Letter to the editor: Septal myectomy in the era of genetic testing

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To the Editor:

The article "Septal myectomy in the era of genetic testing" by Vincent Chauvette et al. has been read with a great deal of passion and curiosity. 1 This manuscript, which is brief and enlightening for readers, is the result of the writers' extraordinary efforts. Positive genetic tests are associated with different features than

negative ones. Four distinct echocardiographic characteristics are exhibited by patients. Only patients with a positive genetic test exhibit the type 3 phenotype. Importantly, regardless of the outcome of the genetic test, the midterm outcomes of septal myectomy appear to be favorable. Nonetheless, we feel compelled to highlight certain issues that would have considerably improved the quality of this post and influenced its outcomes.

First, we anticipate that the matching of samples would have raised several doubts regarding the reliability of the results. The authors recruited 21% females in the negative testing group and 45% females in the positive testing group, with no gender parity. For illustration,a 2021 research by Roy Huurman et al.2 included 94 percent females and 95 percent males, recognizing the significance of a proper proportions of both sexes, and concluded that women undergo septal myectomy in older age and appear to have more aggressive disease based on echocardiographic findings. However, the lag to myectomy was not longer, indicating that the generational gap is not primarily due to a delay in care. The clinical results of myectomy are satisfactory for both men and women. Furthermore, authors should have emphasized on the midterm and long-term survival outcomes after septal myectomy and its consequences. Iatrogenic ventricular septal rupture is a well-known consequence of the treatment, and electrocardiographic alterations such as left and right bundle branch block, atrial fibrillation, and ventricular arrhythmias are serious complications after septal myectomy.3 As mitral valve leaflets and subvalvular apparatus anomalies play a significant role in the pathophysiology of HOCM, the surgical care of the mitral valve has been regarded as a crucial component of myectomy. Mitral regurgitation is caused by the lack of coaptation of mitral leaflets caused by SAM. Although medical therapy remains the firstline treatment, septal myectomy is intended to improve symptoms and reduce sudden cardiac deaths when the peak instantaneous left ventricular outflow tract gradient is 50 mmHg at rest or with incitement and there are distressing symptoms resistant to medical care.3

Fourth, more than 11 mutations of the sarcomere genes are known to be responsible for HOCM. Two mutations in sarcomere-encoding genes, -myosin heavy chain-(MYH7) and myosinbinding protein C, account for 70% of HOCM cases in individuals for whom genetic testing was possible (MYBPC3). Asymptomatic patients with MYH7 and MYBPC mutations exhibited an increased left ventricular ejection fraction (LVEF) as well as diastolic dysfunction.4 Surgical septal myectomy (modified Morrow technique) and alcohol septal ablation are intended to decrease the LV bulk (ASA). Surgical treatment is regarded as the gold standard, with an American Guidelines class IIa recommended.

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