Movement abnormalities in the left ventricle of thalassemia major patients
Antonella Meloni1, Vincenzo Positano1, Pier Paolo Bitti1, Antonella Carollo3, Letizia Gulino1, Antonino Vallone1, Chiara Tadisca1, Ellisabetta Chiodi6, Massimo Lombardi1, and Alessia Pepe1


Introduction. Movement abnormalities of the left ventricle (LV) have been reported in thalassemia major (TM) patients [1]. Movement abnormalities can be detected through a qualitative analysis of cine MR images. Moreover, MR is the gold standard technique for the evaluation of myocardial iron overload (MIO) [2], biventricular global systolic function [3] and myocardial fibrosis [4]. The aim of this study was to investigate the relationships between movement abnormalities and MIO, left ventricular (LV) function and myocardial fibrosis.

Materials and methods. CMR (1.5T) was performed in 1092 TM patients (537 male; 30.6 ± 8.5 years) enrolled in the Myocardial Iron Overload in Thalassemia (MIOT) Network [5]. Cine SSFP images were acquired in vertical and horizontal long-axis, and in sequential 8-mm short-axis plans (gap 0 mm) from the atrio-ventricular ring to the apex. These sequence were used to evaluate the wall motion and to quantify LV volumes and ejection fraction (EF) by means of the MASS software in a standard way [3]. For MIO assessment, three parallel short-axis views of the LV were acquired using a T2* GRE multi-echo sequence [2]. To detect myocardial fibrosis, late gadolinium enhanced (LGE) images were acquired in the same views as used for cine images after the gadobutrol (1.0 mol/l) (0.2 mmol/kg) intravenous administration. During image analysis the 17-segment LV model of the standard AHA/ACC was taken into account. On the cine images, segmental wall motion was visually assessed by skilled observers (with at least 5 years of experience in CMR) and scored as 1=normal, 2=hypokinesia, 3=akinesia and 4=dyskinesia. The T2* value in all segments as well as the global value were calculated. Presence/absence of enhancing area was assessed for each segment.

Results. Abnormal motion of LV was found in 66 (6%) patients: 60 were hypokinetic while 6 were dyskinetic. Our data demonstrated predominant involvement of wall motion abnormalities in the medium anterior, anterolateral and septal segments. Table 1 shows the comparison between patients with normal and abnormal motion. Patients with abnormal motion were significantly older, they had significantly lower global heart T2* value and a significantly higher number of segments with T2*<20 ms. Left volumes and mass indexed by body surface area were significantly higher in patients with abnormal motion while the EF was significantly lower.

LGE areas were detected in 196 patients (18%) and they were predominantly located in the mid-ventricular septum. There was a significant correlation between the presence of enhancement and the abnormal motion.

Conclusions. Movement abnormalities in the left ventricle were not really frequent in TM patients but were associated with age, MIO, LV dilation and dysfunction, and myocardial fibrosis.