Letter to the Editor

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RE: Relation of Late Gadolinium Enhancement to Increased Ventricular Wall Stress in Dilated Cardiomyopathy

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With great interest, we read the study by Yoon et al. (1) confirming the previously reported relationship between late gadolinium enhancement (LGE) and increased left ventricular (LV) wall stress (2). Assessment of post-contrast T1 values by cardiac magnetic resonance imaging (CMR) using look-locker sequences is related to myocardial tissue characteristics of LV remodeling, LV compliance and increased N-terminal pro-B-type natriuretic peptide, a marker of increased ventricular distending forces and wall stress (3). Interestingly, the findings are independent of LGE.

Causes and consequences of LGE in dilated

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This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (http://creativecommons.org/licenses/by-nc/3.0) which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited. cardiomyopathy are still controversial (4). Since LGE delineates myocardial postinfarct scar accurately, it was assumed that LGE also represents fibrosis in dilated cardiomyopathy. However, morphology, type and extent of LGE varies markedly between different etiologies (5). In dilated cardiomyopathy, LGE typically occurs in a streak-like midwall pattern and at the septal hinge points. In contrast, the extent of fibrosis, as shown by histology, increases from the right to left side of the interventricular septum and from the subepicardial to subendocardial myocardial layers of the free wall, which does not match a midwall pattern (6).

We defined the wall stress index to calculate LV wall stress based on LV cavity size and myocardial mass/volume as assessed by CMR (7). Increased wall stress is reportedly associated with LGE in dilated cardiomyopathy (2). However, in dilated cardiomyopathy, the synonymous use of the terms LGE and fibrosis is not recommended (4).

It is unclear whether LGE is reversible after load reduction, assuming functional causes. Support comes from the finding of increased ventricular wall stress in transient apical ballooning, i.e., Takotsubo syndrome, which is frequently associated with temporary LGE emergence (8). Underlying fibrosis appears unlikely, since the short-term reversibility within few days is not in line with the usual collagen turnover rate (9).

The present study (1) substantially contributes to the understanding and interpretation of LGE. In particular, the results indicated that LGE cannot provide sufficient information on the degree of interstitial fibrosis. Current pre- and post-contrast T1 mapping techniques appear to be helpful for further characterization.

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