Letter by Andò et al Regarding Article,
“Prevalence, Clinical Significance, and Natural History of Left Ventricular Apical Aneurysms in Hypertrophic Cardiomyopathy”

To the Editor:

We read with great interest the article by Maron et al1 about the cohort of patients with hypertrophic cardiomyopathy (HCM) and left ventricular apical aneurysms. In the mid-1990s, the same authors drew our attention to a small group of patients with HCM and uncommon apical bulges regarded as *diverticula*.2 Cardiac magnetic resonance now sheds light on the structure of these apical bulges and the surrounding myocardium and demonstrates transmural myocardial fibrosis of the aneurysmal rim, largely extending in the contiguous ventricular walls.1

Apical aneurysm has been classically considered a complication of acute myocardial infarction. This comprehensive series1 demonstrates that apical aneurysms can form in patients in whom obstructive coronary artery disease is ruled out. Therefore, the occurrence of myocyte loss somehow related to microvascular dysfunction is likely to play a crucial pathophysiological role.

In light of the previous article,2 we recently used the term *pseudodiverticulum*3 to identify an apical bulge associated with HCM that now seems indistinguishable from those outlined in the current series.1 The use of such a term to describe a relatively common feature in HCM is likely to be abolished or, rather, to be limited to those congenital outpouchings typically known as *diverticula*.4 Indeed, Maron et al did not include in their series1 those young patients in whom the walls of the apical protrusion consisted of muscular tissue.2

Catastrophic complications are likely to occur more frequently in this subset of patients with HCM. Besides the established role of a prophylactic implantable cardioverter-defibrillator, the recent advances in catheter-based structural cardiac interventions will likely allow us to propose percutaneous aneurysm occlusion, as an alternative to oral anticoagulation, in forthcoming years to selected patients.5

The mechanisms contributing to the formation of apical aneurysms in HCM are multiple and still to be clarified. In the case we observed,1 gadolinium enhancement was confined to the ventricular walls adjacent to the neck of the aneurysm. The peculiarity was the intriguing discontinuation of a normally contracting myocardium, because the walls of the apical ancillary chamber consisted of only pericardium and did not show gadolinium enhancement. The left ventricular apex has to be considered a *locus minoris resistentiae* due to the thinner helical architecture of myocardium in the apical loop. A critical mechanism may consist of the attempt to counterbalance the pressure exerted by the hypertrophied hypercontractile ventricle on the apex. The abnormal intracavitary stress may force a herniation of the endocardium toward the pericardium. Independently from midcavitary obstruction and according to the Laplace law, Nature in such a case would aim to normalize the wall stress of the apex, which could get less hypertrophied than the rest of the ventricle, by creating an ancillary chamber with a small radius and a thin wall thickness. This speculation might explain why ventricular rupture of apical aneurysms in HCM is uncommon.1 Simply put, wall stress inside the apical aneurysm would be unexpectedly low.

Disclosures

None.

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