Letter apropos ''Reversible perfusion defect in hypertrophied papillary muscle on myocardial perfusion imaging: The 'filled doughnut sign'''

To the Editor,

I read with special interest the article by Vadi *et al.*[1] entitled ''Reversible perfusion defect in hypertrophied papillary muscle on myocardial perfusion imaging: The 'filled doughnut sign'''' about hypertrophied papillary muscle (PM) ischemia, an uncommon situation particularly with vasodilator stressors. According to the resting SPECT images provided, a higher uptake than background was noted in the mid-cavity zone of the LV compared to more distal and proximal zones, giving an impression of ''filled-in doughnut'' in mid-ventricular short-axis slices as offered by the authors. Owing to its reversibility and the fact that on echocardiography and contrast-enhanced magnetic resonance, a hypertrophied PM is present, the authors concluded that the findings suggest vasodilator-induced ischemia in the hypertrophied PM. It seems that in this case, the story is more complicated and challenging to simply and solely attribute the findings to PM ischemia, as some clinical factors and also issues related to the science and physics of imaging may have played a role. PMs, in general, do not have a large-enough arterial branch to create a significant flow heterogeneity during vasodilator challenge. However, by exercise stressors, particularly in patients with valvular disorders, the PM may go under traction and thus an ischemia occurs in the PM and at the site of its attachment to the LV wall. Hence, mitral regurgitation and transient murmur may occur.[2] The diagnosis of PM ischemia is compromised in SPECT imaging because of limited spatial resolution. Positron emission tomography with its superior spatial resolution seems to be an ideal modality for evaluation of PM ischemia.[3] The authors provided the nongated SPECT slices which are considered to suffer from spatial and temporal blurring of information. Providing unblurred gated slices may be more helpful. The LV cavity is less dilated in rest vs stress so that the hypertrophied myocardial walls at the mid-zone of LV lie somewhat closer to each other that opens a room for the effect of partial volume averaging of different proportions in both phases. Additionally, the effect of using color spectrums, compared to linear grayscale one, and the resultant false perception of the relative brightness and edges (or pseudocontouring) cannot be neglected.[4,5] The same appearance with much less intensity is present in mid-cavity zone of the LV in stress mid-ventricular short-axis images. Providing further evidence of ischemia of PM vs partly or full contribution of mid-ventricular hypertrophy to such findings is of great interest. Lastly, I am inclined to refer to another case,[6] we published recently, of circumferential mid-ventricular hypertrophy and apical aneurysm, as a subtype of hypertrophic cardiomyopathy in that the findings are in accordance with this case.

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Disclosures

Mohsen Qutbi declares that he has no conflict of interest.

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doi:10.1007/s12350-019-01890-x

J Nucl Cardiol 2020;27:1043.
1071-3581/$34.00