Alcohol Septal Ablation for Hypertrophic Cardiomyopathy Guided by Intracoronary Myocardial Contrast Echocardiography to Reduce Myocardial Damage

CME Credits

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Abstract

Septal reduction therapy (SRT) is indicated for drug-refractory hypertrophic obstructive cardiomyopathy (HOCM). SRT includes surgical myectomy and alcohol septal ablation (ASA). The outcome between SRTs are similar except complete atrioventricular (AV) block. Intracoronary myocardial contrast echocardiography is used to minimize myocardial damage by ASA. We report a case of 40-year-old male who was diagnosed of HOCM with progressed symptoms under optimal medication. Echocardiography revealed peak velocity cross left ventricular outflow tract (LVOT) 5.3 m/s, systolic anterior motion (SAM) of mitral valve with eccentric mitral regurgitation (MR) and interventricular septal thickness 16 mm. Alcohol (99.5%) 1.5 mL was injected into the first small branch of the first septal artery, under precise localization by intracoronary myocardial contrast echocardiography. The pressure gradient of apex-LVOT-aorta reduced from 90 to 20 mmHg after ASA. No AV block was noted after the procedure and echocardiography revealed improved peak velocity cross LVOT and interventricular septal thickness. No more SAM or eccentric MR was observed. Previous studies recommended ASA reserved for patients with higher surgical risk and severe comorbidities. However, a recent study showed that young adults had better long-term survival and only one-half pacemaker implantation rate than older group following ASA. Under the guidance of intracoronary myocardial contrast, target vessel could be precisely localized to small branch from a septal artery to decrease myocardial damage. Therefore, ASA may be considered as the first-line SRT for symptomatic HOCM due to minimal invasiveness and effective outcome.

Keywords: Alcohol ablation, contrast echocardiography, hypertrophic obstructive cardiomyopathy

Introduction

Hypertrophic obstructive cardiomyopathy (HOCM) is an inheritable disease that occurs in approximately 1 in 500 individuals.[1] The current medical guideline for HOCM suggests septal reduction therapy (SRT) for drug-refractory heart failure (New York Heart Association [NYHA], functional class III or IV) and severe left ventricular outflow tract (LVOT) obstruction (peak pressure gradient ≥50 mmHg).[2] SRT includes surgical myectomy (SM) and alcohol septal ablation (ASA). ASA is considered for older patients with severe comorbidities who are at a high surgical risk.[2] However, selecting the septal branch of left anterior descending (LAD) coronary artery for ASA is difficult. Intracoronary myocardial contrast echocardiography improves myocardial perfusion imaging and guides ASA. We tried to use contrast echocardiography to minimize the territory of alcohol ablation and reduce myocardial damage. Here, we report a case with intracoronary myocardial contrast echocardiography-guided ASA for HOCM and a review of relevant literature (current guidelines and review articles).

Case Report

A 40-year-old male who had a history of thalassemia minor presented to our hospital with year-long chronic progressive

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postexercise chest tightness, dyspnea, and palpitation. Initially, the patient was diagnosed with HOCM through echocardiography and receive valsartan (80mg QD), amiodarone (100mg QD), propranolol (10mg BID), diltiazem (30mg BID) over 1 year. Therefore, ASA was performed after a multidisciplinary discussion.

After admission, the patient was afebrile and his vital sign were as follows: heart rate 75 beats/min, respiratory rate 18 breaths/min, and blood pressure 123/60 mmHg. Cardiac examination revealed S4 positive and pan-systolic ejection murmur (grade 3/6) at mid left sternum border and from the apex to the aortic area. Electrocardiography showed sinus arrhythmia with left ventricular hypertrophy and left atrial enlargement. Chest radiograph revealed borderline cardiomegaly with grade I pulmonary venous hypertension. Transthoracic echocardiography demonstrated ejection fraction about 74%, dilation of the left atrium, systolic anterior motion (SAM) of the mitral valve [Figure 1a], interventricular septal thickness about 16 mm [Figure 1b], severe eccentric mitral regurgitation (MR) [Figure 1c], and peak velocity crossing the LVOT about 5.3 m/s [Figure 1d]. Diagnostic cardiac catheterization performed before ASA revealed a patent coronary artery. The pressure gradient from the aorta to the LVOT was 90 mmHg. The first septal artery of the LAD artery branched into three small parts [Figure 1e]. Initially, we selected the first small branch as the target vessel supplying blood to the basal septum [Figure 1f]. The basal septum was enhanced by injecting a contrast agent, (Definity, Bristol-Myers Squibb Medical Imaging, Billerica, MA) into the target vessel [Figure 1g and h]. Then, 1.5 mL alcohol (99.5%) was injected into the same branch and the further coronary angiography revealed target vessel obstruction [Figure 1i]. The aorta to LVOT pressure gradient decreased to 20 mmHg after ASA immediately. The procedure was done smoothly without atrioventricular conduction disturbance. After 1 week, the echocardiography indicated no SAM [Figure 1j], LVOT peak velocity of 2.2 m/s [Figure 1k], and improvement of MR [Figure 1l]. ASA improved the symptoms of heart failure, and the patient return to work after 1 week. Echocardiography performed after 2 years of ASA revealed that the interventricular septal thickness and LVOT peak velocity were reduced to 12 mm and 1.4 m/s respectively.

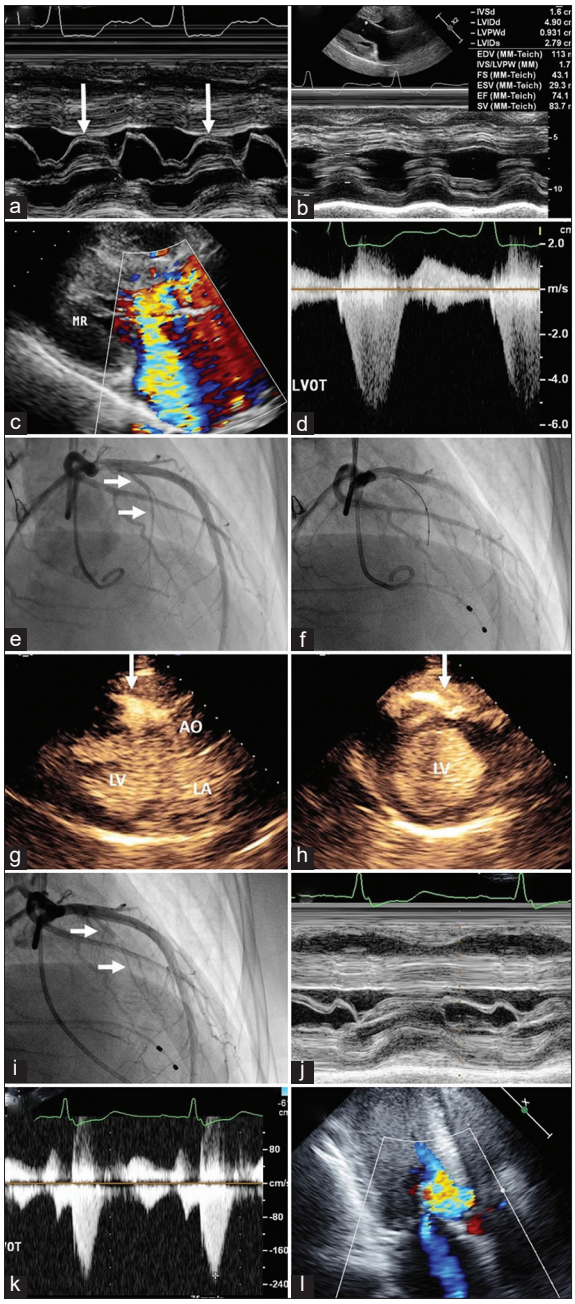

Figure 1: (a) M-mode echocardiography showing SAM (arrow) from the anterior leaflet of the mitral valve. (b) M-mode echocardiography (PLAX/view) showing an interventricular septum distance of 1.6 cm. (c) PLAX view showing severe eccentric MR. (d) LVOT peak velocity of approximately 5.3 m/s.(e) Coronary angiography showing three small branches of the first septal artery of the LAD artery. (f) Balloon and wire were inserted into the first small branch. (g) PLAX view showing basal septum enhancement (arrow) through intracoronary myocardial contrast. (h) Parasternal short axis view showing basal septum enhancement (arrow) through intracoronary myocardial contrast. (i) Limited flow (arrow) in the first small branch after ASA. (j) M-mode echocardiography showing no SAM after ASA. (k) LVOT peak velocity of approximately 2.2 m/s after ASA. (l) No eccentric MR and reduced severity after ASA. PLAX: Parasternal long axis, SAM: Systolic anterior motion, MR: Mitral regurgitation, LVOT: Left ventricular outflow tract, LAD: Left anterior descending coronary artery, AO: Ascending aorta, LV: Left ventricle, LA: Left atrium, ASA: Alcohol septal ablation

Discussion

The management of symptomatic HOCM due to drug-refractory or severe LVOT obstruction has long been debated between SM and ASA. Except in patients with a high resting LVOT pressure gradient (≥100 mmHg), extreme septal thickness (≥30mm), and some specific anatomic features of LVOT (abnormally positioned papillary muscle, anomalous papillary muscle insertion directly into mitral valve, accessory muscle bundles and abnormal chordal connection), ASA ensures outcomes same as those of SM.[2] Although ASA is minimally invasive, repeat interventions, increase conduction

disturbance and potentially arrhythmogenic scars have been considered when choosing therapeutic modalities.[2] The clinical efficacies can be comparable to SM by proper selection of septal perforator arteries.[2] Although the first large septal artery is generally selected as the target vessel for basal septum, some areas of the right ventricular wall or papillary muscle can also be considered.[3] Excessive anatomic variation in the septal artery system precludes the direct injection of alcohol.[3] According to intracoronary myocardial contrast echocardiography, the small branches of the first septal artery can be selected as target vessels. The relatively small septal infarct area may help reduce the incidence of pacemaker implantation, defibrillator implantation, and mortality.[4] Through contrast echocardiography, the target vessel must be precisely located to reduce the volume of alcohol required for ASA and avoid damage to other cardiac structures.[3] In addition, intracoronary myocardial contrast echocardiography could be used for three-dimensional imaging to accurately locate the target septal zone to decrease long-term events.[5] The major complications after ASA include complete heart block and permanent pacemaker requirement.[2] Injecting ≥1 septal perforators, using high volumes of alcohol and not using myocardial contrast enhancers for echocardiographic localization are the potential risk factors for complete heart block.[3] The risk of permanent pacemaker implantation is approximately 10% after ASA and 5% after SM.[2] Long-term mortality, NYHA functional class improvement, and sudden cardiac death are common with various SRTs.[6] The risks of post-ASA long-term mortality, NYHA function class deterioration, atrioventricular block, pacemaker implantation, and annual adverse arrhythmic events are lower in young adults than in older adults.[7,8] Nevertheless, owing to its minimal invasiveness and effective outcomes, intracoronary myocardial contrast echocardiography guided ASA should be considered as the first-line intervention for patients with symptomatic HOCM.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent form. In the form the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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Conflicts of interest

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