Similar complication rates were recorded at the 3-month follow-up between the HBP group (13%, n = 6) and the LBBAP group (6.8%, n = 3) ($P = .48$). Lead-related complications were observed in 5 (10.6%) in the HBP group (loss of capture in 2, macrodisplacement in 1, and a significant increase in pacing threshold in 2) vs 1 (2.3%) in the LBBAP group (macrodisplacement, $P = .2$).

Nonselective HBP was obtained in 28 (75.7%) of the 37 patients and selective HBP in 9 (24.3%). The pacing threshold was lower and the sensed R-wave amplitude was higher in the LBBAP group at implantation and after 3 months. Threshold and amplitude were increased in both groups at 3 months vs baseline. The difference was statistically significance in the LBBAP group (figure 1A).

In the 29 successful cases with a baseline QRS of < 120 ms (14 in HBP and 15 in LBBAP), the paced QRS was significantly increased (97.3 ± 7.1 vs 105.5 ± 10.3 ms; $P < .001$) and the mean QCI was $8.7 \pm 10.5\%$, with a slightly lower increase in the HBP group ($5.2 \pm 10.2\%$ vs $12 \pm 10\%$; $P = .08$). Better results were obtained with selective HBP than with nonselective HBP or LBBAP (figure 1B).

In the 48 successful cases with a baseline QRS of ≥ 130 ms (25 in LBBAP and 23 in HBP), 23 had right bundle branch block, 19 had left bundle branch block, and 6 had QRS paced by a previously implanted device. There was a trend ($P = .055$) toward a greater reduction of QCI with LBBAP vs HBP and an even greater reduction vs conventional CRT in failed cases (figure 1C).

Among the patients with left bundle branch block (12 in HBP and 11 in LBBAP), the success rate was 100% in the LBBAP group vs 66.7% in the HBP group ($P = .09$). Among successful cases, the paced QRS width was lower in the LBBAP group (112 ± 9 vs 127 ± 26 ms; $P = .16$), although this difference was not statistically significant (figure 1). Among the patients with right bundle branch block (14 in HBP and 13 in LBBAP), the success rate did not differ between the HBP (85.7%) and LBBAP (84.6%) groups ($P = 1$), but paced QRS was lower (106 ± 7 vs 122 ± 16 ms; $P < .01$).

The main findings of this study were that narrower QRS complexes and better pacing outcomes were obtained at implantation and at 3 months with LBBAP than with HBP. Yiran Hu et al.[6] described a similar success rate between LBBAP and HBP, although our population also included CRT indication. The radiological exposure time was shorter with the LBBAP technique because it does not require a search for the His-bundle electrogram (essential in HBP).

In conclusion, LBBAP achieves a narrower paced QRS, lower threshold, improved R-wave detection, and shorter fluoroscopy time with a similar complication rate compared with HBP.

CONFLICTO DE INTERESES

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Usefulness of myocardial T_1 and T_2 mapping with magnetic resonance in transfusion-dependent patients with low-risk myelodysplastic syndrome

Utilidad del mapeo miocárdico T_1 y T_2 mediante resonancia magnética cardiaca en pacientes transfundidos con síndrome mielodisplásico de bajo riesgo

To the Editor,

Iron overload cardiomyopathy is common in low-risk myelodysplastic syndrome (MDS) patients requiring repeat red blood cell (RBC) transfusions. Early diagnosis is essential to establish an effective treatment with iron chelators and improve their survival,[1] and the detection of myocardial iron overload (MIO) modifies its strategy by intensifying the therapy.

T_2* by cardiac magnetic resonance (cMR) is the gold standard for MIO diagnosis. Recently, small studies carried out in thalassemia mayor (TM) suggest the usefulness of new imaging techniques such as T_1 mapping being reduced in individuals with MIO.[2,3]

We performed a prospective observational study to analyze the usefulness of T_1 and T_2 mapping in the assessment of transfusion-dependent low-risk MDS patients (including very low, low and intermediate risk groups from the Revised-International Prognostic Scoring System classification [IPSS-R]), older than 18 years, who provided their authorization by signing informed consent. Exclusion criteria were those patients belonging to very high or high-risk MDS groups according to IPSS-R classification and those who had never received transfusions. The study was approved by the local ethics committee.

Thirty-one low-risk MDS patients were recruited between January 2016 and February 2017 (table 1). Patients underwent a 1.5-Tesla cMR (Philips Healthcare, Netherlands) including cardiac morphology and function assessment, late gadolinium enhancement, myocardial and hepatic T_2* mapping (multiecho-gradient sequences including 15 echo times from 1-16 milliseconds), native T_1 (modified

Table 1
Baseline characteristics, main results of the cardiac magnetic resonance assessment and evolution.

Baseline characteristics (N = 31)	Age, y	76 ± 10	≥ 1 cardiovascular risk factor	29 (93.5)
	Sex: male	17 (55)	Previous cardiac disease Angina Myocardial infarction Atrial arrhythmias Heart valve disease ≥ moderate Systolic dysfunction Heart failure	12 (38.7) 3 (9.7) 2 (6.5) 7 (22.6) 5 (16.1) 2 (6.5) 2 (6.5)
	Number of RBC units transfused	47 [286]		
	Transfusion burden			
	low (< 25 RBC)	10 (32.3)		
	medium (25-125 RBC)	12 (38.7)		
	high (> 125 RBC)	9 (29.0)		
	Iron chelation treatment	20 (64.5)		
Morphological and functional cMR study results (N = 31)	*Main findings*		*Pathological results*	
	LV end-diastolic volume, mL LV end-systolic volume, mL LV mass, g	155 ± 49 59 ± 35 125 ± 36	LV dilatation LV hypertrophy	8 (25.8) 4 (12.9)
	RV end-diastolic volume, mL RV end-systolic volume, mL	141 ± 39 54 ± 22	RV dilatation	9 (29.0)
	LV ejection fraction, % RV ejection fraction, %	63.6 ± 10.7 656 ± 6.6	LV systolic dysfunction RV systolic dysfunction	7 (22.6) 2 (6.5)
	LA area, cm^2 RA area, cm^2	29.2 ± 7 23.4 ± 8.2	LA dilatation RA dilatation	17 (54.8) 4 (12.9)
	Aortic root diameter, mm Ascendant aorta diameter, mm	32.9 ± 3.4 33.3 ± 3.9	Aortic root dilatation Ascending aorta dilatation	1 (3.2) 13 (41.9)
			Late gadolinium enhancement Valve disease (≥ moderate) Pericardial effusion	1 (3.2) 3 (9.7) 6 (19.4)
Tissue characterization analysis (N = 31)	*Relaxation times*		*Pathological results*	
	Myocardial T_2^* time, ms	33 ± 8.7	Myocardial T_2^* time (< 20 ms)	3 (9.7)
	T_2 time, ms	55 ± 8.7	T_2 time (< 44 ms)	5 (16.1)
	Native T_1 time, ms	995 ± 84	Native T_1 time (< 932 ms)	5 (16.1)
	Hepatic T_2^* (ms)	7.1 ± 7.8	Hepatic T_2^* (< 6,4ms)	13 (41.9)
Combined event at follow-up (N = 31)	*Death*	10 (32.3)	*Cardiovascular event*	8 (25.8)
	Cardiovascular Infection Intracranial hemorrhage Mesenteric ischemia Unknown	4 (12.9) 3 (9.7) 1 (3.2) 1 (3.2) 1 (3.2)	Heart failure Heart failure admission Atrial fibrillation Atrial flutter	6 (19.4) 5 (16.1) 2 (6.5) 1 (3.2)

cMR, cardiac magnetic resonance; LA, left atria; LV, left ventricle; RA, right atria; RBC, red blood cells; RV, right ventricle.
Data are expressed as No. (%) or mean ± standard deviation.
Measures and pathological results according to established normal values[4] and for T_1 and T_2 mapping those extracted from a cohort of 292 healthy controls performed using the same local technique (native T_1 993 ± 62ms, T_2 52 ± 8ms).

look locker inversion recovery -MOLLI-with-5(3)3 acquisition scheme) and T_2 (multi-echo gradient-spin-echo sequence) mapping according to recommendations.[4] Mapping postprocessing was performed using Medis 2.1 software, plotting the ROI or "region-of-interest" in the left ventricle mid-septum segment in short axis.

cMR study revealed a high prevalence of unknown structural heart disease (51.6%) (table 1) and up to 16.1% of patients showed pathological reduction in relaxation times (table 1).

We studied the T_1 and T_2 mapping correlation with iron overload parameters (transfusion burden, biochemical parameters, and hepatic and myocardial T_2^*).

T_2^*, T_1 and T_2 times were significantly reduced as the RBC transfusion burden increased (figure 1A). We found a 65 RCB received cutoff to detect a pathological reduction of T_1 (< 932 ms) (area under the curve 0.762, sensitivity 80%, specificity 66%).

T_2^*, T_1 and T_2 times were also significantly reduced as the serum ferritin levels increased (T_2^* R = -0.533, P = .001; T_1 R = -0.501, P < .001; T_2 R = -0.36, P = .039).

T_1 and T_2 times showed a positive correlation with the values of the gold standard myocardial T_2^* (figure 2 B). In addition, T_1 and T_2 times showed a statistically significant positive correlation. Patients with MIO defined by T_2^* (< 20 ms) showed significantly lower values of T_2 (42 vs 56 ms, P = .014) and T_1 (803 vs 1012 ms; P < .001) compared with patients with normal T_2^*. In contrast, T_1 and T_2 mapping did not correlate with liver T_2^*.

We analyzed the association of T_1 and T_2 mapping with the combined event: mortality from any cause and/or cardiovascular event (newly diagnosed HF, atrial or ventricular arrhythmia) during follow-up.

After a median of 2.4 [0.7] years, 10 (32.3%) patients had the combined event (table 1). A total of 19.4% of patients developed both cardiovascular event and later death.

All patients with a T_2^* < 20 ms at the inclusion (3 patients; 9.7%) had the combined event at follow-up compared with those with normal T_2^* (100% vs 76%; P = .023). Patients who had the combined event had significantly lower native T_1 (974 [131] vs 1029 [66];

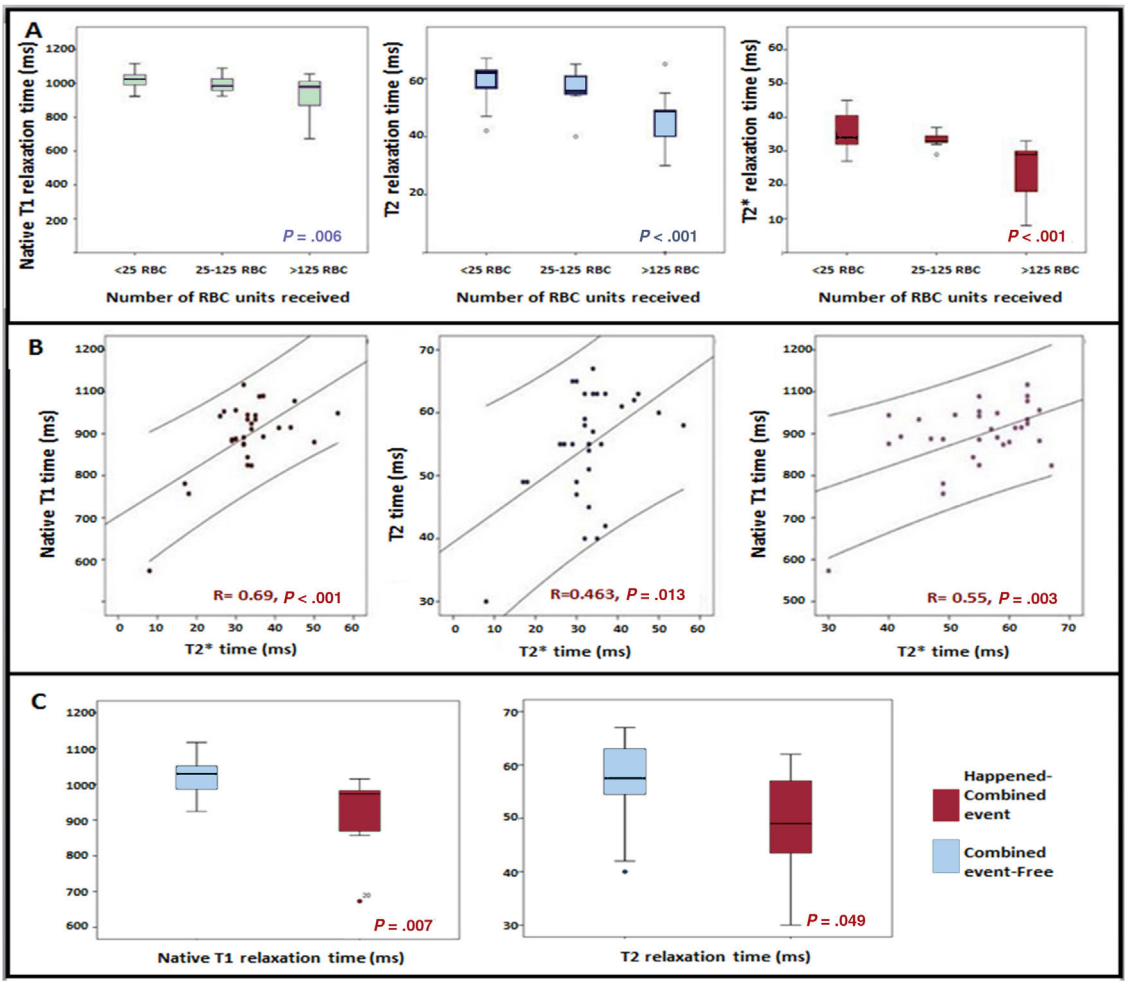

Figure 1. Myocardial T_1, T_2 and T_2^* mapping results in low-risk myelodysplastic syndrome patients. A: relationship between T_1, T_2 and T_2^* times with the RBC transfused. B: correlation between T_1, T_2 and T_2^* times. C: association between T_1 and T_2 times and the combined event: mortality from any cause and/or cardiovascular event. RBC, red blood cell.

P = .007), T_2 (49 [16] vs 58 [9]; P = .049) and T_2 (30 [15] vs 33 [5]; P = .047) times than those with an event-free course (figure 1C).

To the best our knowledge, this is the first study in MDS patients that analyzes cMR tissue characterization techniques other than T_2^*.

Our results highlight the relationship between T_1 and T_2 maps and MIO defined by T_2^*, the transfusion burden, and ferritin levels. The highest proportion of patients with pathological reduction of T_1 compared with reduced T_2^* in our population suggests that T_1 could be affected earlier than T_2^* in the presence of MIO, similar to TM reports.[5] T_1 and T_2 mapping reduction probably suggests incipient iron deposition in patients with normal T_2^* and absence of other heart disease. The correlation observed between T_1 and the gold standard T_2^* is stronger than in the case of T_2. Moreover, the 65 RCB cutoff observed to detect pathological reduction of T_1 is lower than the classic 100 RCB for MIO risk. This could play a role in the early detection of iron overload cardiomyopathy, leading to optimize the iron chelating treatment that would modify the course of the disease.

Regarding the prognostic usefulness of parametric techniques, our study in the MDS population corroborates the relationship of pathological myocardial T_2^* to the development of adverse events similar to TM patients. Regarding native T_1 and T_2, we suggest for the first time its relationship to adverse events in a hematological disease with transfusion dependence. Advanced age, comorbidities, and cardiac remodeling secondary to chronic anemia probably contribute to the high prevalence of previously undiagnosed structural heart disease and the high incidence of cardiovascular events observed in our population.

As other limitations, this is a single-center observational study, with a small number of patients and short-term follow-up.

In conclusion, myocardial tissue characterization with T_1 and T_2 mapping in patients with transfusion-dependent low-risk MDS is significantly related to transfusion burden, ferritin levels, and myocardial T_2^*. Patients who develop the combined cardiovascular event and/or death from any cause in the follow-up more frequently exhibit myocardial T_2^* below 20 ms and lower values of myocardial native T_1 and T_2. T_1 seems to be affected earlier than T_2^* and to provide prognostic usefulness, and is probably a marker for early chelation therapy in this population.

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Expanding donation niches. Heart transplant from a donor on veno-venous ECMO

Expandiendo los nichos de donación: trasplante cardiaco de un donante con ECMO veno-venoso

To the Editor,

The shortage of donor hearts, lengthening [0]waiting times, and the need for urgent transplants[1] necessitate an expansion of the clinical criteria for heart transplant (HT) to include donors with a nonstandard-risk. This category includes patients on veno-venous extracorporeal membrane oxygenation (V-V ECMO) as bridge therapy to lung transplantation or recovery. Not all of these patients survive, creating a niche of potential donors. We present the case of an adult male patient who underwent successful HT from a brain-dead donor on V-V ECMO.

The donor was a 28-year-old man with labial herpes simplex virus infection who was admitted to hospital with fever, myalgia, and cough. A chest X-ray and computed tomography scan indicated bilateral pneumonia. Blood cultures were negative, and serological analysis detected immunoglobulin M (IgM) antibodies to syncytial respiratory virus. The patient was placed on broad-spectrum antibiotic therapy and required mechanical ventilation. A tracheostomy was performed on day 14 of hospitalization, and the patient was transferred to our hospital. After hospitalization for 19 days, the patient's worsening respiratory profile required respiratory V-V ECMO support (Cardiohelp-Getinge Group, Sweden) with cannulation of the right femoral and jugular veins. The patient was treated according to the local protocol, including continuous anticoagulation with sodium heparin to maintain the activated coagulation time between 160 s and 180 s. On post-HT day 9, the patient developed bilaterally fixed and dilated pupils, and cranial computed tomography detected evidence of hypoxic-ischemic encephalopathy. Brain death was confirmed by clinical examination and cerebral computed tomography angiography. An apnea test was inconclusive for hypoxemia.

After obtaining family consent, the patient was painstakingly evaluated as a potential donor. Favorable results were obtained from hemodynamic and respiratory analysis and ancillary tests (table 1). The heart, liver, and kidneys were removed under V-V ECMO support.

The recipient was a 55-year-old man (87 kg, 167 cm) diagnosed with hypertrophic cardiomyopathy and with a previous sternotomy. He had been on the elective HT waiting list for 75 days.

Orthotropic HT was performed by the bacaval method with standard sternotomy. Total ischemia time was 125 min, and the patient showed good postoperative progress. Post-transplant echocardiography detected good biventricular function of the donor organ. The patient was discharged 17 days after admission under treatment with standard doses of mycophenolate mofetil, tacrolimus, and prednisone. Over 48 weeks of follow-up, the patient underwent 6 scheduled endomyocardial biopsies: 4 showing mild cellular rejection (grade 1 R on the ISHLT 2004 working formulation) and 2 showing no evidence of rejection. The patient developed febrile neutropenia in post-HT week 11, with no signs of infection.

The use of V-V ECMO has increased due to the accumulated experience with this procedure and technological advances. The indications for V-V ECMO have expanded as a result, and this approach has become more generally used at more hospitals. The overall incidence of complications is 7.1%, with brain death accounting for 23% of them.[2]

Published data appear to confirm that organs from brain-dead donors supported by V-V or veno-arterial ECMO perform similarly to those from brain-dead donors without ECMO support.[3–5] Despite the evidence for the safety of organs transplanted from ECMO-supported donors,[4] there is a continuing reluctance among transplantation teams to use organs from these patients, owing to