TRANSIENT ISCHEMIC DILATION: A NEW PERSPECTIVE UTILIZING CARDIAC MRI

Poster Contributions
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Background: Stress-induced global LV myocardial ischemia is difficult to detect by nuclear myocardial perfusion imaging (MPI). Digital LV volume software is used to assess changes in overall scintigraphic LV volume after stress, and is termed transient ischemic dilation (TID). The mechanism of TID is thought to be due to global endocardial isotopic hypoperfusion. Cardiac MRI (CMR) offers improved spatial and temporal resolution compared to MPI. This study was designed to investigate the mechanisms of TID using CMR.

Methods: A standard CMR myocardial perfusion protocol was modified by adding a short axis SSFP imaging sequence at the level of the papillary muscle tips, just before the 1st pass perfusion study at peak regadenoson effect. Parameters for this study included LV end-diastolic diameter (EDD) and slice volume (EDV), LV end-systolic diameter (ESD) and slice volume (ESV), anteroseptal LV wall thickness (AWT), infero-posterior LV wall thickness (IWT) and myocardial perfusion findings, all of which were assessed at rest and at peak regadenoson stress. For this study, TID was defined as an EDD increase with stress.

Results: The 124 TID patients had a mean slice LV EDV increase of 1.96 ml during stress compared to rest, while 246 non-TID patients showed a mean slice LV EDV decrease of 1.73 ml during stress (p<0.001). Among patients with stress related increases in LV EDD and slice EDV, mean end-diastolic AWT decreased from 1.01 cm to 0.92 cm (p<0.001), while mean IWT decreased from 0.92 to 0.85 cm (p<0.001). The TID patients had a non-significant increase in total cardiac diameter from 7.32 cm at rest to 7.43 cm at peak stress (p=0.2), while the non-TID patients showed a significant decrease in total cardiac diameter from 7.46 cm to 7.23 cm (p<0.05). Of the TID patients, only 31 of 124 (25%) showed perfusion defects and none had global endocardial hypoperfusion. Only 7 showed reversible ischemia, while 24 showed a fixed perfusion defect with infarction on LGE.

Conclusion: Stress-induced LV dilation is due to an increase in LV end-diastolic diameter and volume with thinning of the LV wall, but no change in the overall cardiac diameter. This phenomenon does not appear to be related to global endocardial hypoperfusion.