References


Occurrence of late gadolinium enhancement in ventricular ballooning or Tako-Tsubo syndrome: increased wall stress should not be overlooked: reply

Alter and Rupp suggest that late gadolinium enhancement (LGE) in Tako-Tsubo cardiomyopathy might be caused by increased wall stress. This is an interesting theory; however some points need to be clarified.

There is strong evidence that LGE depends largely on different volumes of distribution of gadolinium between healthy and structurally altered myocardium—namely, fibrosis or necrosis—and less on wash-in/wash-out kinetics. The mechanisms leading to LGE proposed by the authors—increased capillary permeability and reduced venous return—cannot cause LGE because they cause uniform, not focal, distribution of gadolinium, which can only be measured as early gadolinium enhancement ratio (EGER) or by T1-mapping.

In contrast, there are several papers demonstrating the histologically proven relationship between fibrosis and LGE for various disease entities. Therefore, if increased wall stress causes LGE, it has to be mediated by either focal fibrosis and/or apoptosis/necrosis.

We could show significantly and focally increased collagen contents of patients exhibiting LGE when compared with patients without LGE. Early and immediate collagen turn over has been described previously. We therefore think that it is safe to assume that LGE is caused by increased interstitial fibrosis.

However, we acknowledge that this increased amount of fibrosis might, at least in part, be a consequence of increased wall stress as we have not measured wall stress.

Nonetheless, we do not believe that the relationship between wall stress and LGE in other forms of cardiomyopathies is as straightforward as claimed by the authors. It might also be possible that the extent of LGE and wall stress is simply confounded as they both correlate strongly with disease severity. This is certainly true for ischaemic cardiomyopathies where the extent of LGE depends solely on the waveform of necrosis and corresponding replacement fibrosis. Therefore, it might also be possible that LGE is a predictor of ensuing increase in wall stress and not the other way round. But this is a matter of further research.

References


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