HOCM underwent surgery. Indications for surgery were peak LVOT gradient $\geq$50 mmHg at rest or during stress, and persistence of symptoms despite optimal medical therapy [13]. The mean age was 58 ± 11 years (range 29–76), 13 males and 15 females. All were symptomatic in NYHA class II (4), III (20), or IV (4), with a mean NYHA of 3 ± 0.5. One patient underwent a second reoperation at our institution because of failure of previous surgeries performed elsewhere. None underwent previous alcohol septal ablation.

All patients underwent preoperative echocardiogram at our institute. The mean left ventricle end-diastolic and end-systolic diameters were 42 ± 5 mm (range 32–50 mm) and 27 ± 5 mm (range 20–36 mm), respectively. The mean interventricular septum thickness was 18 ± 4 mm (range 11–27 mm). Intraventricular peak gradient at rest was 86 ± 34 mmHg (range 42–174 mmHg). Mitral regurgitation was grade <3+/4+ in 11 patients (40%) and grade $\geq$3+/4+ in 17 patients (60%). The mean grade was 3 ± 1.

Ten patients underwent cardiac magnetic resonance before the operation. The average ejection fraction and interventricular septum thickness were 72 ± 5% (range 62–79) and 22 ± 6 mm (range 14–36). The mean left ventricle end-diastolic and end-systolic volume indexes were 90 ± 17 ml/m^2 (range 67–120) and 27 ± 9 ml/m^2 (range 16–42), total mass (myocardial wall mass + papillary muscles mass) and myocardial wall mass indexes were 116 ± 60 gr/m^2 (range 48–202) and 111 ± 57 gr/m^2 (range 45–193), respectively. Specific findings included accessory PM and abnormal mitral leaflet length. The average PM mass index was 7.3 ± 3.8 gr/m^2. Myocardial late gadolinium enhancement was present in five patients (50%), 1.3 ± 1.9% of total myocardial mass. Papillary muscles delayed enhancement was not detected.

Echocardiographic Insights

Twenty-two patients (78%) had resting SAM resulting in dynamic LVOT obstruction (Figures 1 A-B-C and 1S and Video 2 in the online version at doi:10.1016/j.hlc.2018.02.006). Intrinsic mitral valve disease was identified preoperatively in 16 operations (57%) and this included leaflets elongation (six patients; Figures 1 A-B-C and 1S), PM abnormalities (16 patients; Figure 1 D-E-F), leaflets fibrosis, abnormal chordae going from the posterior leaflet (PML) to the inferior wall or from the AML to the interventricular septum (seven patients, Figure 1 G-H), and hypertrophic apical-basal MT (bands of tissue from the apex to the basal segment of either the septum or the anterior wall, 11 patients) (Figure 1 I). The average length of the AML and PML were 27 ± 3 mm and 18 ± 3 mm, respectively.

Intraoperative Pathoanatomic Findings and Additional Procedures to Myectomy (Table 1)

Structural valve abnormalities were discovered through intraoperative transoesophageal echocardiography or direct valve inspection. Secondary chordae tendineae tractioning the AML to interventricular septum and SAM were seen in 22 (78%) (Figure 2); PM malformations in 14 (50%), including 7 (25%) PM hypertrophied and fused (Figure 3 A-B); MT hypertrophy was detected in 10 (35%). Secondary mitral valve chordae resection was performed in 26 (3%); PM and MT resection in 20 (71%); main PM splitting and elongation in 8 (28%) (Figure 3 C-D); mitral valve repair was performed in 9 patients (32%). Further chordae resections and PM splitting were performed in order to reduce the traction of the AML, even if morphologically normal.

Procedures on Mitral Valve Leaflets

Among the nine patients who underwent procedures on mitral valve leaflets, six involved the PML, and three the AML. In these patients, the mean interventricular septum thickness was 17 ± 2 mm. Redundancy and excessive tissue of the PML was the most common finding, requiring surgical resection and shortening (Figure 2S and Video 1 and Video 2 in the online version at doi:10.1016/j.hlc.2018.02.006). Transaortic AML plication and transposition of secondary chordae to the free edge (working functionally as primary chordae) were performed in one case, respectively. In one patient, after the resection of chordae to the AML and weaning from cardiopulmonary bypass, a significant MR due to prolapse (scallop A3) was detected. The prolapse, due to a cutting of the primary chordae, was repaired by a plication of the scallop using interrupted polypropylene 4/0 stitches.

Combined Procedures

Aortic valve replacement with biological prosthesis because of rheumatic combined stenosis/regurgitation aortic valve disease was performed in one patient. Coronary artery bypass grafting on the left anterior descending was done in one patient, and on the right coronary artery in another.

Early Reoperation

One patient, at the beginning of our experience, in 2007, underwent early reoperation after a complicated postoperative course. A mechanical mitral prosthesis for recurrent SAM and severe MR was implanted on postoperative day 55. The patient was discharged home 6 days after the reoperation.

Postoperative Course

No intraoperative, in-hospital, or 30-day mortality occurred. The mean postoperative length of stay was 12 ± 10 days (median 9 days). Thirteen patients (46%) had uneventful postoperative course. None had complete atrioventricular block, but two patients (7%) required pacemaker implantation because of tachy-brady syndrome. Surgical revision for bleeding, atrial fibrillation and ventricular tachycardia occurred in two (7%), four (14%) and one (3%) patient, respectively. Pancreatitis and pulmonary embolism occurred in one patient.

Follow-Up and Late Events

Follow-up was complete for all patients; mean follow-up time was 5 ± 2.8 years at 28 February 2018. A late death occurred in one patient due to pneumonia 717 days after surgery. This patient underwent reoperation 97 days after the HOCM surgery because of endocarditis on aortic prosthesis and pacemaker lead. At latest follow-up the NYHA class was I in 18/27 (66%), II in 6/27 (22%), and III in 3/27 (11%); the mean NYHA was 1 ± 0.7.

At the last available follow-up, echocardiography showed 1+/4+ or 2+/4+ MR in all survivors, with a mean MR grade 1 ± 0.6 ($p < 0.0001$), no SAM, a mean maximum intraventricular gradient at rest of 21 ± 18 mmHg ($p < 0.0001$), a mean interventricular septum thickness of 14 ± 2 mm ($p < 0.0001$), and a mean ejection fraction of 63 ± 6%.

Discussion

Our 10-year experience shows that: 1) HOCM is a wide-spectrum disease in which septal hypertrophy and the mitral valve with all its components contribute to the dynamic LVOT obstruction; 2) the subvalvular mitral

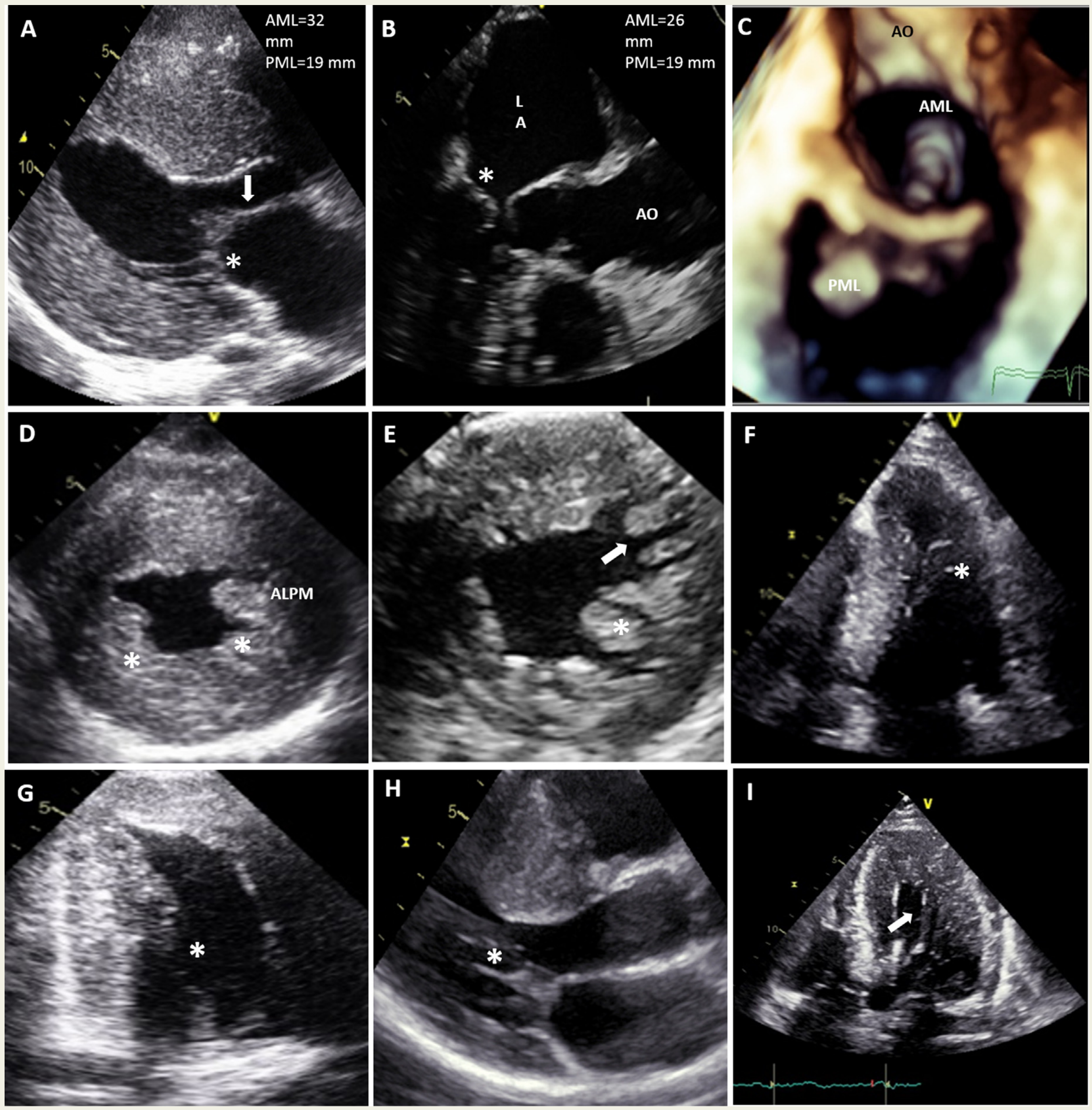

Figure 1 Systolic anterior motion (SAM) of mitral valve obstructing the left ventricle outflow tract (LVOT). Transthoracic parasternal (panel A) and transoesophageal long-axis views (panel B) showing SAM due to anterior mitral leaflet elongation (arrow), further displaced toward the left ventricular septum by an equally elongated posterior leaflet (*). Panel C: 3D real time transoesophageal echocardiogram may best show anterior leaflet obstructing LVOT. Panel D: short-axis view showing anterior fusion broadening the base of the PM attachment to the underlying left ventricular wall (*). Of note, the anterior papillary muscle (PM) is hypertrophic. Panel E: antero-lateral hypertrophic and bifurcated PM (*). Two small accessory anterior PM (arrow). Panel F, apical 4-chamber view: apically displaced anterior PM (*) promoting abnormal intra-ventricular gradient. Panel G, apical 2-chamber view: restricted and fibrotic secondary chordae (*) of both the posterior and anterior leaflets (Panel H, parasternal long axis view) limiting the excursion and inducing an anterior displacement of the coaptation point. Panel I, apical 4-chamber view: accessory apical-basal muscle trabeculae (arrow).
Abbreviations: LA, left atrium; AO, aorta; AML, anterior mitral leaflet; PML, posterior mitral leaflet; ALPM, antero-lateral papillary muscle.

apparatus (chordae and PM) promote SAM and contribute to the obstruction; 3) a combined surgical approach, involving the septal myectomy through the aortic valve and the correction of the mitral valve anomalies, achieves the best outcome; 4) iatrogenic damage of the mitral valve can occur during subvalvular apparatus remodelling; 5) the surgical remodelling of the subvalvular apparatus does not impair the mitral valve function at mid-term follow-up; and 6) a dedicated HOCM team (dedicated cardiologists, radiologists and surgeons) with complete preoperative cardiac

Table 1 Pathoanatomic findings and their surgical treatment in addition to myectomy in a series of 28 consecutive patients with HOCM.

Pt	ECHO				Intraoperative pathoanatomic findings				Procedures		
	IVS	ALL	PLL	SAM	PM malformation	MT hypertrophy	Pathologic chordae	MV	II chordae MV resection	MV plasty	PM or MT resection
1	19			x					x		
2	21			x	x			P1-P2 cleft	x	P3 flail and cleft correction	x
3	16			x	x		x	P1 redundancy	x	P1 resection + edge to edge	x
4	21			x		x	x		x		x
5	15			x		x	x		x		
6	14				x	x	x		x		x
7	18			x	x			P2-P3 cleft	x	cleft correction	x
8	11	21	18			x					x
9	27	28	15	x	x[1]		x		x		x[2]
10	21	29	17	x	x[1]	x	x		x	AML plication[*]	x[2]
11	27				x	x	x		x		x
12	22	25	19	x					x		
13	16	22	11				x		x		
14	15	27	18	x			x		x		x[2]
15	16	23	15	x			x		x		
16	14	24	14	x			x		x		
17	18	24	23	x		x	x		x	transposition II chordae AML[*]	x
18	19	28	16	x			x		x		
19	20	26	18	x		x	x		x	AML repair because of iatrogenesis	x
20	15	22	20	x	x[1]		x		x		x[2]
21	17	24	17								
22	20	24	16	x	x[1]		x		x		x[2]
23	16	26	19	x	x[1]		x	P2 redundancy	x	P2 resection and shortening	x[2]
24	16	32	19		x		x		x		x
25	22	33	22	x	x	x	x		x		x
26	16	32	22	x	x[1]		x	P2 redundancy	x	P2 resection and shortening	x[2]
27	17	32	19	x		x	x	P2 redundancy	x	P2 resection and shortening	x
28	16	28	19	x	x[1]		x	P1-P2 cleft	x		x[2]

Abbreviations: HOCM, hypertrophic obstructive cardiomyopathy; IVS, interventricular septum (mm); ALL, anterior leaflet length (mm); PLL, posterior leaflet length (mm); SAM, systolic anterior motion; SMS, subaortic membrane stenosis; PM, papillary muscles; MT, muscular trabeculae; MV, mitral valve; AML, anterior mitral leaflet.

[1]Main PM hypertrophied and fused.

[2]Main PM splitting and elongation.

[*]Through the aortic valve.

imaging is crucial for planning the most appropriate surgical strategy.

The classical transaortic approach allows accurate analysis of the interventricular septum. Moreover, after septal bulge resection, the aberrancies of the PM, increased length of the AML, and thickened secondary chordae tendineae should be identified and corrected.

In our series, septal myectomy was performed in all cases, confirming the well-defined role of this technique in HOCM [1–3,14,15]. The 0.4% (17/3695 patients) of the composite

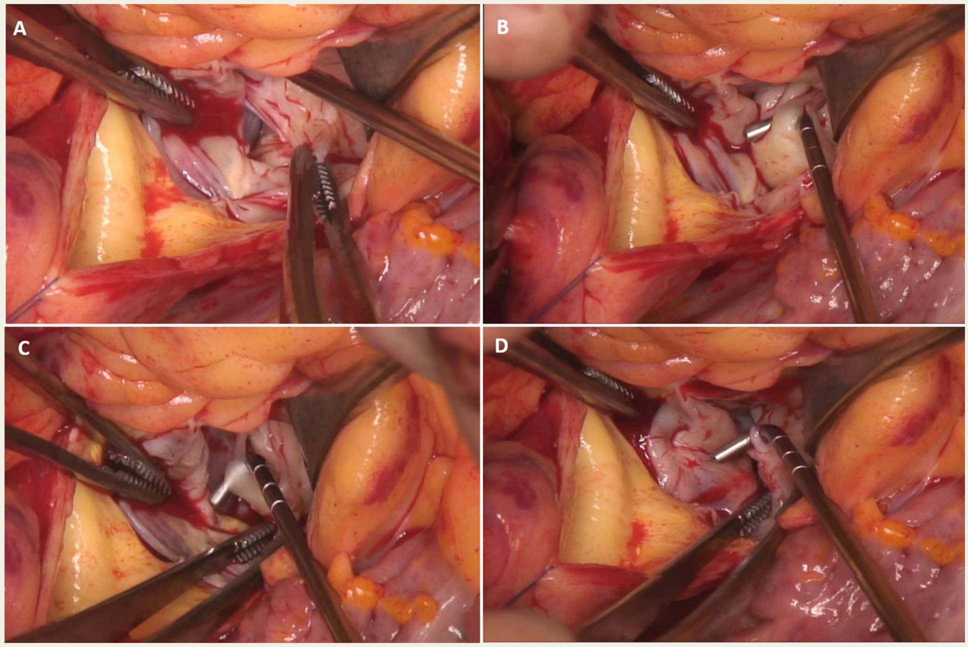

Figure 2 Transaortic left ventricle outflow tract inspection. Panel A: analysis of subvalvular mitral valve apparatus. Panel B-C-D: secondary chordae anchoring the anterior leaflet of the mitral valve to the septum were identified and resected. The chordae appeared thickened and retracted.

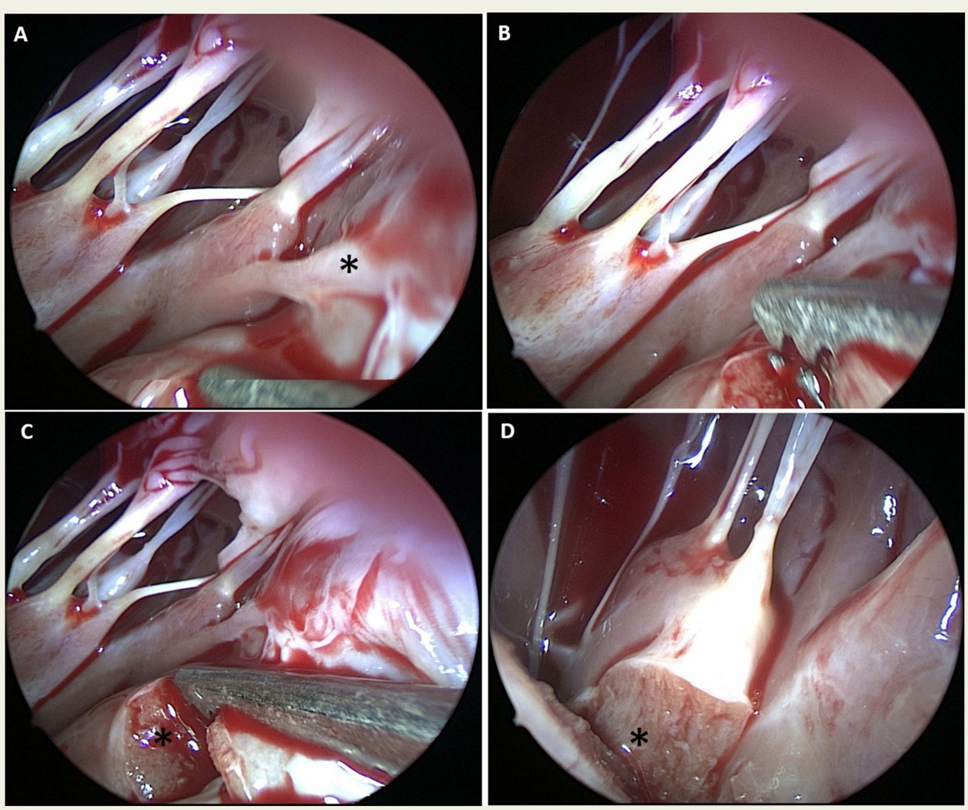

Figure 3 Papillary muscle (PM) aberrancy. Panel A-B: hypertrophy of the PM and secondary chordae thickened. A muscular connection between the PM head and left ventricle free wall (*) was identified. Panel C: a muscle trabecula connecting the left ventricle wall to the PM was resected (*). Panel D: the PM was split at the end of the procedure. Transaortic view using a fiberscope.

operative mortality from five major high-volume centres in North America highlights the role of dedicated HOCM units [15]. In Europe [14], a review of 124 consecutive patients with heart failure symptoms due to HOCM undergoing extended left ventricular septal myectomy revealed a low (<1%) early operative mortality, and a reduction in outflow tract gradients and SAM-mediated MR.

From our surgical experience, we report special pathoanatomic findings.

Abnormal PM Morphology

Papillary muscles displacement, bifid PM [16], hypertrophy, anterior fusion, and inward orientation have been frequently observed in patients with LVOT obstruction [8,10,12,17]. An anterior and basilar displacement of the base of the anterolateral PM and abnormal muscular connections with the anterolateral wall, inserting into or near the A1 scallop of the mitral valve, are the most common pathoanatomic findings [8]. Bifid and hypermobile PM increase the obstruction even without significant basal septal hypertrophy [16], and PM hypertrophy occurs in more than half of patients with HOCM [18]. The relief of PM fusion and the resection of anomalous chordae tendinae (false chordae) and fibrous–muscular attachments between the PM and ventricular septum or free wall [14], in addition to an extended septal myectomy, improves surgical outcomes [19] and reduces the risk of reoperation [20]. In our experience, the PM malformations causing dynamic LVOT obstruction occurred frequently and were surgically treated with the transaortic approach. In addition to the wide septal myectomy [21], we 1) released the anterolateral PM by extending the resection laterally into the free wall above its base and thinning the hypertrophied heads, and 2) divided and excised with scissors and lancet all the attachments between the PM head and the left ventricle free wall, which became more evident after the septum resection. To reduce the traction of the AML toward the septum, PM splitting may further explicitly facilitate the ejection stream.

Secondary Chordae Anomaly

Thickened and retracted secondary chordae that insert beyond the free margin and rough zone of the AML may contribute to dynamic obstructive pathophysiology, lifting and tenting the AML anteriorly toward the LVOT [7], and predisposing to SAM [8]. Their role in obstruction, especially in the relatively thin septum, has been recently demonstrated by Ferrazzi [7]. The anomalous chordae resection (a median of 3, range 1–8) associated with a shallow myectomy in 39 patients (with a ventricular septal thickness $\leq$19 mm) showed better clinical and haemodynamic results compared with a control group (only myectomy, 29 patients). Conventional surgery jeopardised the repair of the mitral valve and increased the need for prosthetic rings, compared with transaortic chordal cutting. During 23 $\pm$ 2 months of follow-up, 1/39 patients showed MR $\geq$3. Secondary chordae cutting was performed in almost all patients in our series, and after 4 years of follow-up this has not altered mitral valve function. However, one patient required intraoperative repair of an AML prolapse due to erroneous resection of primary chordae, revealing the complexity of the anatomy of the LVOT in HOCM patients.

Mitral Leaflet Elongation

One third of patients with hypertrophic cardiomyopathy present a primary increase in mitral leaflet length [11], and this is even more pronounced in HOCM [8], where the protruding leaflets increase the risk of SAM. Patients with HOCM usually present with an AML greater than 30 mm [22]. Thus, surgical correction could be reasonable. The outcomes after horizontal plication, aimed at shortening and stiffening the AML, in addition to the myectomy and PM release, the so called resect-plicate-release strategy, have been reported in 252 patients with HOCM [8,22]. The elegant algorithm guiding this surgical strategy, which shows 0.4% in both operative mortality and early reoperation and 0.6% in pacemaker implantation, is reported by Sherrid [8]. Vriesendorp reported the benefit of AML extension in addition to myectomy in 98 patients with HOCM [5], with an average AML length of 34 mm. The procedure stiffens the midportion of the AML and is carried out through the transaortic approach. No hospital mortality was reported, and during the long-term follow-up (8 $\pm$ 6 years), the patients experienced significant symptomatic and haemodynamic improvement. The rate of pacemaker implantation, need for reoperation for patch dehiscence, and residual obstruction were 4%, 2%, and 1%, respectively. Three patients underwent further mitral valve replacement during follow-up.

Isolated elongation of the PML can anteriorly push the coaptation with the AML, causing SAM, mitral-septal contact, and LVOT obstruction [8,10]. Dulguerov [6] reported a three-step procedure (transaortic septal resection, transmitral septal resection through the AML, which is detached and reconstructed using autologous pericardial patch, and PML resection followed by rigid mitral valve annuloplasty) to treat 16 patients with HOCM and a PML more than 20 mm long. This procedure is aimed at, 1) completing and extending the septum resection down to the ventricular apex through the mitral valve, and 2) posteriorly moving the coaptation plane of the mitral valve away from the septum, increasing the AML area, and restricting the PML motion (with PML resection and annuloplasty). The excellent mid-term surgical results, despite a high rate of pacemaker implants (18%) occurring at the beginning of their experience, clearly address the role of the PML in the pathogenesis of LVOT obstruction.

In our series, we adopted several techniques to treat the mitral valve during HOCM surgery, and both the AML and PML required procedures. We suggest a length of the PML greater than 18 mm for consideration for plication/resection in HOCM surgery. The release of the AML by the transaortic subvalvular apparatus remodelling, and the reduction in

PML length we report, based on the same pathophysiologic concepts of ‘‘resection-release” [8] and ‘‘moving posteriorly the coaptation plane of the mitral valve” [6], achieved the complete resolution of LVOT obstruction. Differently from others [6], the transaortic approach enables us to reach the ventricular apex without further surgery on the AML, and we avoid the annuloplasty ring as a risk factor for SAM [23]. Pacemaker implantation rate was consistent with data from other single-centre report [2,3,5,6,19]. Our research highlights the complex anatomic interactions in HOCM, and adds further issues to the debate over surgical myectomy or alcohol septal ablation, though the use of the latter has increased in Europe in recent decades [13,14]. This report is also consistent with several other reports from the United States [15], Europe [5,6] and Italy [7,14] showing the dramatic and definitive benefits with septal myectomy in experienced centres with evolving surgical programs [14,15]. The preoperative planning, based on individual anatomic findings, and the tailored surgical treatment, resulted in an excellent outcome.

This is a single-centre series of a small number of consecutive patients retrospectively reviewed. Though grouped, our study population showed a heterogeneity of HOCM morphology. The lack of a control group with different forms of treatment (isolated myectomy, alcohol septal ablation and medical treatments) and the non-obstructive hypertrophic cardiomyopathy is a major limitation of this descriptive study. Missing data (e.g., type of HOCM and hypertrophy localisation, genetic mutations, medications, resected LV mass and CMR findings) is a further limitation.

Conclusion

The mitral valve, particularly its subvalvular apparatus, substantially contributes to LVOT obstruction in patients with HOCM. Thus, its surgical correction in addition to extended myectomy is recommended during surgery. Surgeons with expertise in mitral valve anatomy and extensive repair techniques, guided by a dedicated team for planning the proper operative strategy, can help guarantee the best operative results. Long-term follow-up data are needed to assess the effects of the mitral subvalvular remodelling during HOCM surgery on mitral valve function.

Disclosures

The authors declare no funding sources and actual or potential competing financial interests.

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