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

We thank Drs Alter and Taylor et al for their attention on our article and the interesting issues raised.

With regard to Dr Alter’s comments, the correlation between late gadolinium enhanced (LGE) detected by contrast-enhanced cardiovascular magnetic resonance and replacement fibrosis seen on histology has been described in several autopsy cases of idiopathic dilated cardiomyopathy. In 7 explanted idiopathic dilated cardiomyopathy hearts, Gulati et al. showed an excellent agreement between replacement fibrosis at histopathology and LGE as seen with in vivo cardiovascular magnetic resonance. In our study, we disregarded spotty LGE confined to the interventricular septum insertion points because whether this pattern identifies areas of fibrosis remains a matter of debate. For these reasons, we think that LGE in our patient population likely reflected replacement fibrosis. Acute or chronic myocardial damage of any cause and subsequent derangements associated with adverse ventricular remodeling may potentially lead to myocyte necrosis followed by replacement fibrosis. Increased ventricular wall stress (at end diastole, end systole, or both) caused by geometric remodeling of the left ventricle is one of the promoters of myocardial injury and, consequently, of replacement fibrosis. However, although wall stress is an instantaneous measure of hemodynamic burden of the failing ventricle, LGE on the contrary is a comprehensive marker of disease severity which takes into account the initial myocardial injury along with the ensuing neurohormonal and hemodynamic abnormalities. Furthermore, fibrosis may play a primary role in the development of ventricular dysfunction and life-threatening ventricular arrhythmias.

With regard to Dr Taylor et al’s comments, our considerations on device therapy, far from conclusive, should be considered hypothesis generating. Our findings support the concept that the identification of myocardial fibrosis by LGE during the initial evaluation of idiopathic dilated cardiomyopathy may distinguish between patients who are likely (LGE negative) or unlikely (LGE positive) to respond to optimized medical therapy. In fact, the absence of LGE was a strong and independent predictor of left ventricular reverse remodeling irrespective of other baseline variables, including the severity of clinical status, the degree of left ventricular dilatation and dysfunction, and ECG parameters (including QRS duration). In this sense, LGE status may improve the risk stratification and management of patients with idiopathic dilated cardiomyopathy. Notwithstanding, we agree with Taylor et al. that our hypothesis, as outlined in the discussion section of our article, needs to be tested in properly designed studies.

References
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