Letter by Alter Regarding Article, “Myocardial Fibrosis as a Key Determinant of Left Ventricular Remodeling in Idiopathic Dilated Cardiomyopathy: A Contrast-Enhanced Cardiovascular Magnetic Study”

To the Editor:

Late gadolinium enhancement (LGE) as assessed by MRI is a frequent finding in various cardiac diseases. While delineating postinfarct scar accurately, there are controversies on causes and consequences in dilative cardiomyopathy (DCM). The thorough study by Masci et al1 examined absence of LGE and deduced nonexistence of myocardial fibrosis as prognostic predictor of a favorable left ventricular (LV) remodeling in 58 patients with DCM. Of note, patients’ medical heart failure therapy was optimized after enrollment in the study.

For interpretation of LGE, differentiation of a variety of types and forms seems to be required. In particular, the most common streaky-like occurrence in DCM at the (mechanically loaded) septal midwall or at the hinge points is of major interest. It was recently shown in 300 patients with DCM that LGE correlates with increased LV wall stress.2 Studies on DCM demonstrating increased fibrosis by histology and a matching LGE pattern have been rare. Beyond a total rise of fibrosis, the degree involves a gradient that increases from epicardial to endocardial layers of transmural LV sections and from the right to the left side of the septum not matching a midwall pattern.3 Thus, it remains ambiguous whether there is indeed a one-to-one relation between LGE and fibrosis. Currently, emerging techniques of pre- and postcontrast T1 mapping seem to be very promising to provide further insight into tissue characterization by cardiac MRI.

The question arises whether other prognostic determinants could have been involved in the study.1 We have recently shown in 502 patients with DCM that LV dilatation is correlated with increased wall stress, which precedes the development of LV hypertrophy.4 Because the ensuing hypertrophy is still inappropriate, greater dilatation is associated with wall stress increase. Increased LV wall stress exhibits various adverse consequences, for example, an impaired autonomic tone, favoring of arrhythmias by opening of ion channels, increased oxygen consumption, and an adverse remodeling with unfavorable prognostic consequences. Applying these findings to the referenced study, it has to be inferred that at time of enrollment LV wall stress was markedly increased in patients responding to initiation or intensification of heart failure treatment with a favorable remodeling, which is also reflected by the fact of increased N-terminal pro–brain natriuretic peptide as reported.1 Brain natriuretic peptide correlates with ventricular wall stress.5 Based on the reported LV mass and volume and using the wall stress index, it has to be assumed that LV end-diastolic and end-systolic wall stress was increased to 4.9 kPa versus 4.6 kPa (end-diastolic LV pressure assumed at 16 mmHg, normal end-diastolic wall stress <4 kPa) and to 28.0 kPa versus 21.0 kPa (peripheral systolic pressure inserted as reported, normal end-systolic wall stress <18 kPa), respectively.4 Because load reduction is the principal feature of heart failure treatment, it is probable that previously untreated or undertreated patients with increased LV wall stress responded to a greater degree. Thus, we suggest to calculate wall stress and evaluate the hypothesis whether increased wall stress is a causative prognostic risk factor in DCM.

Disclosures

None.

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References


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