Commentary: Reoperative apical myectomy, a long run for a short slide, or a clear benefit yet to be determined

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For more than 60 years, surgical septal myectomy has proven to be the most effective and definitive strategy for reversing progressive heart failure symptoms due to left ventricular (LV) outflow tract obstruction in patients with drug-refractory advanced heart failure. In experienced hypertrophic cardiomyopathy (HCM) centers, surgical myectomy has a low operative mortality (<1%) and is responsible for relief of symptoms in 90% to 95% of patients, while also providing a survival benefit. In a small proportion of cases (4%), myectomy fails to achieve symptom relief, even after the outflow gradient has been abolished. In such nonresponders, other mechanisms emerge as responsible for symptoms, including comorbidities, and systolic or diastolic dysfunction.

In their report, Sun and colleagues1 examine the outcomes of 6 patients with advanced heart failure symptoms after previous transaortic septal myectomy who underwent reoperative transapical myectomy to enlarge the LV cavity with the primary intention of increasing stroke volume and lowering diastolic filling pressures. Over an average follow-up of 0.6 years, the authors report an increase in the end systolic and end diastolic chamber size resulting in an improvement in physical capacity and patient-perceived functional status in 5 of the patients.

The authors first reported the approach described in the report for patients with nonobstructive apical HCM but now describe its potential application in HCM patients who had previously undergone successful septal myectomy.2 The rationale to consider apical myectomy in these patients was on the basis of the authors previous report which showed improvement in patient symptoms attributed to augmentation of stroke volume and therefore cardiac output by increasing LV cavity size. The authors also suggest that the benefit of apical myectomy can be attributed to improved LV compliance. However, whether the procedure also results in an improvement in diastolic function remains uncertain. Measurement of diastolic function before and after surgery using either traditional echocardiographic approaches or cardiac magnetic resonance imaging, would give further insight into the potential physiologic benefits of the procedure.

As it relates to the efficacy of apical myectomy in this situation, 3 of the 5 patients experienced only “some” improvement in symptoms. Assessment of clinical status was limited to a subjective survey questionnaire with all of the expected limitations of such methodology. It would be important to obtain follow-up imaging to determine if the morphologic changes noted immediately after surgery remained the same or changed, including whether systolic dysfunction could have developed and therefore be responsible for relatively suboptimal improvement in heart failure.

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symptoms. With that said, it brings to question whether these patients stand to benefit enough to justify undergoing a complex reoperation. Longer follow-up might serve to clarify this concern.

This is clearly a very unique and highly select patient subset, as evidenced by the small number of patients from a large-volume HCM center. With such a small cohort, one is left to consider how translatable these outcomes are even at other high-volume HCM centers with expertise in subaortic myectomy but without an experience in performing apical myectomies. We commend the authors on the valuable contribution their results will make toward this unique surgical treatment option for this complex patient population. They have enlightened us on the potential to treat this challenging group of patients while also reminding us of the importance of specialized centers of excellence to treat patients with HCM.

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