VIA MEDICA

Native myocardial T1 mapping in β-thalassemia major patients with and without iron overload

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Three patients with known β -thalassemia major who received multiple blood transfusions were referred for myocardial iron overload by magnetic resonance imaging (MRI). Standard cardiac MRI sequences Cine Steady State Free Precession were acquired in short axis and 4-chamber planes for calculation of ventricular volumes and functions. For clinical quantification of myocardial iron overload, T2* mapping sequence was obtained in short axis.

We present the cases of the three patients: one without, one with mild, and one with severe myocardial iron overload. The T2* values were 29 ms denoting no myocardial overload, and 17 ms and 5.2 ms denoting mild overload and severe myocardial overload respectively (normal >20 ms, mild overload 15–20, moderate 10–15, severe iron overload <10 ms) [1] (see Figure 1).

Additionally, native myocardial T1 mapping sequence was obtained (modified look locker inversion recovery MOLLI--Siemens Healthcare) and revealed T1 values of 944 ms (within the normal reference range), 865 ms (mildly reduced), and 523 ms (severely reduced) respectively. The Z scores were -0.3, -4 and -20 respectively.

Myocardial iron deposition results in local magnetic field inhomogeneities causing reductions in T1, T2 and T2* relaxation times [2]. Currently, T2* mapping is the method



Figure 1. Cardiac magnetic resonance (MR) mid short-axis T2* color maps in β -thalassemia major patients without myocardial iron overload (A), with mild myocardial iron overload (B), and with severe myocardial iron overload (C). Cardiac MR mid short-axis native T1 color maps in β -thalassemia major patients without myocardial iron overload (D), with mild myocardial iron overload (E), and with severe myocardial iron overload (C).

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of choice for cardiac iron quantification and is incorporated into clinical guidelines [3].

Recently, myocardial T1 mapping was proposed as a potential complementary technique for iron overload quantification [4]. A significant correlation was found between native T1 and T2* values in patients with β -thalassemia major [2]. The reported normal reference T1 mean value is 972 ± 43 ms, with upper and lower limits of 885 and 1,059 ms [1]. A cutoff T1 value of 904 ms has been proposed to distinguish between β -thalassemia major and healthy subjects [5]. In patients with myocardial iron overload evident on T2* (<20 ms), the T1 value ranged from 474 to 804 ms, mean 653 ± 133 ms [6].

One advantage of T1 mapping is its better reproducibility compared to T2*, which is important for serial studies monitoring disease progression [5]. In addition, T1 mapping is less vulnerable to magnetic field inhomogeneity compared to T2*, and therefore it may be useful when T2* is borderline reduced to differentiate between local field inhomogeneities and iron content [6].

Authors' contributions

All authors have participated in article preparation and have approved the final article.

Conflict of interest

The authors declare no conflict of interest.

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Ethics

The work described in this article has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans; EU Directive 2010/63/EU for animal experiments and uniform requirements for manuscripts submitted to biomedical journals.

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