We thank Drs Finsterer and Stöllberger for their interest in our work.\(^1\)

We recommend using T1 mapping to detect left ventricular non-compaction (LVNC) fibrosis instead of late gadolinium enhancement (LGE) for the following reasons: (1) pathology samples of LVNC showed diffuse fibrosis rather than focal scar formation;\(^2\) (2) the LGE technique is qualitative and unable to detect diffuse myocardial fibrosis,\(^3,4\) so T1 mapping would be more appropriate to evaluate LVNC tissue characteristics; and (3) we found a significant correlation between native T1 values and LGE, and we also observed that several LGE− patients with low left ventricular ejection fraction (LVEF) had higher native T1. In addition, the increased T1 values in the LGE− group compared with the control group indicated there was myocardial interstitial fibrosis in LVNC patients without LGE.

The question of LVNC classification has been argued for many years. According to the AHA, LVNC is classified as a genetic cardiomyopathy.\(^5\)

As to the question of “clinical presentations or complications”, we believe this is a syntactical issue. We understand that although most LVNC patients are asymptomatic, the common clinical presentation for symptomatic patients includes progressive dysfunction, arrhythmia and thromboembolism.\(^6,7\)

Oftentimes, asymptomatic patients are not admitted to hospital until clinical signs appear and it’s often difficult to discern whether such a presentation is indeed a presentation or a resulting complication.

We systematically screened all LVNC patients for neuromuscular disorders (NMD) and 6 patients were referred to neurologists. Only one was suspected of having NMD based on quadriceps biopsy. However, DNA exon test showed no genetic mutations related to NMD. All patients in this study were free from NMD.

In our study, we observed that family history was more often positive for LVNC in LGE− LVNC patients as compared with LGE+ LVNC patients. However, we cannot extend the conclusion that “LGE− patients more frequently have a genetic background than LGE+ patients”. In addition, the mean native T1 of family members was significantly higher than that of non-family members among LVNC patients with preserved LVEF. Thus, we recommend using T1 mapping to detect LVNC fibrosis.

LGE+ LVNC patients presented with chest pain more frequently than LGE− LVNC patients. Other ongoing study in our center indicated that most LVNC patients have abnormal microcirculation, especially LGE+ patients. We believe this is the reason that LGE+ patients are more prone to have chest pain.

Disclosures
No conflicts of interest are disclosed.

References

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