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NEW TECHNIQUE OF ANTERIOR MITRAL LEAFLET PLICATION FOR CORRECTION OF SAM SYNDROME IN OBSTRUCTIVE HYPERTROPHIC CARDIOMYOPATHY. CLINICAL CASE

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Abstract

Background and Objective: A variety of surgical techniques have been developed to correct systolic anterior motion (SAM) of the mitral valve, typically performed in conjunction with septal myectomy for obstructive hypertrophic cardiomyopathy (oHCM). However, many of these approaches carry a substantial risk of postoperative functional complications. We present a novel technique for plication of the elongated mitral anterior leaflet designed to reduce left ventricular outflow tract (LVOT) obstruction and associated mitral regurgitation while preserving leaflet geometry and function, ultimately improving heart failure symptoms.

Materials and methods: The proposed technique is performed through an aortotomy and consists of targeted plication of the elongated A2 segment and its chordae, creating a controlled duplication that eliminates excess leaflet length without distortion. This approach restores physiologic coaptation of the mitral leaflets. A single plication loop is used for shortening up to 5 mm, whereas multiple interrupted sutures are applied for greater lengths.

Results: A 68-year-old female patient with progressive obstructive HCM, SAM syndrome, NYHA Class III functional status, and stage C heart failure (ACC/AHA) underwent anterior mitral leaflet plication using the described method combined with septal myectomy. The intervention significantly reduced the postoperative peak LVOT gradient (from 98 mmHg to 15 mmHg) and resolved mitral regurgitation (from grade III to grade 0).

Conclusion: This initial clinical experience with targeted anterior mitral leaflet plication for SAM correction in obstructive HCM suggests that the technique is both effective and safe in the short term. It substantially decreases the severity of SAM and mitral regurgitation while preserving valve function. Further research is required to define optimal patient selection and assess long-term outcomes.

Keywords: *obstructive hypertrophic cardiomyopathy; systolic anterior motion (SAM); mitral regurgitation; mitral valve plication.*

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Резюме

НОВЫЙ СПОСОБ ПЛИКАЦИИ ПЕРЕДНЕЙ СТВОРКИ МИТРАЛЬНОГО КЛАПАНА ДЛЯ КОРРЕКЦИИ SAM-СИНДРОМА ПРИ ОБСТРУКТИВНОЙ ГИПЕРТРОФИЧЕСКОЙ КАРДИОМИОПАТИИ. КЛИНИЧЕСКИЙ СЛУЧАЙ

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Предпосылки и цель: Существует широкий спектр методов хирургической коррекции SAM-синдрома, выполняемых в сочетании с септальной миоэктомией при обструктивной гипертрофической кардиомиопатии. Однако данные вмешательства относятся к группе высокого риска развития послеоперационных функциональных нарушений. Мы предложили метод пликации удлиненного митрального клапана, который может уменьшить обструкцию выходного тракта левого желудочка (LVOT) и сопутствующую митральную регургитацию, тем самым

способствуя улучшению симптомов сердечной недостаточности, при этом сохранив функциональное состояние створок.

Материалы и методы: Предлагаемый метод выполняется через аортотомный доступ и предусматривает точечную пликацию удлинённого сегмента А2 и его хорд с формированием дупликатуры, что позволяет устранить избыточную длину створки без её деформации. Техника обеспечивает восстановление нормальной кооптации митральных створок и применяется с использованием одной пликационной петли при удлинении до 5 мм, так и с несколькими узловыми швами при большей протяжённости.

Результаты: Пациентке 68 лет с диагнозом гипертрофическая кардиомиопатия, обструктивный тип, прогрессирующая форма; SAM-синдром; ХСН 3ФК (NYHA), стадия С (ACC/AHA) проведена пликация передней створки митрального клапана по предложенному методу в сочетании с септальной миоэктомией. Процедура снизила послеоперационный максимальный градиент выносящего тракта левого желудочка (LVOT obstruction) (с 98 мм рт. ст. до 15 мм рт. ст.) и степень митральной регургитации (с III степень до 0).

Выводы: Первый опыт применения точечной пликации передней створки митрального клапана для коррекции SAM-синдрома при обструктивной гипертрофической кардиомиопатии показывает, что данный метод может быть эффективным для облегчения симптомов за счёт уменьшения выраженности SAM и митральной регургитации, оставаясь при этом безопасным с точки зрения краткосрочных функциональных исходов. Требуются дальнейшие исследования для уточнения целевых групп пациентов и оценки долгосрочных клинических результатов.

***Ключевые слова**: гипертрофическая кардиомиопатия, обструктивная форма, систолическое переднее движение (SAM), митральная регургитация, пликация.*

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Түйіндеме

ОБСТРУКТИВТІ ГИПЕРТРОФИЯЛЫҚ КАРДИОМИОПАТИЯДАҒЫ SAM-СИНДРОМДЫ ТҮЗЕТУГЕ АРНАЛҒАН МИТРАЛЬДЫҚ ҚАҚПАҚШАНЫҢ АЛДЫҢҒЫ ЖАПЫРЫҒЫН ПЛИКАЦИЯЛАУДЫҢ ЖАҢА ӘДІСІ. КЛИНИКАЛЫҚ ЖАҒДАЙ

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Кіріспе және мақсаты: Обструктивті гипертрофиялық кардиомиопатия кезінде септальды миоэктомиямен бірге орындалатын SAM-синдромды хирургиялық түзетудің кең ауқымды әдістері бар. Алайда бұл араласулар операциядан кейінгі функционалдық бұзылыстардың жоғары қаупімен байланысты. Біз митральдық қақпақшаның ұзарған алдыңғы жапырғысын пликациялаудың жаңа әдісін ұсынамыз, ол сол жақ қарыншаның шығу жолының (LVOT) обструкциясын және оған байланысты митральды регургитацияны азайтуға мүмкіндік береді, жүрек жеткіліксіздігі белгілерін жеңілдетеді және жапырғы функциясын сақтайды.

Материалдар мен әдістер: Ұсынылған әдіс аортотомиялық қолжетімділік арқылы жүзеге асырылады және ұзарған А2 сегменті мен оның хордаларына нүктелі пликация жүргізуді қамтиды. Дупликатура түзу арқылы жапырғының артық ұзындығын деформациясыз жоюға болады. Техника митральдық жапырғылардың қалыпты коаптациясын қалпына келтіреді және ұзындығы 5 мм-ге дейін болғанда бір пликациялық ілмекпен, ал одан ұзын болғанда бірнеше түйінді тігістермен орындалады.

Нәтижелері: 68 жастағы әйел пациентке «обструктивті түрдегі гипертрофиялық кардиомиопатия, үдемелі нысаны; SAM-синдром; ЖСЖ III ФК (NYHA), С сатысы (ACC/AHA)» диагнозымен ұсынылған әдіс бойынша митральды қақпақшаның алдыңғы жапырғысына пликация жасалды, процедура септальды миоэктомиямен біріктірілді. Операциядан кейін сол жақ қарыншаның шығу жолындағы максималды градиент 98 мм с.б.-дан 15 мм с.б.-ға дейін төмендеді, митральды регургитация III дәрежеден 0 дәрежеге дейін жойылды.

Қорытынды: Обструктивті гипертрофиялық кардиомиопатиядағы SAM-синдромды түзету үшін митральдық қақпақшаның алдыңғы жапырғысының нүктелі пликациясын қолданудың алғашқы тәжірибесі бұл әдістің SAM мен митральды регургитация айқындылығын төмендету арқылы симптомдарды жеңілдетуде тиімді әрі қысқа мерзімді функционалдық нәтижелер тұрғысынан қауіпсіз екенін көрсетеді. Пациенттердің мақсатты топтарын нақтылау және ұзақ мерзімді нәтижелерді бағалау үшін қосымша зерттеулер қажет.

Түйінді сөздер: *обструктивті гипертрофиялық кардиомиопатия, систолалық алдыңғы қозғалыс (SAM), митральды қақпақша жетіспеушілігі, пликация.*

Дәйексөз үшін:

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Introduction

Hypertrophic cardiomyopathy (HCM) is an autosomal dominant disorder affecting approximately 1 in 500 individuals [5,10]. It is characterized by asymmetric myocardial hypertrophy, most commonly involving the basal portion of the interventricular septum, and is frequently accompanied by abnormalities of the mitral valve and subvalvular apparatus [7]. The obstructive form of HCM (oHCM) is defined by narrowing of the left ventricular outflow tract (LVOT) due to dynamic obstruction. The principal pathophysiological mechanism underlying this obstruction is systolic anterior motion (SAM) of the anterior mitral leaflet, which further narrows the LVOT and is frequently associated with mitral regurgitation (MR) [3].

In many patients with oHCM, the mitral leaflets are elongated compared to those of healthy individuals [1]. Typically, the elongated portion of the anterior leaflet extends beyond the coaptation plane, a feature central to the development of SAM [4]. These anatomical abnormalities of the mitral apparatus are increasingly recognized as a phenotypic expression of HCM and contribute to LVOT obstruction, particularly in patients whose basal septal thickness is only moderately increased [2]. During late diastole and early systole, blood flow is directed against the protruding leaflet, generating aerodynamic forces that displace it toward the septum [16]. Once contact occurs between the anterior leaflet and the interventricular septum, further systolic displacement into the outflow tract is driven by intraventricular pressure, thereby exacerbating the obstruction [17].

Transaortic septal myectomy (Morrow procedure), first introduced in 1968, remains the gold standard for surgical management of oHCM [21]. This technique – consisting of resection of the basal interventricular septum – achieves high rates of success in reducing gradients, improving symptoms, and ensuring long-term survival, with low operative mortality [14]. However, residual SAM and MR persist in a subset of patients after myectomy, underscoring the importance of mitral valve correction as an adjunctive or independent surgical intervention. Elongation of the anterior mitral leaflet is one of the major determinants of persistent SAM following septal reduction. Studies demonstrate that residual MR and SAM remain in approximately 10–15% of patients, tripling the risk of future reinterventions due to recurrent severe MR [15].

Multiple techniques for mitral valve modification have been described. Vertical plication (McIntosh–Cooley) reduces leaflet width by creating a vertical fold but often results in substantial leaflet deformation and impaired coaptation, leading to central MR [11]. Horizontal plication (Swistel), used when the anterior leaflet length exceeds 30 mm or 17 mm/m² of body surface area as part of the RPR (resection-plication-release) strategy, shortens leaflet height but also causes significant tissue trauma and may induce central regurgitation [4]. The edge-to-edge (Alfieri) repair effectively prevents SAM by approximating the A2 and P2 scallops [9], yet it is associated with progressive postoperative mitral stenosis. Retention plasty according to Hetzer provides additional fixation of leaflet edges, preventing anterior leaflet displacement [12], but requires extended exposure and is often followed by mixed dysfunction (stenosis and regurgitation).

Contemporary surgical management of oHCM increasingly emphasises individualised treatment strategies. In patients with pronounced mitral valve pathology, plication or shortening of the leaflets may be added to septal myectomy to address both the septal and valvular components contributing to SAM [19]. This approach aims to prevent persistent or recurrent obstruction after isolated septal reduction.

Given this anatomical and clinical heterogeneity, developing a plication technique for the anterior mitral leaflet that effectively eliminates SAM, improves leaflet coaptation, and avoids postoperative dysfunction is of substantial clinical importance. In this study, we introduce a new surgical method involving precise plication of the A2 segment of the anterior mitral leaflet. This localized technique targets only the elongated region, aiming to reduce SAM and improve hemodynamics without requiring extensive myectomy or complex mitral reconstructions.

Methods / Surgical Technique

Approbation of the Method

Clinical testing of the proposed mitral valve plication technique was performed in the Department of Cardiac Surgery with the Laboratory of Cardiopulmonary Bypass and the operating suite of the Research Institute of Cardiology and Internal Diseases (Almaty) from April 2023 to December 2024.

Technique of Anterior Mitral Leaflet Plication

The procedure is performed under standard conditions. A median sternotomy is followed by normothermic cardiopulmonary bypass using a single, dual-stage venous cannula placed through the right atrium. The left atrium is vented via the right superior pulmonary vein. Cardiac arrest is achieved with cold blood cardioplegia administered antegrade following transverse transection of the ascending aorta. Subsequent doses of cardioplegia are delivered selectively after aortotomy.

The aorta is opened obliquely in a transverse direction toward the base of the non-coronary sinus. Through the aortotomy, with gentle displacement of the aortic valve leaflets, the elongated portion of the anterior mitral leaflet and its elongated chordae – corresponding to the A2 scallop – is visualized (Fig. 1).

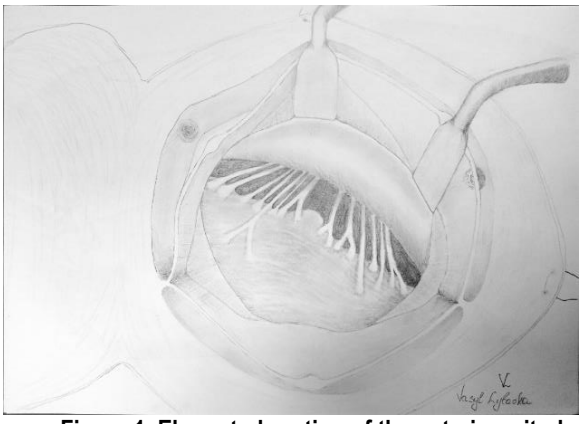

Figure 1. Elongated portion of the anterior mitral leaflet (A2 scallop) with lengthened chordae.

To correct the elongated segment of A2 that protrudes beyond the coaptation plane during systole, together with its elongated chordae, a targeted plication technique is applied. Using a 5-0 monofilament suture, the free edge of A2 is first pierced from the ventricular side toward the coaptation side at a point adjacent to the elongated chorda insertion. The second needle is similarly passed through the elongated chorda region on the opposite side. Both suture ends are then returned from the coaptation side toward the ventricular side, traversing the free edge of the leaflet over a narrower span. Subsequently, both sutures are passed superiorly through the body of the leaflet near the attachment of secondary chordae (Fig. 2).

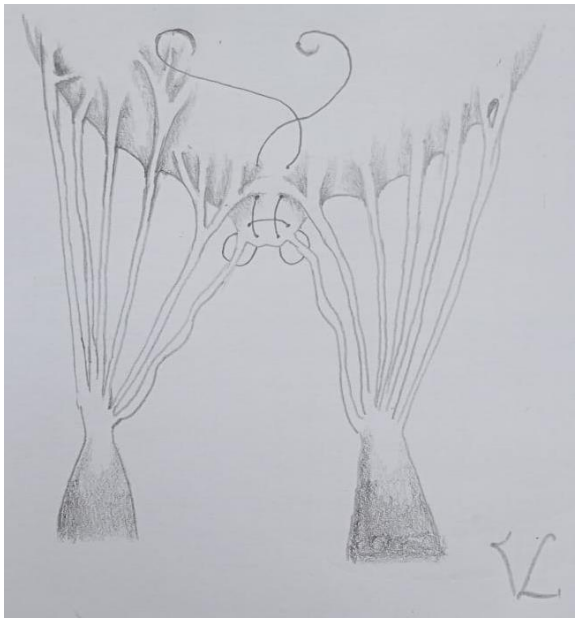

Figure 2. Formation of a plication loop on the elongated portion of the A2 scallop of the anterior mitral leaflet.

The suture loop is then tightened until the free edge approximates the leaflet body, creating a duplication fold (Fig. 3).

This single-loop plication technique is applied when the elongated segment measures up to 5 mm. For segments exceeding 5 mm (Fig. 4), separate interrupted sutures are placed according to the same principle.

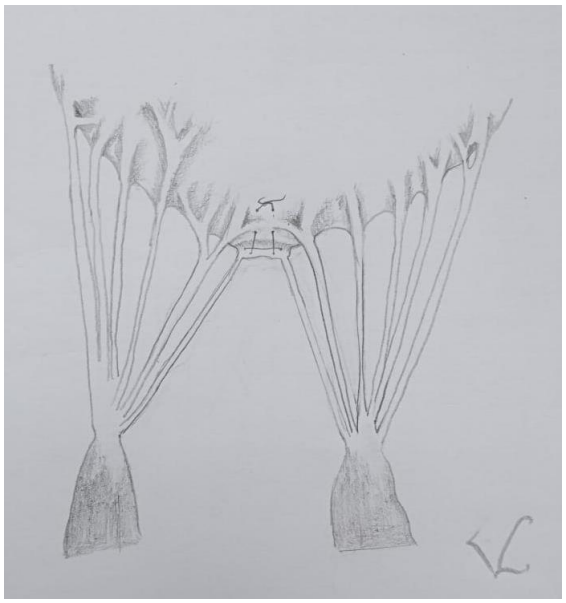

Figure 3. Plication of the elongated portion of the A2 scallop of the anterior mitral leaflet with tensioning of the elongated chordae.

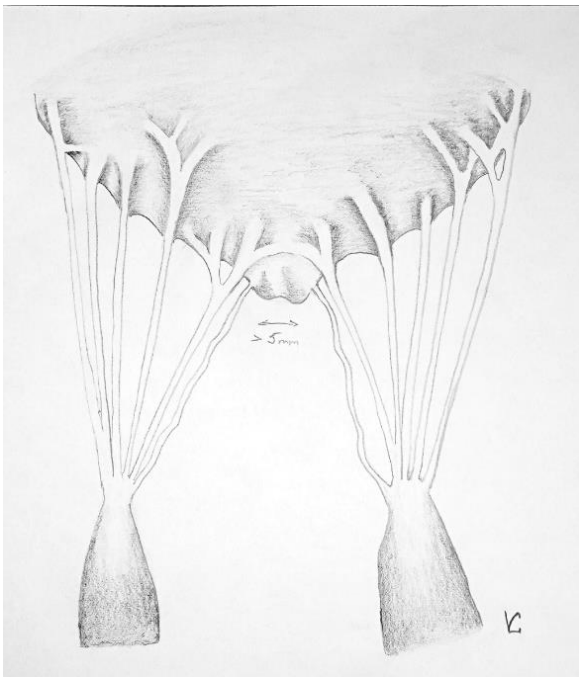

Figure 4. The elongated portion of the A2 scallop of the anterior mitral leaflet measuring greater than 5 mm in width.

Clinical Case

Patient S., a 68-year-old woman, was admitted to the Cardiac Surgery Department of the Research Institute of Cardiology and Internal Diseases with the following diagnosis: obstructive hypertrophic cardiomyopathy, progressive form; SAM syndrome; chronic heart failure, NYHA Class III, Stage C (ACC/AHA).

Preoperative echocardiography demonstrated severe left ventricular outflow tract (LVOT) obstruction with a peak gradient of 98 mmHg and grade III mitral regurgitation (Fig. 5c). A key pathophysiological feature in this patient was marked anterior displacement and elongation of the anterior mitral leaflet, leading to systolic anterior motion (Fig. 5a).

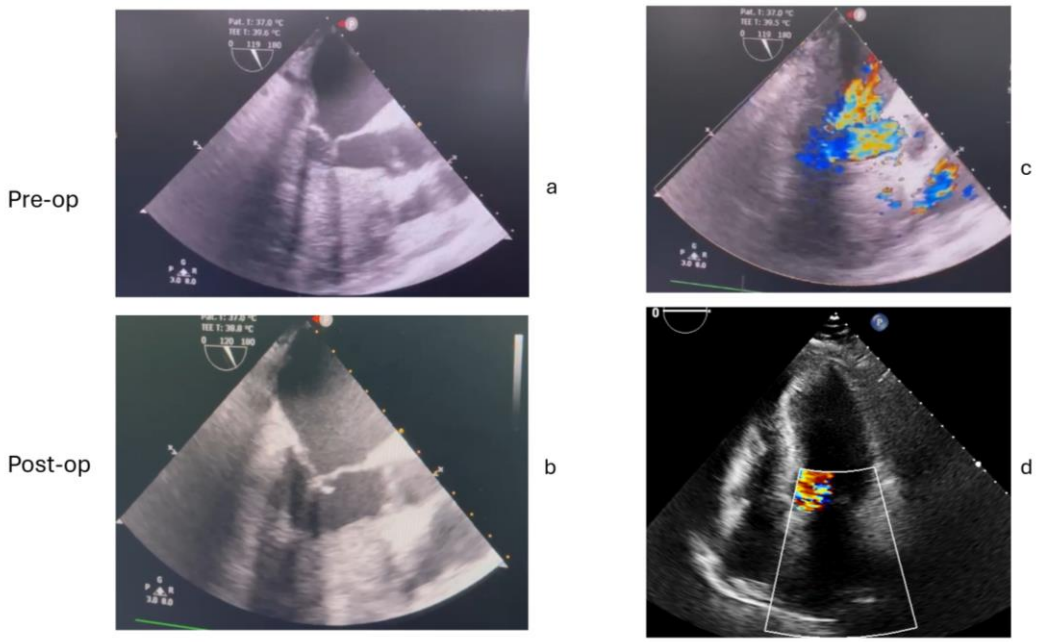

Figure 5. Echocardiographic images in obstructive hypertrophic cardiomyopathy before and after surgical correction: (a, b) systolic anterior motion (SAM); (c, d) mitral regurgitation (MR).

Given these anatomical characteristics, plication of the anterior mitral leaflet became a critical component of the operation. This intervention eliminated excessive leaflet mobility, restored proper coaptation, and significantly reduced the LVOT gradient when combined with septal myectomy. The patient was included in the cohort undergoing surgical correction of SAM syndrome using the proposed anterior mitral leaflet plication technique.

Ethics approval and consent to participate

The report was conducted in accordance with institutional ethical standards. Approval for publication of this case was obtained from the Bioethical Commission of Kazakh National Medical University named after S.D. Asfendiyarov (Protocol #1071, dated March 31, 2021), and written informed consent was provided by the patient.

Results

Clinical Case Description and Surgical Technique

The operation was performed via median sternotomy under normothermic cardiopulmonary bypass, using a single dual-stage venous cannula inserted through the right atrium. The left atrium was vented via the right superior pulmonary vein. Cardioplegic arrest was induced using cold blood cardioplegia administered antegradely after transverse transection of the ascending aorta. Subsequent doses of cardioplegia were delivered selectively following aortic dissection.

The ascending aorta was opened obliquely in a transverse direction toward the base of the non-coronary sinus. Revision and evaluation of the AML and its chordae were performed through the aortic root. At this stage, septal myectomy was carried out to reduce left ventricular outflow tract obstruction.

After confirming the presence of an elongated portion of the A2 segment of the AML with elongated chordae, targeted plication was performed. Using a 5-0 monofilament suture, the free edge of the A2 segment was first pierced from the ventricular side toward the coaptation plane at a point adjacent to the attachment of the elongated chord. The second needle was then passed in a similar manner through the elongated chord on the opposite side. Both suture ends were returned from the coaptation side toward the ventricular side, piercing the free edge over a narrower span. Subsequently, both sutures were passed superiorly through the leaflet body near the secondary chordal attachment. The plication loop was then tightened until the free edge approximated the leaflet body, creating a duplication of the elongated segment. The operation was completed in the standard manner.

Postoperative Outcomes

In the early postoperative period, echocardiography demonstrated complete resolution of SAM syndrome (Fig. 5b) and a significant reduction of mitral regurgitation to grade 0 (Fig. 5d). The LVOT gradient decreased from the preoperative value of 98 mmHg to 15 mmHg, reflecting effective relief of the obstruction. Echocardiographic assessment indicated restoration of mitral leaflet coaptation and stabilization of left ventricular function in the early postoperative phase.

Discussion

In our study, we describe a novel method for eliminating systolic anterior motion (SAM) in a patient with obstructive hypertrophic cardiomyopathy, aiming not only to abolish SAM but also to preserve maximal valvular and ventricular function in the postoperative period. The mechanism of action of the proposed A2 segment plication of the anterior mitral leaflet is based on targeted reduction of the protruding free-edge portion and stabilization of the coaptation line. The creation of a duplicate fold limits excessive anterior displacement of the anterior mitral leaflet into the left ventricular outflow tract, thereby reducing SAM and concomitant mitral regurgitation. The principal advantage of this technique is the establishment of a more stable coaptation line with minimal chordal tension while maintaining leaflet mobility, which minimizes the risk of

postoperative mitral stenosis. The procedure is simple, reproducible, and does not require complex reconstructions or implantation of additional material, facilitating standardization and broader clinical application.

Surgical correction of SAM, performed adjunctively with septal myectomy, remains a subject of ongoing debate. Despite advances in surgical techniques, the optimal approach to mitral valve correction continues to be discussed. For instance, recent data from Carvalho et al. indicate that anterior leaflet length does not always correlate directly with residual LVOT gradients or survival following isolated myectomy [8]. Conversely, other studies demonstrate that plication or "shortening" of the leaflets yields durable results, including long-term reduction of LVOT gradients, decreased MR, and favorable survival [19].

A retrospective analysis of 57 patients with oHCM who underwent combined sectoral myectomy and anterior leaflet retention plasty (ALRP) demonstrated significant reductions in LVOT gradient and septal thickness, while minimal MR was preserved in 87% of patients, and SAM was completely eliminated [22]. The main limitations of this study included its retrospective design, absence of a control group, requirement for left atrial access, and potential technical challenges in cases with complex subvalvular anomalies. These results support the efficacy of ALRP as an adjunct to myectomy; however, further studies are required to assess long-term safety and generalizability.

Interventions targeting the anterior mitral leaflet in SAM, including Resect-Plicate-Release (RPR) and AML shortening, have been shown to reduce valve length, decrease the protruding portion, eliminate LVOT gradients and SAM, reduce MR, and improve NYHA functional class [4,19]. Overall advantages of these approaches include verified echocardiographic efficacy, clinical improvement, and high procedural safety. Limitations include short- to medium-term follow-up, technical variability, retrospective study designs, and procedure-specific risks such as leaflet injury, thrombosis, or recurrent regurgitation. These findings support the development of point-specific plication techniques for the A2 AML to optimize SAM correction.

In a recent study, combining myectomy with targeted mitral valve correction (including anterior leaflet folding and interventions on the subvalvular apparatus) significantly reduced the incidence of residual SAM and MR compared to isolated myectomy: 5% vs 28% for SAM and 0% vs 15% for MR in a randomized comparison [6]. The main limitations of this study were its retrospective design and small, single-center sample size.

In a larger cohort treated with the "three-step" RPR method – comprising extended myectomy, horizontal AML plication, and correction of abnormal papillary muscle attachments - it was demonstrated that adjunctive AML plication markedly reduces residual SAM and MR while maintaining low operative mortality and favorable clinical outcomes [20].

Research on surgical correction of the anterior mitral leaflet as a primary treatment for patients with oHCM remains limited to case series and small observational studies. In the study by *Sorajja P. et al.,* non-surgical percutaneous (catheter-based) plication of the mitral valve was performed in five patients who were initially candidates for myectomy, aiming to reduce SAM and mitral regurgitation [18]. Overall, most studies have focused on the role of plication as an adjunct to septal myectomy [13,19,20].

Strengths and Limitations

The main strength of this study is the demonstration of the safety and efficacy of A2 segment plication of the anterior mitral leaflet in a specific clinical case. Careful intraoperative assessment of the mitral valve and chordae allowed precise identification of areas requiring correction and the establishment of a stable coaptation line postoperatively.

However, several limitations must be acknowledged. The findings are based on a single clinical observation, which limits the generalizability of the results to a broader patient population. Prospective studies with larger patient cohorts and long-term follow-up are necessary to confirm the reproducibility of the technique and the durability of its outcomes. Additionally, individual anatomical variations of the mitral valve may limit the effectiveness of plication, particularly in cases with markedly elongated AML or atypical chordal configurations. Despite these limitations, the described technique demonstrates potential as a targeted and safe strategy for correcting SAM in oHCM.

The proposed A2 segment plication is particularly indicated in patients with mitral-dependent LVOT obstruction, prolonged AML, and pronounced SAM. Limitations include the width of the elongated segment (>5 mm), potential variability in surgical technique among operators, and the lack of long-term outcome data. Further prospective studies with larger cohorts and extended follow-up are required to validate the safety and effectiveness of this method. Nevertheless, A2 segment plication shows substantial potential as a stand-alone or adjunctive procedure to correct SAM and reduce the risk of recurrent mitral regurgitation in patients with advanced AML elongation.

Conclusion

The proposed technique of mitral valve A2 segment plication effectively eliminates SAM syndrome, restores coaptation of the mitral valve leaflets, and preserves valvular function, thereby optimizing the clinical outcomes of surgical treatment in patients with obstructive hypertrophic cardiomyopathy.

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