LETTER TO THE EDITOR

Myocardial Deformation Analysis and Late-Gadolinium Enhancement: Important Markers of Cardiac Amyloidosis Involvement That Can Masquerade as a False-Negative Diagnosis

To the Editor:
We read with great interest the article by Seitaro Oda and colleagues in which they aimed to evaluate the additive diagnostic role of circumferential strain (CS) by tagging cardiac magnetic resonance (CMR) in patients with suspected cardiac amyloidosis (CA).¹

In our opinion, and according with our previous data, this report is very interesting because it shows new important findings; additionally, this study prompted us to reexamine our previous data about myocardial deformation and CMR findings in CA.

In patients with CA there is an impairment of longitudinal, radial and circumferential deformation with respect to patients without CA. In 2011, our group used echocardiography with 2-dimensional tracking feature to compare sarcomeric hypertrophic cardiomyopathy (HCM) with transthyretin (TTR) CA.²

We showed that in patients with a similar burden of increased left ventricular (LV) thickness, CS at the epicardial layer was more impaired in TTR-CA than in HCM. More recently, we compared myocardial deformation with different degrees of amyloid deposition (no deposition, mild deposition and severe deposition) detected using ⁹⁹mTc-3,3-diphosphono-1,2 propanodicarboxylic acid scintigraphy (⁹⁹mTc-DPD).³ We found that CS is impaired with mild amyloid deposition compared with patients without CA. However, severe amyloid deposition was associated with a supra-normal value of CS that we have hypothesized as a compensative phenomenon. Although this phenomenon of increased CS could identify a false-negative CA, our deformation data on CS are largely confirmed by Oda et al’s recent data obtained with tagging CMR.

Regarding the late-gadolinium enhancement (LGE) CMR findings in Oda et al’s paper, we think that some points need to be discussed.⁴ First of all, a negative LGE is inconclusive to exclude CA. In a comparative study of ⁹⁹mTc-DPD and LGE CMR in TTR-CA patients,⁵ we observed that the cardiac amyloid infiltration burden is significantly underestimated by CMR with LGE as compared with ⁹⁹mTc-DPD. Namely, some patients without LGE had ⁹⁹mTc-DPD radiotracer accumulation in the myocardium. To increase the accuracy of LGE CMR, we suggest analysing not only the LV myocardium but also LGE in the atria, atroventricular valves and in the right ventricle in cases of negative LGE with a high suspicion of CA.⁶

Finally, new CMR techniques such as T1 mapping that have been demonstrated to be more accurate in detecting subtle diffuse amyloid deposition should be used in the diagnosis of CA.⁷

Conflict of Interest
None.

References

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