Surgical Septal Myectomy for the Treatment of Residual Left Ventricular Outflow Tract Obstruction Following Failed Alcohol Septal Ablation

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Summary

The reasons of residual left ventricular outflow tract (LVOT) obstruction following alcohol septal ablation (ASA) remain unclear, and outcomes of myectomy following failed ASA remain underreported.

Thirteen symptomatic patients (10 women, a median age of 60.0 years) who underwent septal myectomy following failed ASA were reviewed. The patients were followed up for a median of 6 months. The clinical characteristics and outcomes of these patients were analyzed and were compared with those of 178 patients who underwent isolated myectomy without previous ASA at our institution during the same period.

In the first ASA procedure, the median number of septal perforator arteries injected was 1.0 with the median value of peak creatine kinase following ablation of 978.5 U/L. Uncontrollable extent and location of infarcted myocardium caused by ablation and mitral subvalvular anomalies were found in four (30.8%) and seven (53.8%) patients, respectively. No operative or follow-up deaths occurred. The median maximum LVOT gradients fell from preoperative 112.0 to 8.5 mmHg at follow-up (P < 0.001). Compared with controls, patients with failed ASA had a higher proportion of mitral subvalvular anomalies (53.8% versus 13.5%, P = 0.001) and developed a higher incidence of complete atrioventricular block following myectomy (15.4% versus 1.7%, P = 0.038).

Low institutional or operator experience with ablation, uncontrollable extent and location of infarcted myocardium caused by ablation, and mitral subvalvular anomalies may be reasons for failed ASA. Surgical myectomy for the treatment of residual LVOT obstruction after unsuccessful ASA may be associated with favorable results.

Key words: Mitral subvalvular anomalies, Complete atrioventricular block

Hypertrophic cardiomyopathy (HCM) is a genetic myocardial disease with the characterization of asymmetrical left ventricular hypertrophy.¹ Up to 70% of patients with HCM may progress to hypertrophic obstructive cardiomyopathy (HOCM), which is characterized by left ventricular outflow tract (LVOT) obstruction at rest or after provocation² and is frequently associated with a worse prognosis.³

In HOCM with accompanied refractory symptoms, septal reduction therapy is indicated.⁴ Septal myectomy has become the preferred treatment for the relief of LVOT obstruction.⁵ In contrast, percutaneous alcohol septal ablation (ASA), which creates a transmural infarction in the basal septum after injection of alcohol into the septal perforator branch, has emerged as a less invasive alternative to myectomy.⁶ Worldwide, and particularly in Europe, ASA has eclipsed myectomy for the treatment of HOCM in terms of annual procedures.⁷ ASA results in symptomatic and hemodynamic improvement in the majority of patients with HOCM. However, in a subset of patients, recurrent symptoms with residual hemodynamic obstruction occur. The rate of recurrence of severe symptoms following ASA is reported to be approximately 20%.⁸ The most recent registration trials have reported that the percentage of patients who are lost at gradient/symptomatic follow-up or suffering obstruction requiring repeated septal reduction therapy is 15%-18%.⁹,¹⁰ In a sizable proportion of patients with recurrent severe symptoms and residual obstruction following ASA, myectomy may be considered in lieu of a second ablation.¹¹
Patients who undergo myectomy due to residual obstruction and recurrent severe symptoms following ASA continue to increase. However, the reasons of recurrent severe symptoms and residual obstruction after ASA remain unclear. In addition, surgical treatment for this subgroup of patients may be challenging, and regarding studies are scarce. This study reviews our single institution’s data of patients with HOCM who initially received ASA and subsequently underwent myectomy because of recurrent severe symptoms and residual obstruction and aims to explore possible reasons for residual obstruction after unsuccessful ASA and to evaluate the results of septal myectomy for the treatment of residual obstruction after ASA.

Methods

Patients: Between January 2015 and December 2019, consecutive patients with HOCM who initially underwent ASA and subsequently required isolated transaortic septal myectomy were reviewed. Inclusion criteria included (1) the maximum LVOT pressure gradient ≥ 50 mmHg at rest or with provocation and (2) severe limiting symptoms refractory to pharmacologic therapy with non-vasodilating β-blockers and/or calcium channel blockers. Patients with LVOT obstruction associated with hypertensive heart disease were excluded. This study excluded patients with septal hypertrophy secondary to severe aortic valvular stenosis.

Preoperative evaluation: Routine transthoracic echocardiographic (TTE) evaluation was used to determine ventricular septal anatomy and thickness, LVOT gradient, systolic anterior motion (SAM) of the mitral valve (MV), and mitral subvalvular anomalies. Mitral subvalvular anomalies included papillary muscle abnormalities and abnormal MV chordal attachments (abnormal chordal attachments and fibrotic and retracted secondary chordae). Cardiac magnetic resonance was used to evaluate the degree of scar tissue left behind and judge the extent of resection required. Coronary artery angiography was performed before myectomy in all included patients, even if the ASA procedure was conducted recently (e.g., 3 months ago). Additionally, 24-hour Holter was required to determine intermittent atrioventricular conduction abnormalities, ventricular tachycardia, or atrial fibrillation.

Study protocol: This study protocol was approved by the ethics committee of Zhongshan Hospital Fudan University (No. Y2020-029) and was consistent with the Declaration of Helsinki. All included patients signed an informed consent approved by the ethics committee. Data collection was performed by trained staff (two people). The trained staff, however, were not informed of the purpose of this study.

Baseline and surgical characteristics and perioperative results of patients with failed ASA (the study group) were obtained from our institutional database and were analyzed. Patients were regularly followed up at 3 and 6 months following surgery and in 6-month intervals thereafter. Follow-up data were obtained through clinic visits, WeChat, or telephone interviews with patients or their families. Additionally, a control group consisted of all patients with a resting LVOT gradient ≥ 50 mmHg who underwent transaortic septal myectomy without previous ASA at our institution during the same period. Perioperative variables were compared between the study and control groups.

Surgical procedures: The patient was gently placed in the reverse Trendelenburg and left lateral decubitus position. After a median incision with sternotomy, cardiopulmonary bypass was performed using ascending aorta and right atrium cannulation with a left ventricular vent placed via the right superior pulmonary vein. Through a low oblique aortotomy approximately 7-10 mm above the right coronary ostium, the aortic valve leaflets were pulled up to gain access to the outflow tract. The extent of infarcted myocardium was difficult to assess at the time of surgery. Identification of scar tissue was beneficial to guide the resection. During surgery, by virtue of direct inspection of septal anatomy, the operating surgeon customized the resection purely according to individual differences. Scalpel resection was usually started at the nadir of the right cusp, 5 mm below the aortic valve and extending leftwards to the left tricuspid. The area of septal excision was then deepened and lengthened beyond the level of the mitral papillary muscles toward the apex. The depth of resection varied according to individual characteristics. In patients with failed ASA, a limited septal myectomy was directed to the right so as to avoid creation of a ventricular septal perforation or of complete atroventricular block. Mitral subvalvular anomalies, including fibrotic and retracted secondary chordae inserted on the anterior mitral leaflet body, abnormal chordae tendineae attached to the ventricular septum or free wall (false cords), and direct papillary muscle insertion into the anterior mitral leaflet, were also corrected.

After resections were completed, the origin of the papillary muscles should be seen by looking through the incision of the aortic root. The left ventricle was then liberally irrigated with saline to remove any residual debris. Immediately after the discontinuation of cardiopulmonary bypass, TEE was used to determine the provoked gradient and the severity of mitral regurgitation (MR). Provocation was frequently conducted by isoproterenol infusion via a deep venous catheter to achieve a heart rate > 120 beats per minute or induce premature ventricular contraction via mechanical stimulation of the right ventricle during the TEE examination. A repeat procedure was immediately performed if the provoked gradient > 30 mmHg and/or moderate or more MR were present.

Statistical analysis: Statistical analysis was performed with the SPSS statistical package version 22.0 (SPSS Inc., Chicago, IL, USA). Continuous variables were expressed as the median with an interquartile range (IQR) and categorical variables as percentages. The Wilcoxon rank sum test was used to compare continuous variables between baseline and postoperative values in the study group and between the study and control groups. The Fisher exact test was used to compare categorical variables between the study and control groups. A two-sided P-value < 0.05 was considered statistically significant.
## Results

### Study population: A total of 255 patients with HOCM underwent septal myectomy with or without other cardiac procedures at our institution between January 2015 and December 2019. Of these, 13 (5.1%) patients (10 women, 3 men), and direct papillary muscle insertion into the annulus; N, no; NYHA, New York Heart Association (classification); RBBB, right bundle branch block; SAM, systolic anterior motion; Sub-MV anomalies, mitral valve subvalvular anomalies; Y, yes; and Uncontrollable-IR, uncontrollable extent and location of infarcted regions.

### Possible reasons for failed ASA: MV-level residual obstruction was observed in seven (53.8%) patients. Mitral subvalvular anomalies determined by TTE (as shown in Figure 1) was found in all seven patients with no deficiency in MV itself, including abnormal chordal attachments (two patients), fibrotic and retracted secondary chordae inserted on the anterior mitral leaflet body (three patients), and direct papillary muscle insertion into the anterior mitral leaflet.

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### Table II. Baseline and Surgical Characteristics

| Variables                                      | Value |
|------------------------------------------------|-------|
| Number of patients                             | 13    |
| Age (years)                                     | 60.0 (51.0–63.0) |
| Gender (Females/Males)                         | 10/3  |
| Previous ASA procedures                        | 14    |
| Elapsed time from ASA to surgery (months)       | 26.0 (16.0–38.0) |
| Concomitant diseases                            |       |
| Hypertension                                    | 4 (30.8%) |
| Coronary artery disease                        | 1 (7.7%) |
| Family history of HCM                          | 2 (15.4%) |
| Family history of sudden death                  | 1 (7.7%) |
| NYHA functional class                           |       |
| II                                              | 1 (7.7%) |
| III                                             | 7 (53.8%) |
| IV                                              | 5 (38.5%) |
| Preoperative medication                         |       |
| β-blockers                                      | 10 (76.9%) |
| Calcium-channel blockers                       | 6 (46.2%) |
| Preoperative electrocardiogram                  |       |
| AF                                              | 1 (7.7%) |
| RBBB                                           | 4 (30.8%) |
| Preoperative TTE data                           |       |
| Interventricular septal thickness (cm)          | 1.8 (1.7–2.0) |
| Maximum LVOT gradient (mmHg)                    | 112.0 (88.0–119.0) |
| Severity of MR                                  | 3.0 (3.0–4.0) |
| SAM                                             | 13 (100%) |
| Left ventricular ejection fraction (%)          | 69.0 (66.0–69.0) |
| Left ventricular endo-diastolic diameter (mm)   | 42.0 (40.0–45.0) |
| Left atrial diameter (mm)                       | 48.0 (45.0–51.0) |
| Possible reasons for failed ASA                 |       |
| Mitral subvalvular anomalies                    | 7 (53.8%) |
| Abnormal chordal attachments                    | 2     |
| Fibrotic and retracted secondary chordae        | 3     |
| PM abnormalities                                | 2     |
| Uncontrollable extent and location of IR        | 4 (30.8%) |
| Transaortic septal myectomy alone               | 6 (46.2%) |
| ACC time (minutes)                              | 31.0 (28.0–42.0) |
| Myectomy plus MV subvalvular procedures         | 7 (53.8%) |
| ACC time (minutes)                              | 35.0 (29.0–44.0) |

Values are expressed as n (%) or median and interquartile range. ACC indicates aortic cross-clamping; AF, atrial fibrillation; ASA, alcohol septal ablation; HCM, hypertrophic cardiomyopathy; IR, infarcted regions; LVOT, left ventricular outflow tract; MR, mitral regurgitation; NYHA, New York Heart Association (classification); PM, papillary muscle; RBBB, right bundle branch block; SAM, systolic anterior motion; and TTE, transthoracic echocardiography.

Cardiac magnetic resonance imaging (as shown in Figure 2) showed that in three patients, infarcted regions were located in the targeted hypertrophic septum, but the infarcted areas were smaller than the hypertrophic zone and located more inferiorly, and in one other patient, infarcted regions were located outside the targeted areas. In addition, one other patient with a baseline interventricular septal thickness of 2.9 cm and a baseline gradient of 168 mmHg received an ASA procedure via a large proximal first septal perforator artery. In the patient with massive septal hypertrophy, the maximum LVOT gradient was reduced to 83 mmHg following ablation with the interventricular septal thickness of 2.7 cm.

**Perioperative and follow-up results:** Immediately following myectomy, TEE showed that the median maximum LVOT gradient decreased to 9.0 (IQR, 5.0-16.0) mmHg, which was significantly lower than the preoperative value (P < 0.001). The median septal thickness was significantly smaller than that preoperatively (1.4 cm versus 1.8 cm, P < 0.001). Although SAM occurred in two patients immediately following myectomy, the degree of SAM decreased significantly. Severity of MR decreased to 1.0 postoperatively (versus 3.0 preoperatively, P < 0.001), with no occurrence of moderate or more MR.

There was no operative mortality. Two (15.4%) patients, including one patient with right bundle branch block after ASA and before myectomy, developed complete atrioventricular block following myectomy and received permanent pacemaker implantation. One (7.7%) pa-
Table III. Echocardiography Data

|                          | Before ASA | After ASA | Before myectomy | After myectomy | Follow-up |
|--------------------------|------------|-----------|-----------------|----------------|-----------|
| Septal thickness (cm)    | 2.0 (1.9–2.2) | 1.9 (1.8–2.1) | 1.8 (1.7–2.0) | 1.4 (1.3–1.5)́ | 1.4 (1.3–1.5)́ |
| Gradient (mmHg)          | 91.5 (81.8–105.0) | 58.0 (50.5–67.3)́ | 112.0 (88.0–119.0) | 9.0 (5.0–16.0)́ | 8.5 (5.0–14.0)́ |
| SAM                     | 13 (100%)   | 10 (76.9%) | 13 (100%)       | 2 (15.4%)́      | 2 (15.4%)́ |
| Severity of MR          | 3.0 (3.0–4.0) | 3.0 (3.0–3.0) | 3.0 (3.0–4.0) | 1.0 (1.0–1.0)́ | 1.0 (1.0–1.0)́ |
| EF (%)                  | 71.0 (67.0–73.5) | 69.5 (66.0–70.5) | 69.0 (66.0–69.0) | 68.0 (65.0–69.0) | 68.0 (65.5–68.5) |

Values are expressed as n (%) or median and interquartile range. ́P < 0.05 for data after ASA versus data before ASA; ́P < 0.05 data (after myectomy or follow-up) versus data before myectomy. ASA indicates alcohol septal ablation; EF, ejection fraction; MR, mitral regurgitation; and SAM, systolic anterior motion.

Figure 1. Mitral subvalvular abnormalities. The echocardiographic image shows fibrotic and retracted secondary chorda inserted on the anterior mitral leaflet body (where the white arrow is pointing). LV indicates left ventricle; LA, left atrium; AO, aorta; and IVS, interventricular septum.

Figure 2. Cardiovascular magnetic resonance images in short-axis plane demonstrated on the late gadolinium enhancement image. A black arrow shows the infarcted myocardial region, which appears white, and the normal myocardium appears dark. LV indicates left ventricle; and RV, right ventricle.

tent developed new-onset atrial fibrillation, and another one (7.7%) developed ventricular tachycardia. Other postoperative morbidity is listed in Table IV. All 13 patients recovered smoothly with the median postoperative hospital length of stay of 6 days.

All 13 patients received a follow-up visit with a median duration of 6.0 (IQR, 6.0–12.0) months. During the follow-up periods, all patients survived with significant improvement of quality of life with recovery from symptoms and NYHA functional class I–II. No instances of re-operation for recurrent obstruction and/or for symptomatic MR were recorded. No late permanent pacemaker implantation was observed. As shown in Table III, the median maximum LVOT gradient at the latest follow-up dropped from the preoperative value (median, 8.5 mmHg versus 112.0 mmHg, P < 0.001), with no occurrence of residual obstruction (the maximum gradient > 30 mmHg). The severity of MR at the latest follow-up significantly decreased to 1.0 (versus 3.0 preoperatively, P < 0.001), with no occurrence of moderate or more MR. SAM was identified in two patients at the latest follow-up. And the two patients were diagnosed with mild MR and were categorized as NYHA class II, and dynamic evaluation was continued.

Comparison with controls: Baseline characteristics and outcomes of 178 patients without previous ASA who underwent isolated transaortic myectomy at our institution during this same period are listed in Table V. Comparatively, patients with failed ASA were more likely to have preoperative right bundle branch block (30.8% versus 1.7%, P < 0.001). Importantly, patients with failed ASA were more likely to have mitral subvalvular anomalies (53.8% versus 13.5%, P = 0.001). Remarkably, patients with failed ASA had a higher incidence of complete atrioventricular block (15.4% versus 1.7%, P = 0.038). No significant difference between the two groups was observed regarding operative mortality (P = 0.786).

Discussion

This study showed that low institutional experience or operator experience with ablation may result in the failure of ASA. In this series, 12 patients with failed ASA who underwent ASA procedures with the median number of septal perforator arteries injected of 1.0 had a median peak creatine kinase after ablation of 978.5 U/L, with the maximum value of 1282 U/L, and all 12 patients had residual gradients of > 30 mmHg with the minimum value of 31 mmHg. The low number of septal perforator arteries (only 1.0) suggested inappropriate selection of target septal perforator arteries and insufficient alcohol ablation, which may be associated with low institutional experience or operator experience with ablation. Inappropriate selection of target septal perforator arteries, insufficient alcohol ablation, and related low creatine kinase, as well as high...
residual gradients, might lead to the failure of ASA. This result suggested that low institutional experience or operator experience with ablation may be an important reason for failed ASA. Our result was in line with previous experience with ablation may be an important reason for failed ASA.17) Our result was in line with previous studies that have reported that patients with residual obstruction and recurrent symptoms following ASA may be those with a peak creatine kinase following ablation of < 1300 U/L and a residual gradient following ablation of > 25 mmHg.13,14)

In the earliest Mayo Clinic study including 16 patients with failed ASA, ElBardissi, et al.15) found that most failures in their series were judged to be due to unfavorable coronary anatomy, i.e., a large proximal first septal perforator artery was absent or did not allow introduction of a cannula. Subsequently, Nagueh, et al.15) reported that the cannulated septal arteries failure to supply the culprit septal segments may be an underlying mechanism of failed ASA. These two reports suggested that ASA was strongly limited by the coronary anatomy, which may vary significantly between individuals. In a recent study of Mayo Clinic, Quintana, et al.10) found that although the ethanol reached the culprit region of obstruction, the depth of the septal muscle after ASA was heterogeneous, leading to unpredictable and potentially suboptimal therapeutic effects. In this series, uncontrollable extent and location of infarcted myocardium caused by ablation were found in 30.8% of patients with failed ASA, suggesting that uncontrollable extent and location of infarcted myocardium may be another reason for failed ASA. This finding was consistent with a previous study.20)

In addition, this study showed that mitral subvalvular anomalies may be a reason for failed ASA. The mechanisms of residual obstruction after failed ASA have been partially understood and described.21,24) Previous studies21,24) have demonstrated that abnormal chordal attachment was associated with dynamic LVOT obstruction independent of other factors, and other reports22,24) have stated that abnormal papillary muscle morphology was independently associated with LVOT obstruction. Although mitral subvalvular anomalies have been reported to be associated with LVOT obstruction, their role in the etiology of residual obstruction after failed ASA has rarely been reported. This study showed that mitral subvalvular anomalies including abnormal chordal attachments and papillary muscle abnormalities were found in more than half of patients with failed ASA. Furthermore, such patients underwent concomitant mitral subvalvular management during myectomy and then achieved favorable results. Importantly,

| Variable                                | Value                  |
|-----------------------------------------|------------------------|
| In-hospital                             | n = 13                 |
| Operative mortality                     | 0                      |
| Complete atrioventricular block         | 2 (15.4%)              |
| New onset of AF                         | 1 (7.7%)               |
| Ventricular tachycardia                 | 1 (7.7%)               |
| Complete left bundle branch block       | 5 (38.5%)              |
| Postoperative hospital stay (days)      | 6.0 (6.0–6.0)          |
| Follow-up                               | n = 13                 |
| Duration of follow-up (months)          | 6.0 (6.0–12.0)         |
| Survival                                | 13 (100%)              |
| Reoperation                             | 0                      |
| Re-ablation after myectomy              | 0                      |
| NYHA class at the latest follow-up     |                         |
| I                                       | 5 (38.5%)              |
| II                                      | 8 (61.5%)              |
| TTE data at the latest follow-up       |                         |
| Residual obstruction                    | 0                      |
| Moderate or more residual MR            | 0                      |
| SAM                                     | 2 (15.4%)              |

Values are expressed as n (%) or median and interquartile range. AF indicates atrial fibrillation; MR, mitral regurgitation; NYHA, New York Heart Association; SAM, systolic anterior motion; and TTE, transesophageal echocardiography.

| Variables                               | Failed ASA (n = 13) | Controls (n = 178) | P       |
|-----------------------------------------|---------------------|--------------------|---------|
| Age at the time of myectomy (years)     | 60.0 (51.0–63.0)    | 60.0 (48.8–66.0)   | 0.954   |
| Preoperative right bundle branch block  | 4 (30.8%)           | 3 (1.7%)           | < 0.001 |
| Preoperative NYHA class III–IV          | 12 (92.3%)          | 155 (87.1%)        | 0.583   |
| Preoperative TTE data                   |                     |                    |         |
| Septal thickness (cm)                   | 1.8 (1.7–2.0)       | 1.8 (1.6–2.0)      | 0.628   |
| Maximum LVOT gradient (mmHg)            | 112.0 (88.0–119.0)  | 93.0 (74.8–108.3)  | 0.428   |
| Severity of MR                         | 3.0 (3.0–4.0)       | 3.0 (3.0–4.0)      | 0.999   |
| Moderate or more MR                    | 10 (76.9%)          | 141 (79.2%)        | 0.845   |
| Mitral subvalvular anomalies           | 7 (53.8%)           | 24 (13.5%)         | 0.001   |
| Aortic cross-clamp time                 | 33.0 (29.0–43.5)    | 32.0 (28.0–42.0)   | 0.746   |
| TEE data after myectomy                 |                     |                    |         |
| Septal thickness (cm)                   | 1.4 (1.3–1.5)       | 1.4 (1.3–1.5)      | 0.888   |
| Maximum LVOT gradients                 | 9.0 (5.0–16.0)      | 12.0 (9.0–15.8)    | 0.511   |
| Severity of MR                         | 1.0 (1.0–1.0)       | 1.0 (1.0–1.0)      | 0.999   |
| Moderate MR                            | 0                   | 3 (1.7%)           | 0.637   |
| Operative mortality                    | 0                   | 1 (0.6%)           | 0.786   |
| Postoperative complete AVB             | 2 (15.4%)           | 3 (1.7%)           | 0.038   |

Values are expressed as n (%) or median and interquartile range. ASA indicates alcohol septal ablation; AVB, atrioventricular block; LVOT, left ventricular outflow tract; MR, mitral regurgitation; NYHA, New York Heart Association; TEE, transesophageal echocardiography; and TTE, transthoracic echocardiography.
compared with patients without previous ASA, patients with failed ASA were more likely to have mitral subvalvular anomalies. These results suggested that mitral subvalvular anomalies may play an important role in the etiology of residual obstruction after ASA (failed ASA). It can be inferred from the results of this study that mitral subvalvular anomalies may predict failure or poor outcomes with ASA. Thus, preoperative patient selection is of great importance, and patients with mitral subvalvular anomalies may be not good candidates for ASA. However, in China, the imbalances and differences of medical diagnosis and treatment among regions and medical institutions are objective realities. In some institutions, physicians found mitral subvalvular anomalies in patients with HOCM before making the decision on the treatment strategy, but they may be lacking of the awareness of the importance of mitral subvalvular anomalies and advised such patients to be treated with ASA. Some cardiologists thought that patients with HOCM may obtain benefit following ASA despite having mitral subvalvular anomalies. These factors may lead to a higher proportion of presence of mitral subvalvular anomalies in patients with HOCM with failed ASA. Because 10 (71.4%) ASA procedures in 9 patients were completed elsewhere, imaging data, especially MRI findings before ablation, were incomplete. In terms of our limited experience, mitral subvalvular abnormalities and a substantial SAM may be associated with insufficient outcome of ASA and symptom recurrence following ASA. However, with its limited sample size, our data contributed to accumulating evidence that the presence of mitral subvalvular anomalies may be a cause of failed ASA, and it remained to be confirmed in large cohort studies.

Another important finding of this study was that myectomy for the treatment of residual obstruction after failed ASA achieved favorable results. An increasing number of patients presented severe symptoms because of residual obstruction after ASA and required septal myectomy procedures, which then carried an increased risk of perioperative complications and life-long sequelae. In the earliest Mayo Clinic study including 16 patients, ElBardissi, et al. found that myectomy after failed ASA was associated with a higher incidence of morbidity and mortality. Subsequently, Nagueh, et al. just reported early outcome with no operative death, but the long-term outcome was not clear. In a recent study of Mayo Clinic, Quintana, et al. reported that advanced heart failure may more likely happen after myectomy at follow-up in patients with HOCM with previous ASA, but statistical significance did not differ. In the very few studies published to date, the outcomes after septal myectomy for the treatment of patients with HOCM with failed ASA were inconsistent. In this study, 13 patients with HOCM with residual obstruction after failed ASA underwent myectomy and then achieved favorable results, including no operative or follow-up deaths, significant improvement of quality of life with recovery from symptoms and NYHA functional class I-II, complete relief of LVOT obstruction, and reliable reduction of MR. Furthermore, the results were similar to the excellent outcomes achieved by myectomy for the treatment of patients with HOCM without previous ASA. Therefore, this study suggested that septal myectomy for the treatment of patients with HOCM with residual obstruction after failed ASA may be associated with favorable results.

Septal myectomy following failed ASA was believed to be more technically demanding. Identification of scar tissue helped in guiding the resection. The scars that were located in the septum facilitated tissue grasping and helped expose more distal septal areas. As a consequence of this, easier “en bloc” mobilization of muscle (by pulling it toward the aortic valve) facilitated a wide excision. The scars were frequently located in the subaortic part of the septum, so the septum in this region may be thin. Excessive subaortic resection may bring about iatrogenic ventricular septal perforation. Favorable results achieved by septal myectomy in this series suggested that septal myectomy may be a safe and effective therapy for residual obstruction after unsuccessful ASA.

Complete atrioventricular block was a major complication after septal myectomy following failed ASA. ASA typically produced a right bundle branch block (in 36% of the patients), whereas septal myectomy produced a left bundle branch block (in approximately 40% of patients). Given the development of left bundle branch block in many patients after myectomy, patients with right bundle branch block after ASA had a higher likelihood of complete atrioventricular block after myectomy. In terms of atrioventricular conductance, the risk of complete atrioventricular block after myectomy following failed ASA was reported to be between 10% and 33%. In this series, 30.8% of patients suffered from right bundle branch block after ASA and before myectomy, and 15.4% of patients developed complete atrioventricular block after the combination of myectomy following ASA, both of which were higher than in those cases where only surgery was performed. These high frequencies may have been attributed to the transmural infarct in the region of the right bundle branch caused by ASA, whereas septal myectomy involved excision of the superficial subendocardial tissue that contained the left bundle branch sheath. Although favorable results were achieved in this study, the high incidence of complete atrioventricular block should be paid particular attention to. This potential complication should be discussed with patients when treatment plans were being considered.

This study had some potential limitations. First, this was a relatively small cohort, and our analysis was limited by the small number of endpoints; thus, our findings should be confirmed in large cohort studies. Second, it was a single-center retrospective study that may subject to selection bias. Third, although multimodality imaging examinations including echocardiographic evaluation by experienced doctors and cardiac magnetic resonance imaging were performed frequently in patients with failed ASA, which may help to identify the presence of mitral subvalvular anomalies and uncontrollable extent and location of infarcted myocardium caused by ablation, the overwhelming majority of patients with HOCM received only echocardiographic evaluation prior to surgery at our institution. Cardiac magnetic resonance imaging did not serve as a regular examination modality at our institution. Fourth, a
functional capacity assessment tool, such as the 6-minute walking test, and a quality of life assessment tool, such as the SF-36 questionnaire, were not utilized in this study. Finally, the follow-up time was relatively short.

Conclusions

Low institutional experience or operator experience with ablation, uncontrollable extent and location of infarcted myocardium caused by ablation, and mitral subvalvular anomalies may be reasons for failed ASA. Surgical myectomy for the treatment of residual obstruction after failed ASA may be associated with favorable results.

Disclosure

Conflicts of interest: None.

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