HJ23
Hospital Universitari Joan XXIII
ICS Camp de Tarragona

CardioRM en el manejo de la insuficiencia cardiaca

Dra Guillén Marzo
Servicio de Cardiología

Pros y contras

A favor:

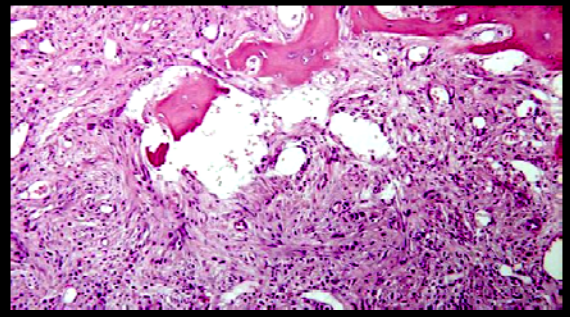
- Elevada precisión espacial
- Alta reproducibilidad de los resultados
- Independencia de la "ventana"
- No utiliza RX
- Permite caracterización tisular

En contra:

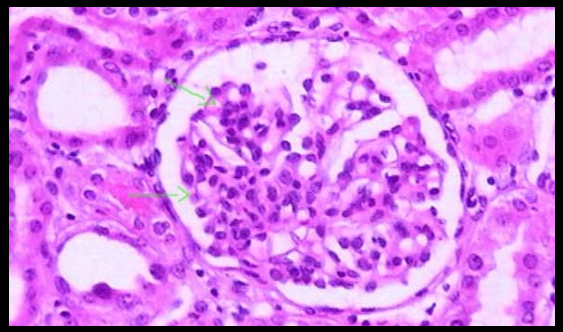
- Duración del estudio
- Disponibilidad
- Coste
- Necesidad de un ritmo cardiaco estable
- Claustrofobia
- Uso de contraste (IR terminal)
- Incompatibilidad con dispositivos implantables

Caracterización tisular

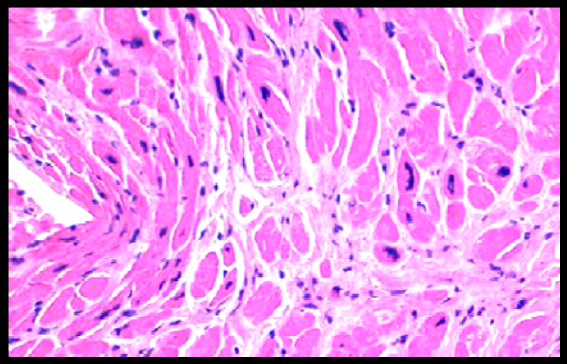
10000 biopsias medulares anuales

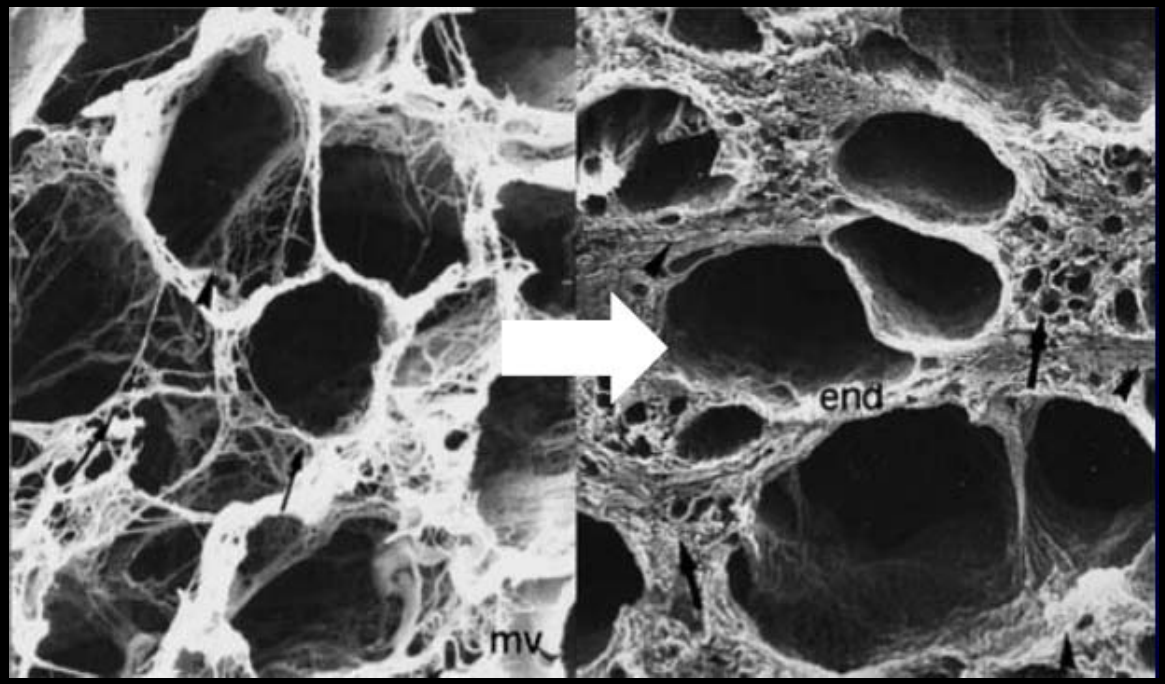


1300 biopsias renales anuales



¿? biopsias cardiacas anuales

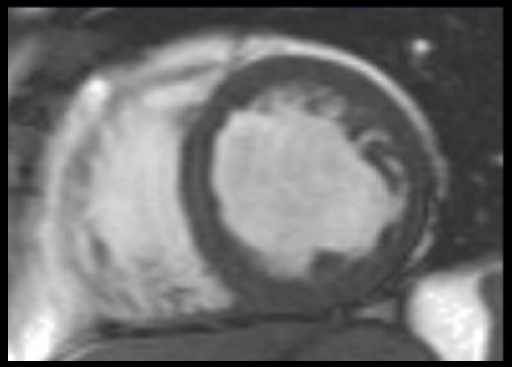




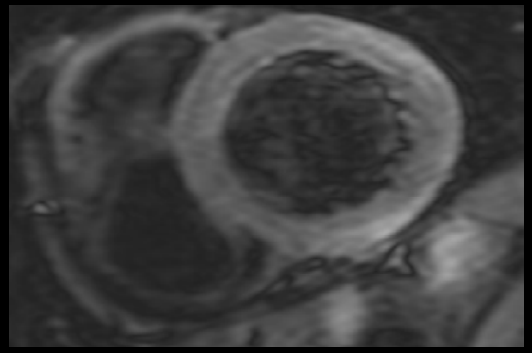
Necrosis celular

Aumento de la
matriz extracelular
modificación
de su arquitectura

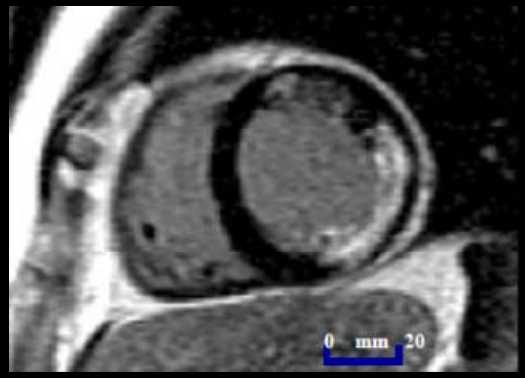
Tipos de secuencias



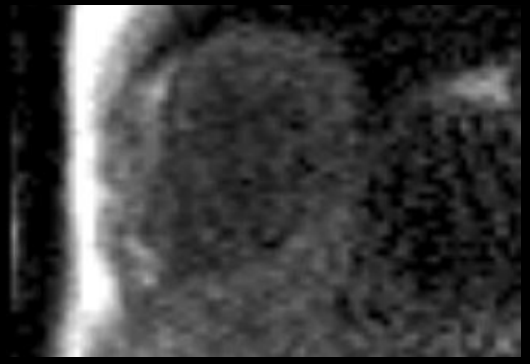
CINES



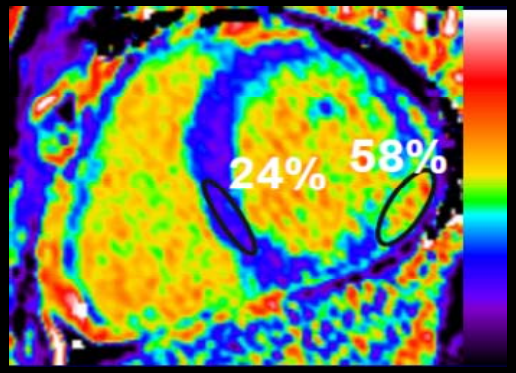
T2



REALCE
TARDIO
GADOLINIO



PERFUSIÓN



MAPPING T1

Causas isquémicas/valvulares

1. Estudio morfológico
2. Detección de isquemia
3. Valoración de viabilidad miocárdica
4. Pronóstico clínico

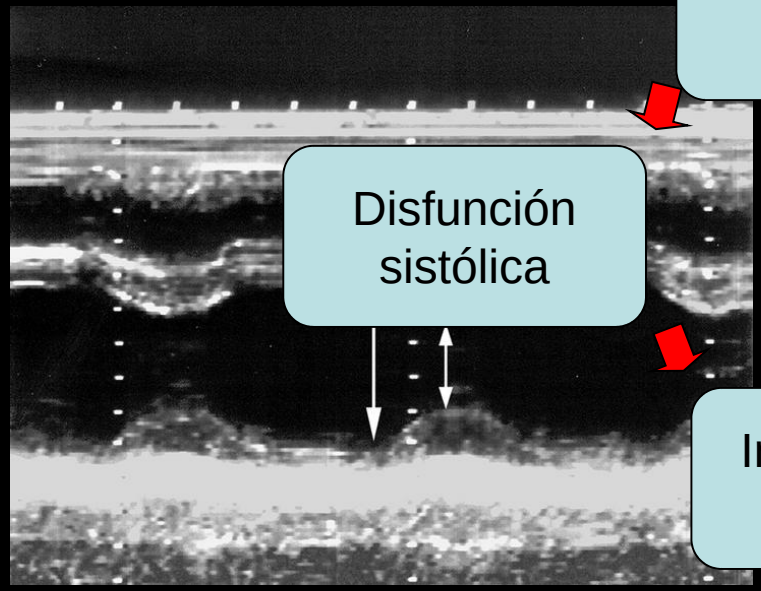
Causas no isquémicas

1. Estudio morfológico
2. Caracterización tisular
   - Edema
   - Fibrosis
   - Hierro
3. Pronóstico clínico

Lesión tisular

Necrosis celular
Fibrosis

Disfunción sistólica



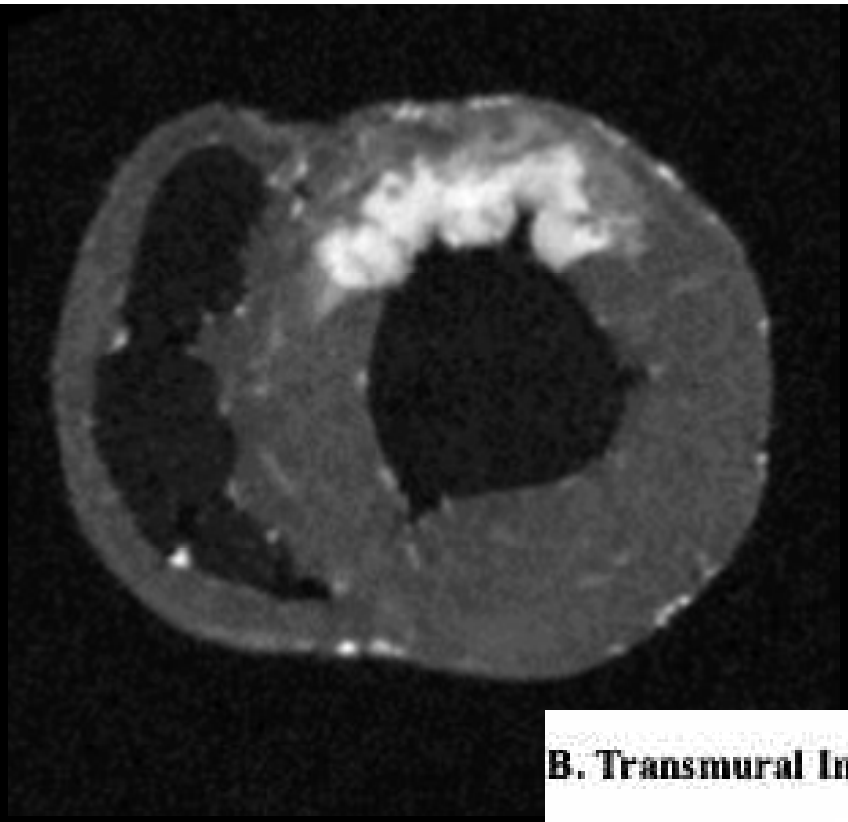
Disfunción diastólica

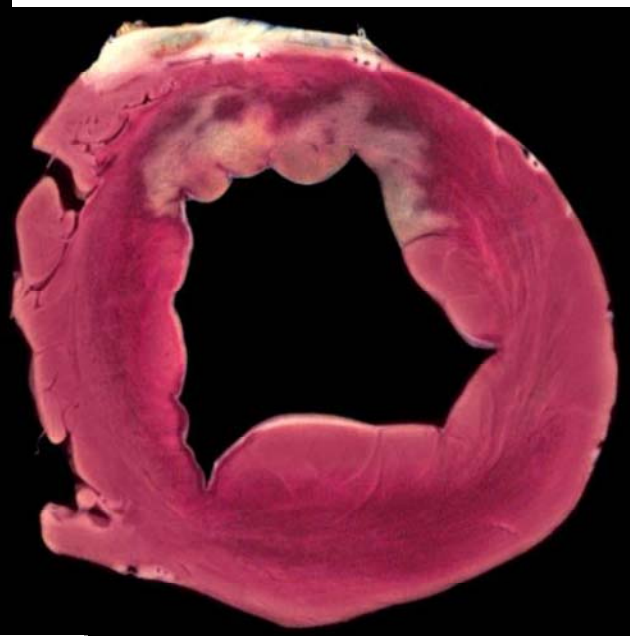
Insuficiencia cardiaca

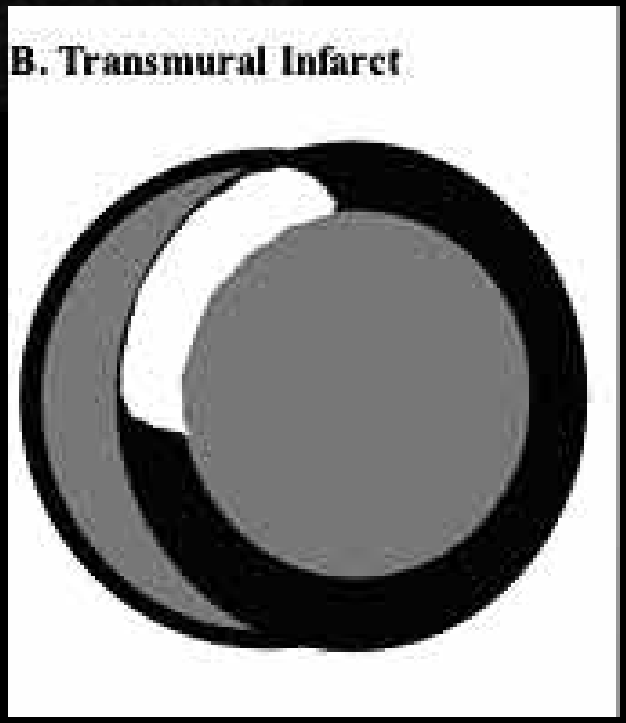
Valoración morfológica

- Morfología y funcionalidad de ventrículo izquierdo
- Anomalías de la contractilidad y presencia de escaras
- Diagnóstico diferencial de aneurismas y pseudoaneurismas
- Presencia de trombos intracavitarios
- Medición directa de valvulopatías aórtica y pulmonar

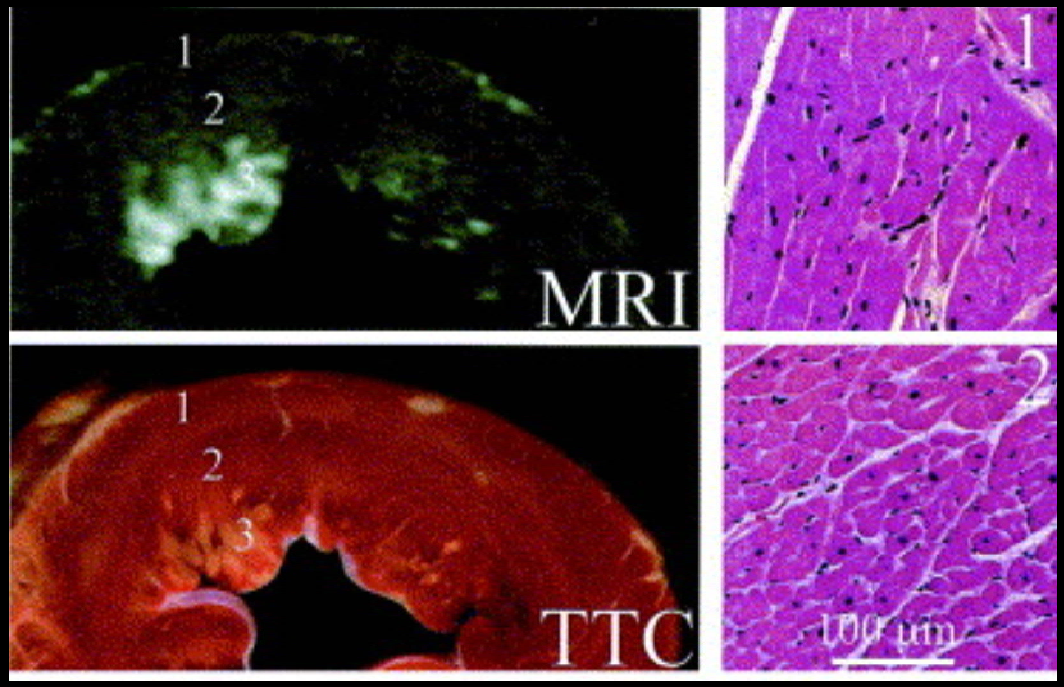
Isquémica/valvular

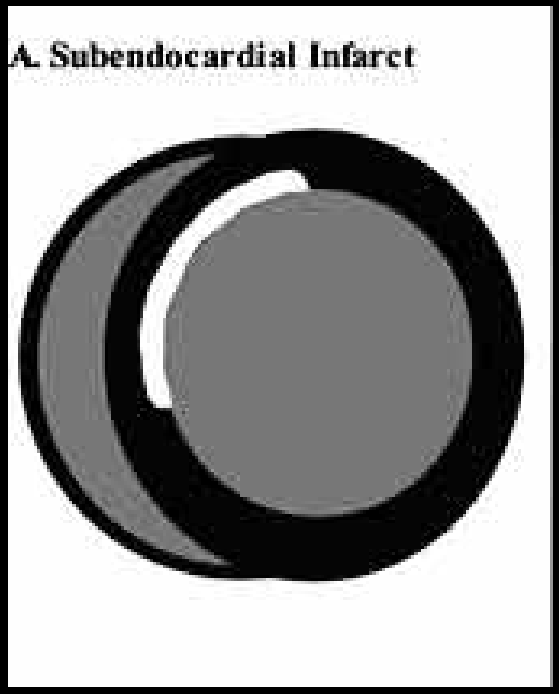




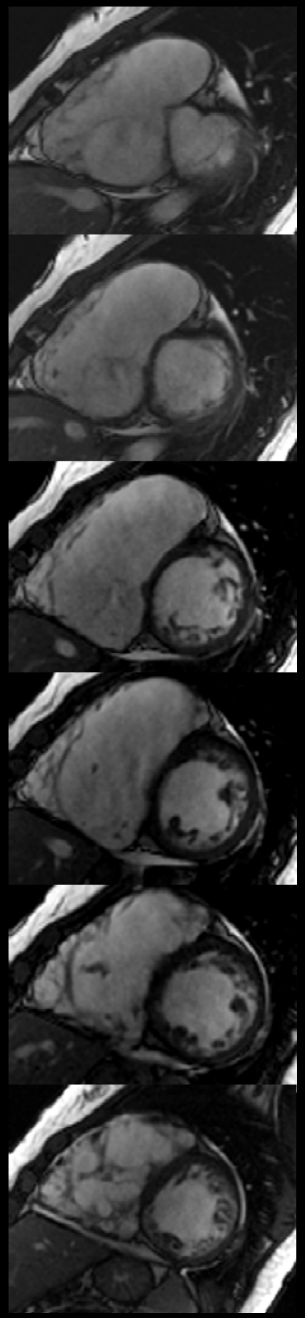


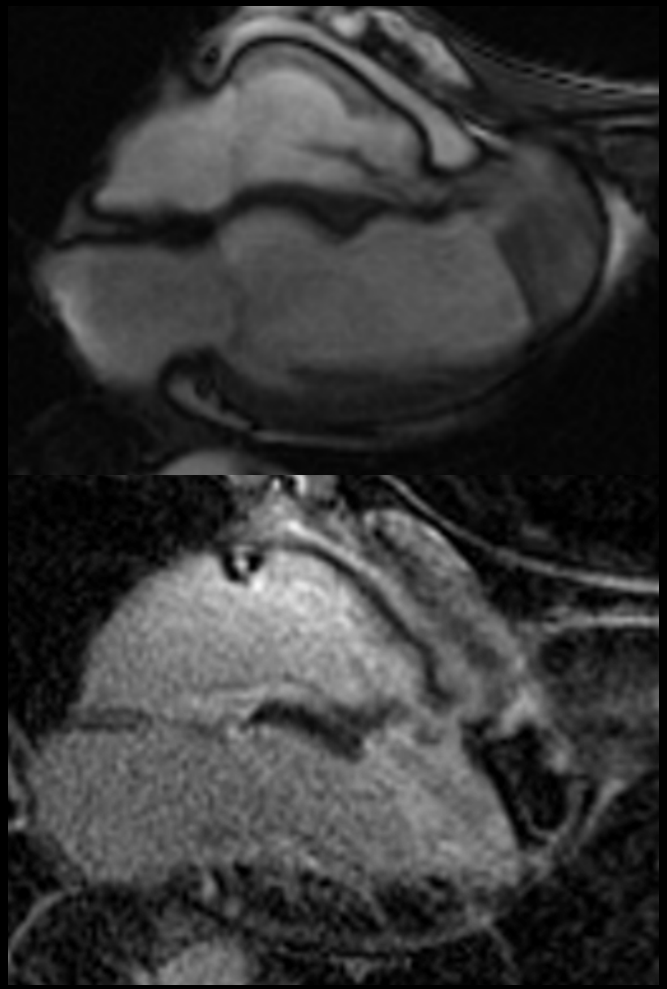
Isquémica/valvular

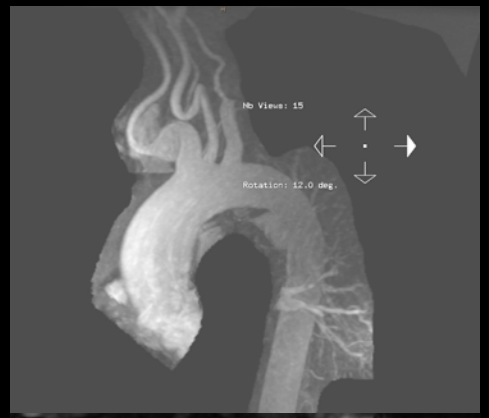


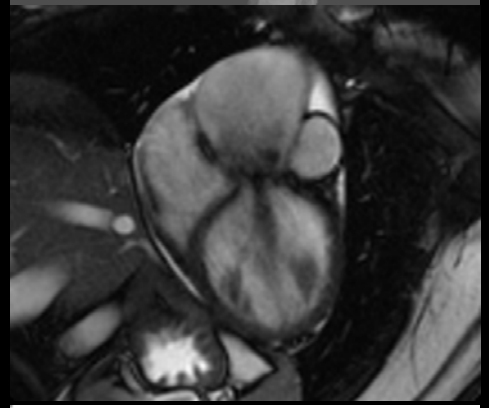


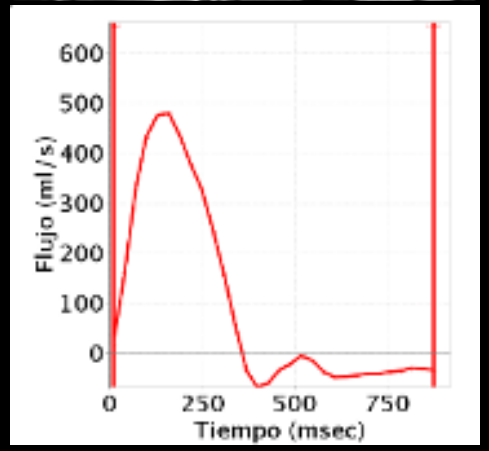
Isquémica/valvular







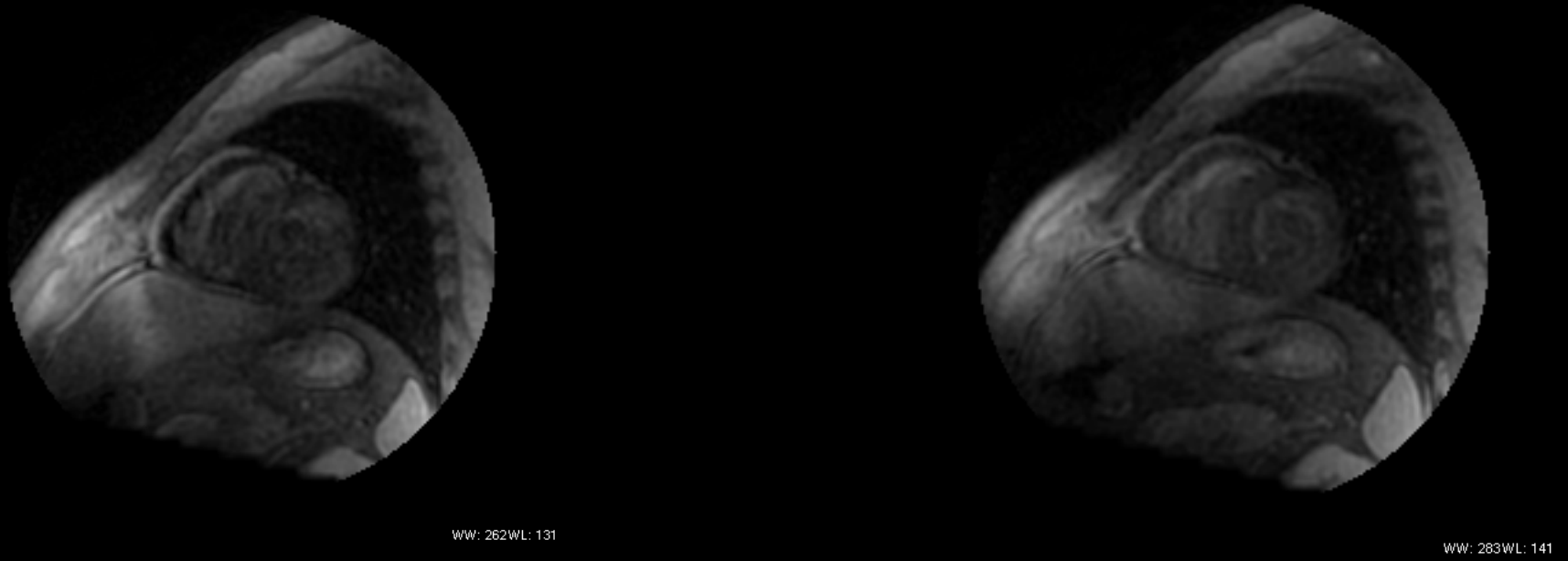




Detección de isquemia miocárdica

Las secuencias de perfusión permiten valorar en tiempo real la llegada de contraste al músculo cardiaco a través de la circulación coronaria.

Se realiza un estrés farmacológico para poner de manifiesto las insuficiencia coronarias y se compara con las imágenes en reposo



MR-IMPACT II: Magnetic Resonance Imaging for Myocardial Perfusion Assessment in Coronary artery disease Trial: perfusion-cardiac magnetic resonance vs. single-photon emission computed tomography for the detection of coronary artery disease: a comparative multicentre, multivendor trial

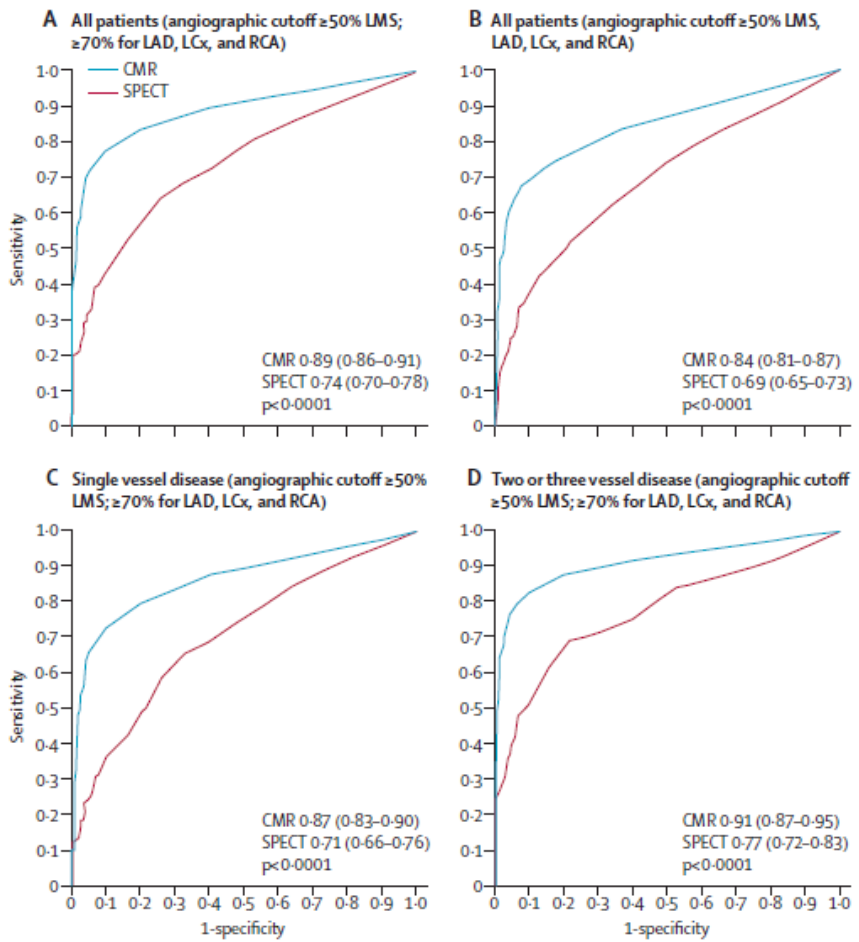
For this evaluation, 26 (5.6% of 465) CMR and 17 (3.7% of 465, $P = 0.21$ vs. CMR) SPECT studies were deemed non-evaluable by the MR and SPECT readers, respectively. The prevalence of CAD on CXA was similar in the studies excluded and included in the efficacy analysis (21 of 40 vs. 206 of 425, respectively, $P = 0.74$). When applying a single, i.e. binary threshold, to the CMR and SPECT images, the sensitivity scores were 0.67 and 0.59, respectively ($P = 0.024$, paired *t*-test), with the lower confidence level for the difference of +0.02, indicating superiority of CMR over SPECT for sensitivity (efficacy population: $n = 465$). The specificity score for CMR and SPECT was 0.61 and 0.72, respectively ($P = 0.038$, paired *t*-test) with a lower confidence level for the difference of −0.17, indicating inferiority of CMR vs. SPECT for specificity. For CMR and SPECT, sensitivities (mean ± SD of all readers) were 75 ± 7 and 59 ± 10%, respectively ($P = 0.03$) and specificities were 59 ± 8 and 72 ± 14%, respectively ($P = 0.03$). Positive and negative predictive values and accuracies (mean ± SD of all readers) for CMR were 70 ± 5, 65 ± 5, and 68 ± 5%, respectively, and for SPECT 73 ± 8, 60 ± 3, and 65 ± 3%, respectively (no significant differences).

The main results of the trial can be summarized as follows: (i) the primary endpoint of non-inferiority of CMR vs. SPECT for the detection of CAD was met for sensitivity, but not for specificity. (ii) No severe adverse effects occurred in the 515 patients who received the MR CM during pharmacological stress CMR.

Schwitter J el al. Eur Heart J 2013 (34): 775-781

Cardiovascular magnetic resonance and single-photon emission computed tomography for diagnosis of coronary heart disease (CE-MARC): a prospective trial

John P Greenwood, Neil Maredia, John F Younger, Julia M Brown, Jane Nixon, Colin C Everett, Petra Bijsterveld, John P Ridgway, Aleksandra Radjenovic, Catherine J Dickinson, Stephen G Ball, Sven Plein



Background In patients with suspected coronary heart disease, single-photon emission computed tomography (SPECT) is the most widely used test for the assessment of myocardial ischaemia, but its diagnostic accuracy is reported to be variable and it exposes patients to ionising radiation. The aim of this study was to establish the diagnostic accuracy of a multiparametric cardiovascular magnetic resonance (CMR) protocol with x-ray coronary angiography as the reference standard, and to compare CMR with SPECT, in patients with suspected coronary heart disease.

Methods In this prospective trial patients with suspected angina pectoris and at least one cardiovascular risk factor were scheduled for CMR, SPECT, and invasive x-ray coronary angiography. CMR consisted of rest and adenosine stress perfusion, cine imaging, late gadolinium enhancement, and MR coronary angiography. Gated adenosine stress and rest SPECT used ^{99m}Tc tetrofosmin. The primary outcome was diagnostic accuracy of CMR. This trial is registered at controlled-trials.com, number ISRCTN77246133.

Findings In the 752 recruited patients, 39% had significant CHD as identified by x-ray angiography. For multiparametric CMR the sensitivity was 86·5% (95% CI 81·8–90·1), specificity 83·4% (79·5–86·7), positive predictive value 77·2%, (72·1–81·6) and negative predictive value 90·5% (87·1–93·0). The sensitivity of SPECT was 66·5% (95% CI 60·4–72·1), specificity 82·6% (78·5–86·1), positive predictive value 71·4% (65·3–76·9), and negative predictive value 79·1% (74·8–82·8). The sensitivity and negative predictive value of CMR and SPECT differed significantly (p<0·0001 for both) but specificity and positive predictive value did not (p=0·916 and p=0·061, respectively).

Interpretation CE-MARC is the largest, prospective, real world evaluation of CMR and has established CMR's high diagnostic accuracy in coronary heart disease and CMR's superiority over SPECT. It should be adopted more widely than at present for the investigation of coronary heart disease.

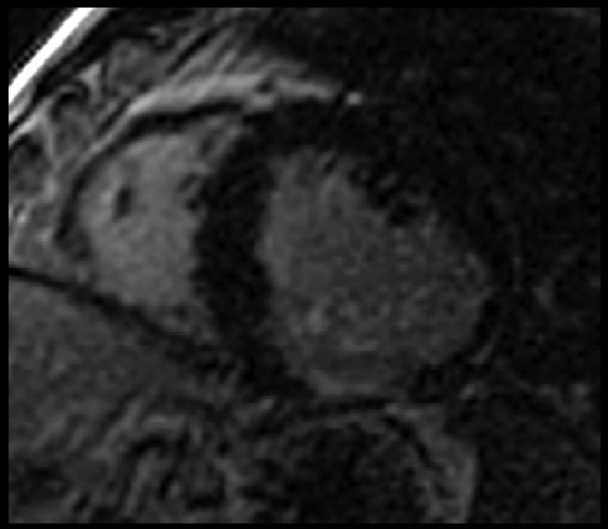
Interpretation

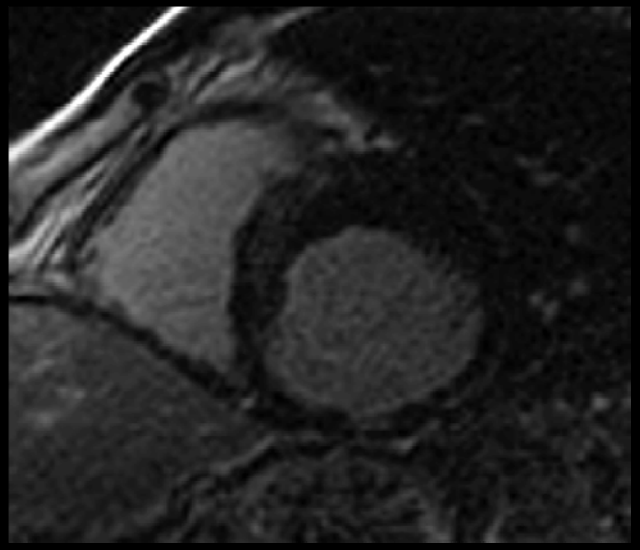
CE-MARC is the first large-scale trial of a multiparametric CMR protocol for the diagnosis of stable coronary heart disease. CMR had better sensitivity and negative predictive values than SPECT for coronary heart disease diagnosis, with much the same specificity. These findings support the wider adoption of CMR for coronary heart disease diagnosis and its inclusion in evidence-based clinical management guidelines.

Greenwood JP et al. Lancet 2012;379:453-60.

Viabilidad miocárdica

- Las secuencias de realce tardío de gadolinio permiten determinar la extensión y el grosor de la lesión isquémica y así como la presencia de miocardio viable y su grosor. Se considera actualmente el método de referencia para la valoración de la viabilidad miocárdica





Artículo original

Valor pronóstico a largo plazo del análisis completo de los índices de resonancia magnética cardiaca tras un infarto de miocardio con elevación del segmento ST

Pilar Merlos[a], Maria P. López-Lereu[b], Jose V. Monmeneu[b], Juan Sanchis[a], Julio Núñez[a], Clara Bonanad[a], Ernesto Valero[a], Gema Miñana[a], Fabián Chaustre[c], Cristina Gómez[a], Ricardo Oltra[d], Lorena Palacios[d], Maria J. Bosch[e], Vicente Navarro[f], Angel Llácer[a], Francisco J. Chorro[a] y Vicente Bodí[a,*]

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Palabras clave:
Resonancia magnética cardiaca
Pronóstico
Infarto agudo de miocardio con elevación del segmento ST
Extensión de la necrosis transmural

RESUMEN

Introducción y objetivos: Se ha demostrado el valor pronóstico de varios índices de resonancia magnética cardiaca a medio plazo tras un infarto agudo de miocardio con elevación del segmento ST. La extensión de la necrosis transmural permite una predicción simple y exacta de viabilidad miocárdica. Sin embargo, se desconoce su valor pronóstico a largo plazo más allá de una completa evaluación clínica y por resonancia. Nuestra hipótesis es que la evaluación semicuantitativa de la extensión de la necrosis transmural es el mejor índice de resonancia para predecir el pronóstico a largo plazo tras un infarto con elevación del segmento ST.

Métodos: Se realizó un estudio cuantitativo con resonancia a 206 pacientes consecutivos tras un infarto con elevación del segmento ST. También se evaluó semicuantitativamente (número de segmentos alterados, modelo de 17 segmentos) edema, contractilidad basal y tras dobutamina, perfusión de primer paso, obstrucción microvascular y extensión de la necrosis transmural.

Resultados: Durante el seguimiento (mediana, 51 meses), 29 pacientes sufrieron un primer evento cardiaco adverso (8 muertes cardiacas, 11 infartos y 10 reingresos por insuficiencia cardiaca). Estos eventos se asociaron con mayor alteración de los índices de resonancia. Tras un ajuste multivariable, la extensión de la necrosis transmural fue el único índice de resonancia con asociación independiente con los eventos cardiacos adversos (razón de riesgos = 1,34 [1,19-1,51] por cada segmento con necrosis transmural > 50%; $p < 0{,}001$).

Conclusiones: Un sencillo análisis semicuantitativo de la extensión de la necrosis transmural es el índice de resonancia cardiaca más potente para predecir el pronóstico a largo plazo tras un infarto agudo de miocardio con elevación del segmento ST.

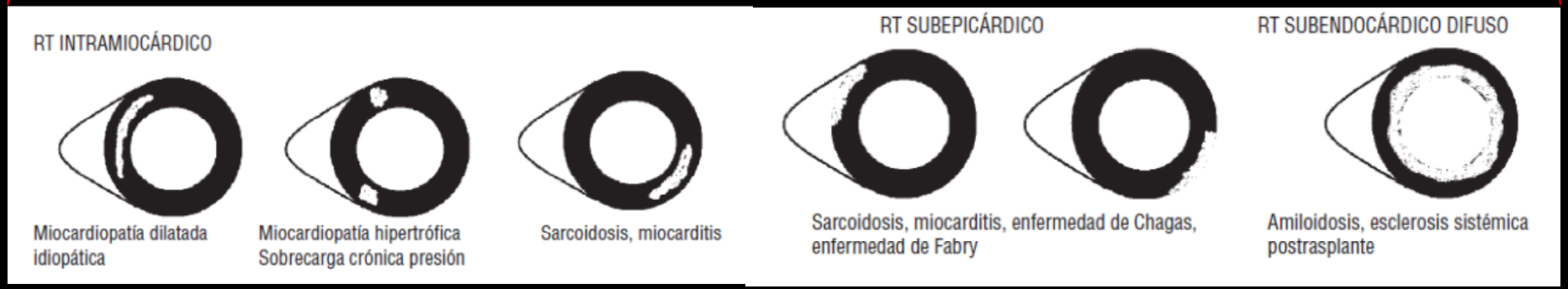
Miocardiopatía no isquémica
Diagnóstico

Secuencia Cine-RM

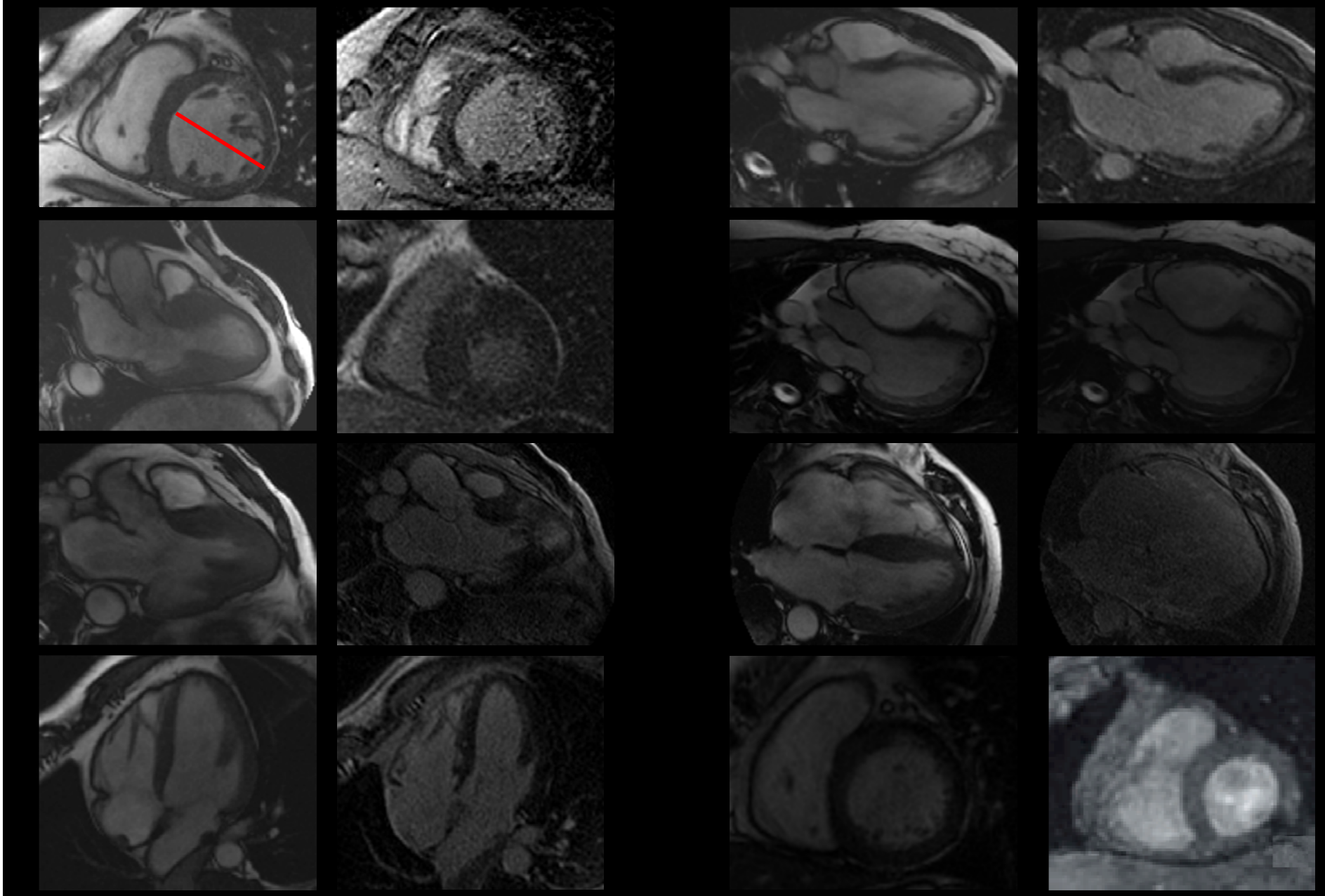
Morfología Característica

Patrón de realce de gadolinio

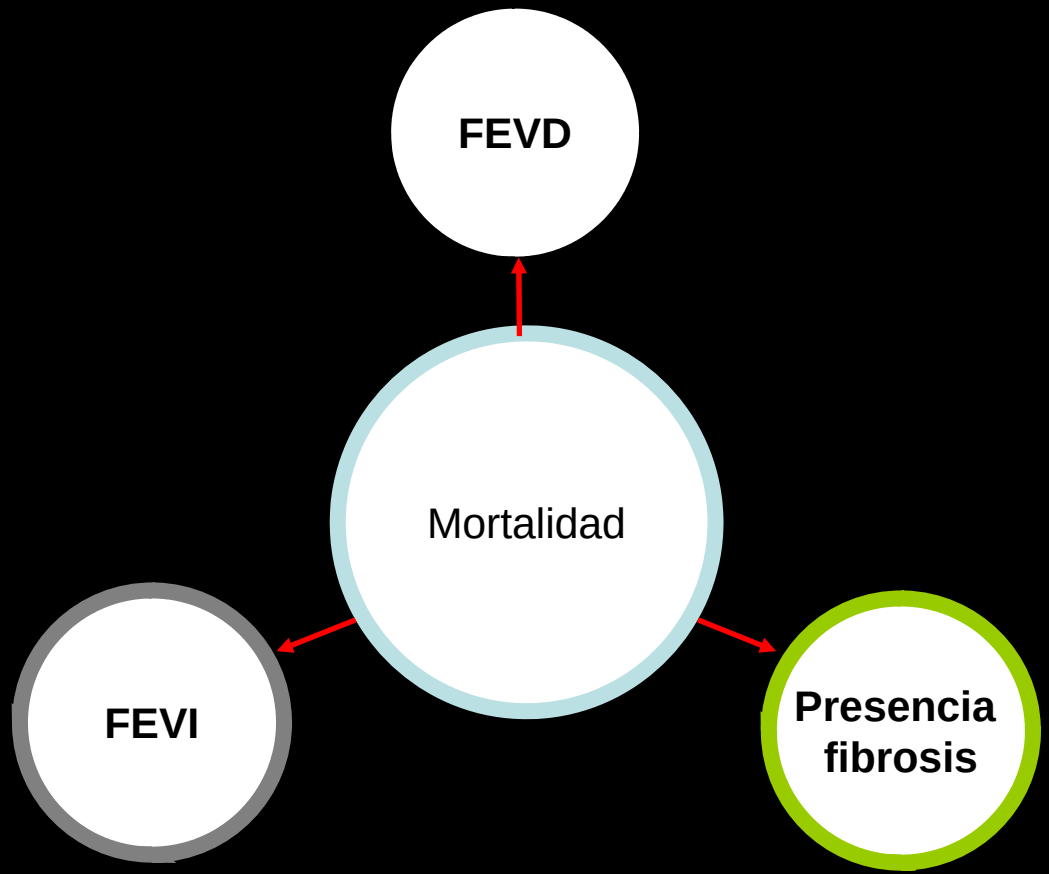
Diagnóstico



No isquémica

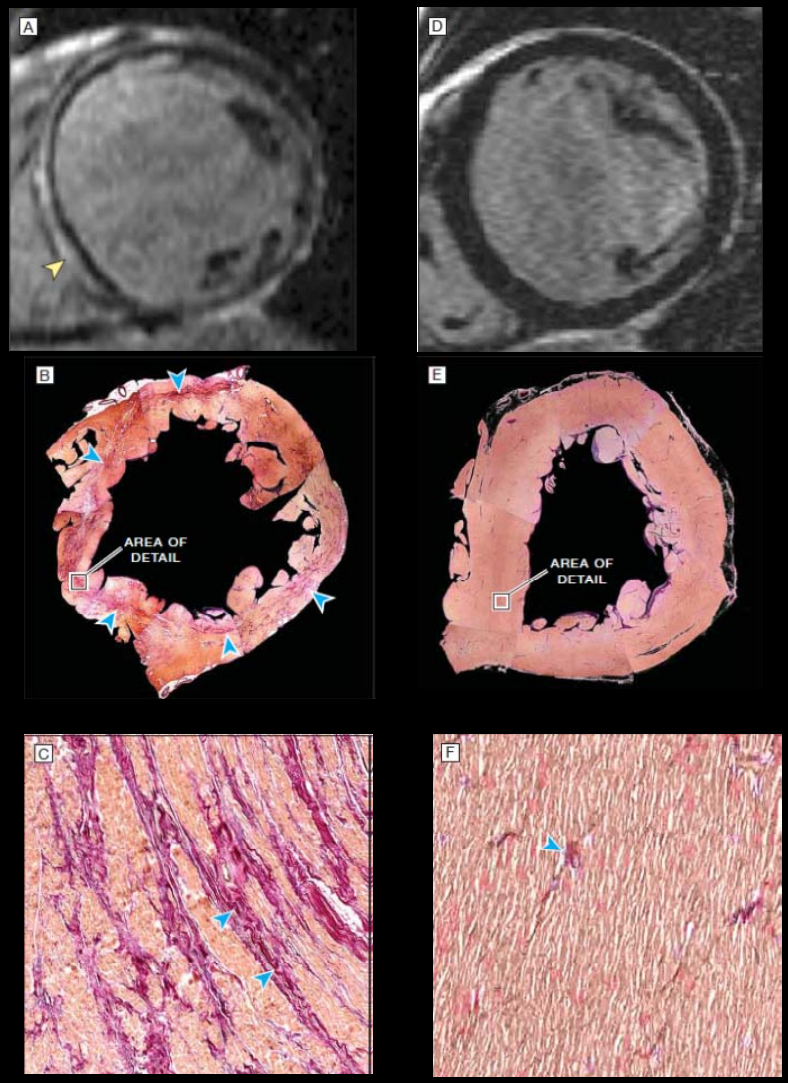


Valoración pronóstica en pacientes con miocardiopatía dilatada idiopática

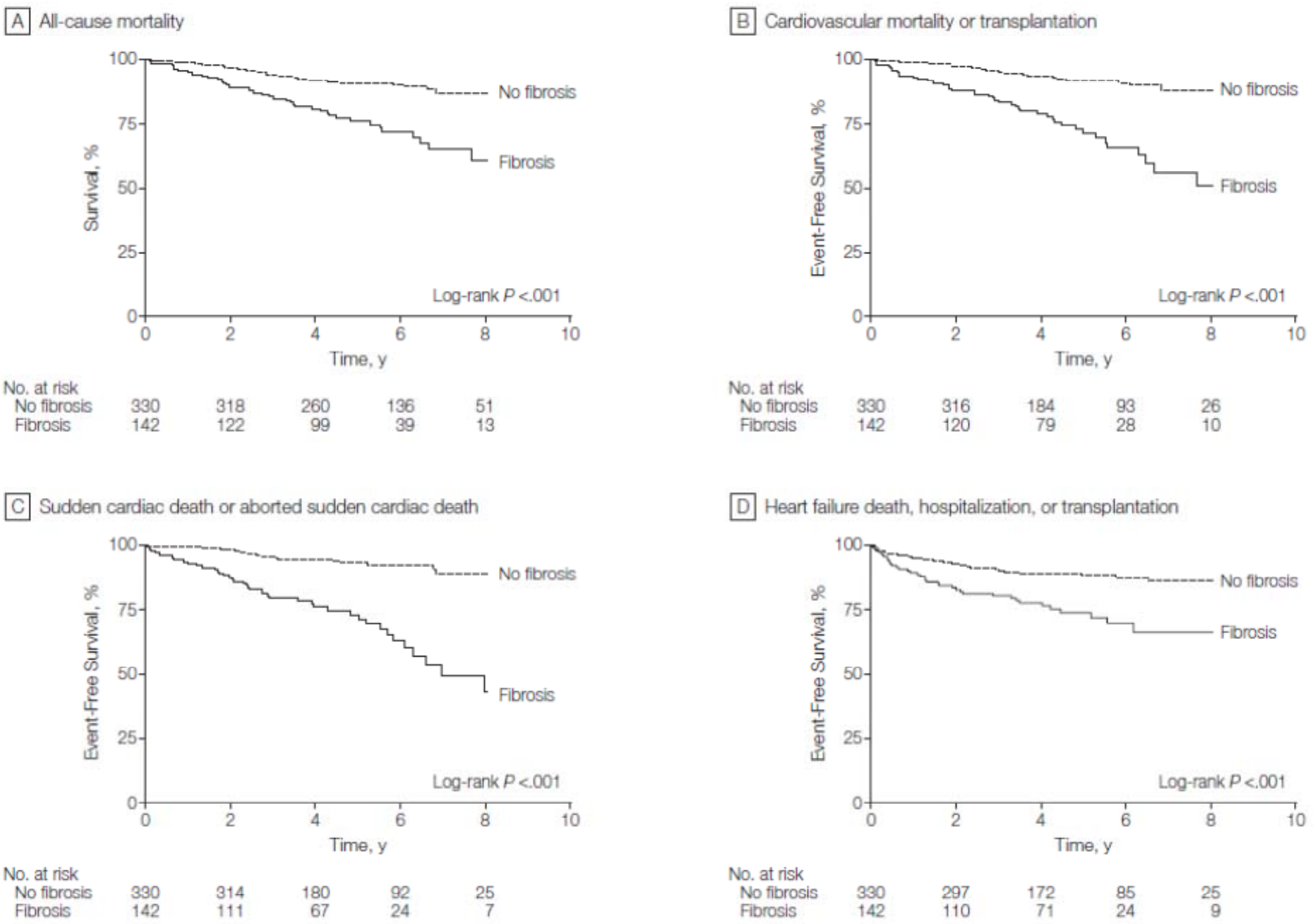


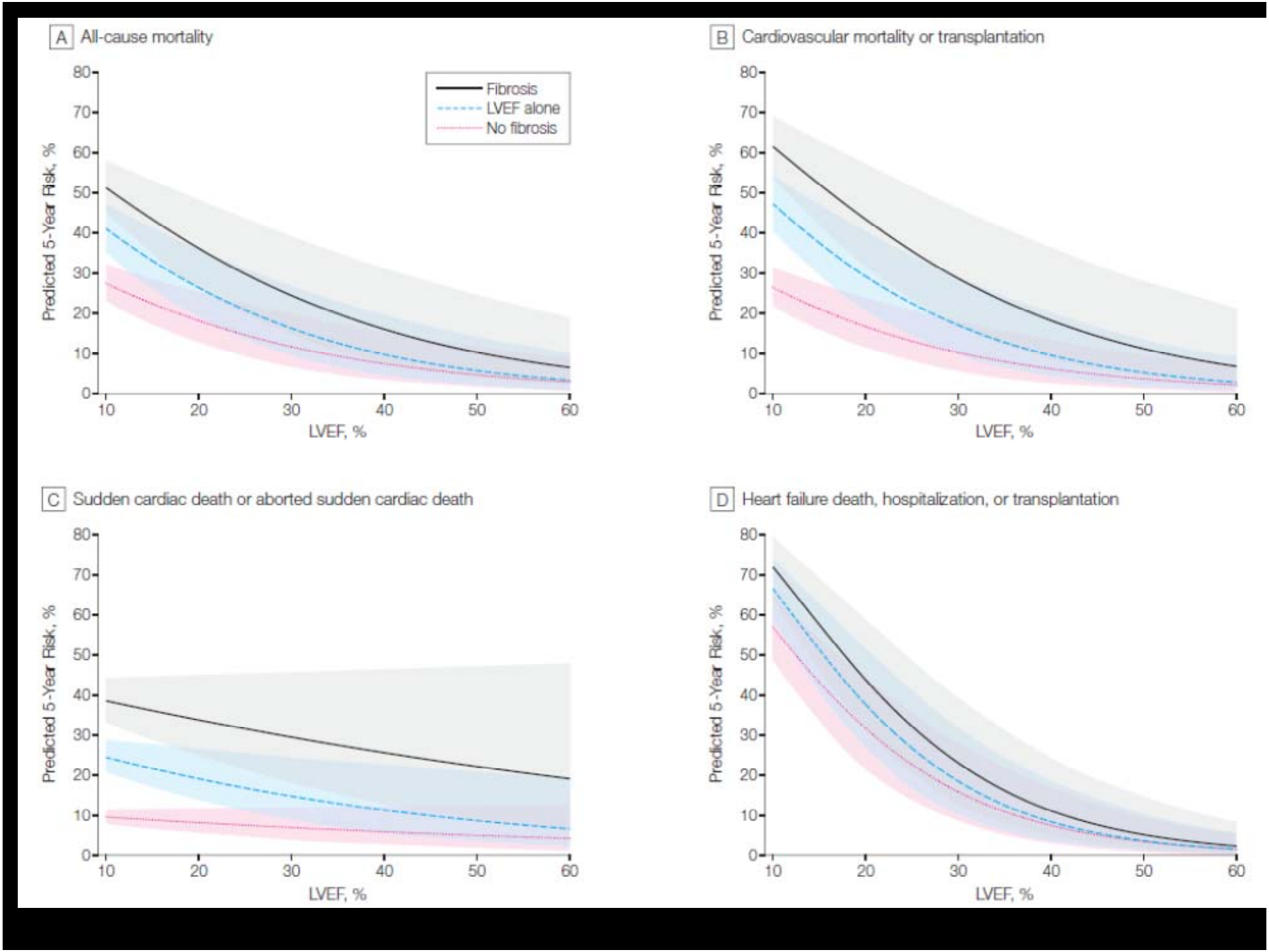
Association of Fibrosis With Mortality and Sudden Cardiac Death in Patients With Nonischemic Dilated Cardiomyopathy

	All Patients (N = 472)	Presence of Midwall Fibrosis: No (n = 330)	Presence of Midwall Fibrosis: Yes (n = 142)	P Value[a]
Age, mean (SD), y	51.1 (14.7)	51.2 (15.0)	50.9 (14.1)	.84
Male sex, No. (%)	324 (68.6)	214 (64.9)	110 (77.5)	.007
Diabetes, No. (%)	35 (7.4)	27 (8.2)	8 (5.6)	.33
Smoker, No. (%)	94 (19.9)	65 (19.7)	29 (20.4)	.86
Medical history, No. (%)				
VF or sustained VT	25 (5.3)	11 (3.3)	14 (9.9)	.004
Atrial fibrillation	82 (17.4)	59 (17.9)	23 (16.2)	.66
Alcohol excess[b]	59 (12.5)	41 (12.4)	18 (12.7)	.94
Family history of DCM, No. (%)	36 (7.6)	21 (6.4)	15 (10.6)	.12
Heart rate, mean (SD), beats/min	74.4 (14.7)	74.0 (14.2)	75.3 (15.7)	.35
Blood pressure, mean (SD), mm Hg				
Systolic	120.1 (18.7)	122.2 (18.9)	115.1 (17.8)	<.001
Diastolic	72.8 (11.2)	73.7 (11.2)	70.8 (10.8)	.009
Left bundle-branch block, No. (%)	129 (27.3)	86 (26.1)	43 (30.3)	.35
NYHA functional class, No. (%)				
I	194 (41.1)	148 (44.9)	46 (32.4)	.03
II	174 (36.9)	120 (36.4)	54 (38.0)	
III	95 (20.1)	57 (17.3)	38 (26.8)	
IV	9 (1.9)	5 (1.5)	4 (2.8)	
Medications at baseline, No. (%)				
ACE inhibitor or ARB	427 (90.5)	293 (88.8)	134 (94.4)	.06
β-Blocker	322 (68.2)	223 (67.6)	99 (69.7)	.65
Loop diuretic	243 (51.5)	145 (43.9)	98 (69.0)	<.001
Aldosterone antagonist	150 (31.8)	93 (28.2)	57 (40.1)	.01
Aspirin	148 (31.4)	103 (31.2)	45 (31.7)	.92
Warfarin	130 (27.5)	90 (27.3)	40 (28.2)	.84
Statin	128 (27.1)	95 (28.8)	33 (23.2)	.21
Digoxin	77 (16.3)	48 (14.6)	29 (20.4)	.11
Amiodarone	36 (7.6)	21 (6.4)	15 (10.6)	.12
Cardiovascular magnetic resonance measurements, mean (SD)				
LV end-diastolic volume index, mL/m^2	135.1 (44.3)	128.9 (39.1)	149.7 (51.7)	<.001
LV end-systolic volume index, mL/m^2	88.6 (45.6)	81.7 (40.6)	104.7 (52.3)	<.001
LV stroke volume, mL	92.1 (28.4)	93.3 (27.5)	89.3 (30.3)	.16
LV ejection fraction, %	37.2 (13.1)	39.1 (12.5)	32.8 (13.4)	<.001
LV mass index, g/m^2	101.3 (29.8)	99.3 (30.0)	106.1 (28.8)	.02
Extent of late gadolinium enhancement, median (IQR), %			2.5 (1.2-4.8)	



JAMA, March 6, 2013—Vol 309, No. 9
Corrected on June 12, 2013


A All-cause mortality
100
75
50
25
0
Survival, %
No fibrosis
Fibrosis
Log-rank P <.001
0 2 4 6 8 10
Time, y
No. at risk
No fibrosis 330 318 260 136 51
Fibrosis 142 122 99 39 13
B Cardiovascular mortality or transplantation
Event-Free Survival, %
No fibrosis
Fibrosis
Log-rank P <.001
Time, y
No. at risk
No fibrosis 330 316 184 93 26
Fibrosis 142 120 79 28 10
C Sudden cardiac death or aborted sudden cardiac death
Event-Free Survival, %
No fibrosis
Fibrosis
Log-rank P <.001
Time, y
No. at risk
No fibrosis 330 314 180 92 25
Fibrosis 142 111 67 24 7
D Heart failure death, hospitalization, or transplantation
Event-Free Survival, %
No fibrosis
Fibrosis
Log-rank P <.001
Time, y
No. at risk
No fibrosis 330 297 172 85 25
Fibrosis 142 110 71 24 9


A All-cause mortality
B Cardiovascular mortality or transplantation
C Sudden cardiac death or aborted sudden cardiac death
D Heart failure death, hospitalization, or transplantation
Fibrosis
LVEF alone
No fibrosis
Predicted 5-Year Risk, %
LVEF, %
0
10
20
30
40
50
60
70
80

CONCLUSIONS

Assessment of midwall fibrosis with LGE-CMR provided independent prognostic information in patients with nonischemic dilated cardiomyopathy. LGE-CMR imaging improved risk stratification beyond LVEF for all-cause mortality and SCD. The potential clinical utility of midwall fibrosis evaluated by LGE-CMR in the risk stratification of patients with dilated cardiomyopathy requires further investigation.

Predicted Risk With LVEF Alone, %	Predicted Risk With LVEF and Midwall Fibrosis Status, % 0-5	5-10	10-20	≥20	Total
Deaths					
0-5	2	0	0	0	2
5-10	0	5	5	0	10
10-20	0	2	11	11	24
≥20	0	0	7	30	37
Total	2	7	23	41	73
Survivors					
0-5	22	4	0	0	26
5-10	46	76	26	0	148
10-20	0	39	66	21	126
≥20	0	0	32	67	99
Total	68	119	124	88	399

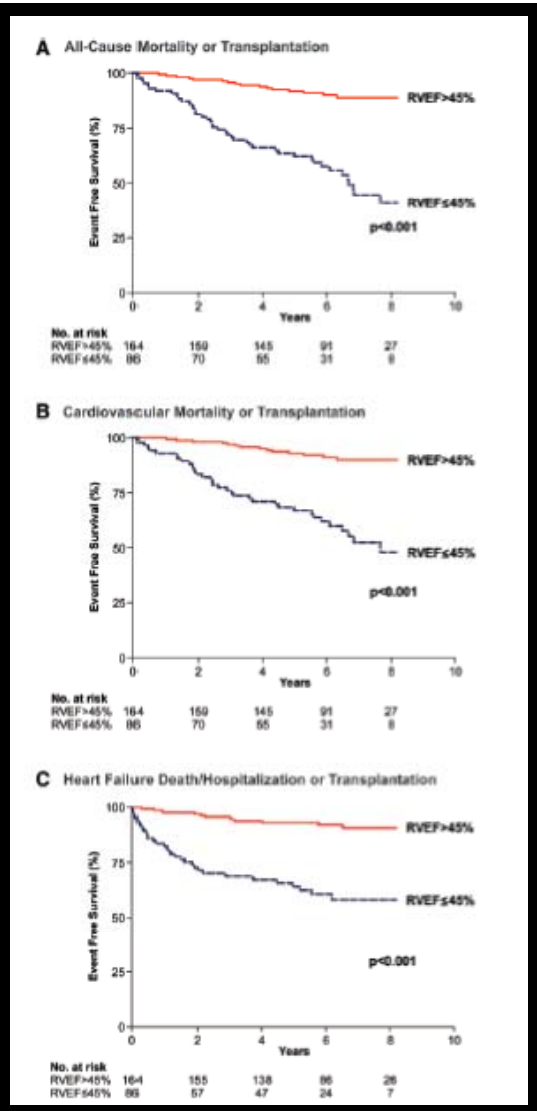
[a] Values represent the number of patients in each risk category (0%-5%, 5%-10%, 10%-20% and ≥20%) according to risk model based on LVEF alone and risk model based on LVEF and midwall fibrosis status (presence or absence) for patients who died or survived during follow-up. Correct reclassifications are shaded light gray and incorrect reclassifications are shaded dark gray.

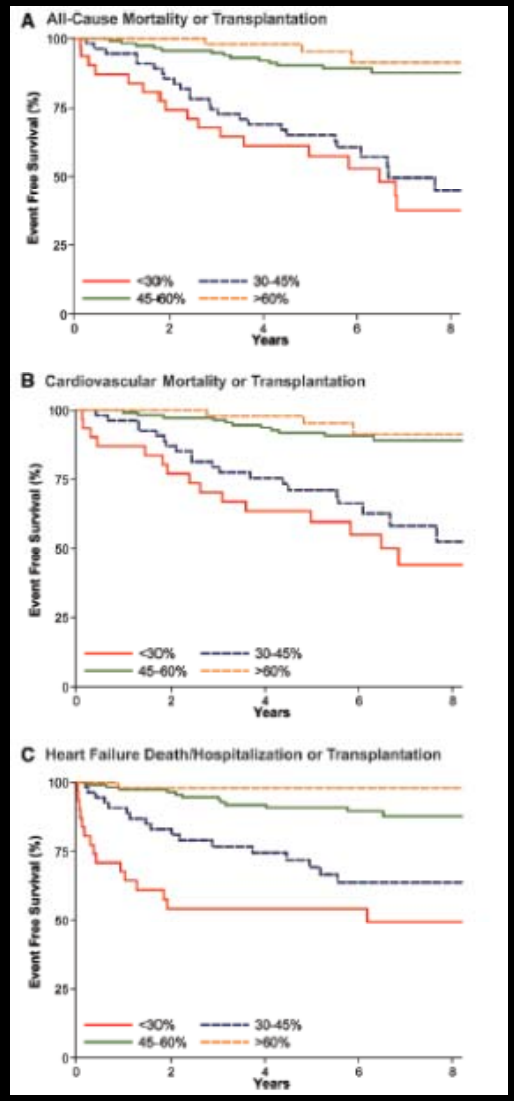
Predicted Risk With LVEF Alone, %	Predicted Risk With LVEF Plus Midwall Fibrosis Status, % 0-15	>15	Total
Patients With Arrhythmic Event			
Predicted risk with LVEF alone			
0-15	12	23	35
>15	11	19	30
Total	23	42	65
Patients Without Arrhythmic Event			
Predicted risk with LVEF alone			
0-15	218	46	264
>15	89	54	143
Total	307	100	407

The Prevalence and Prognostic Significance of Right Ventricular Systolic Dysfunction in Nonischemic Dilated Cardiomyopathy

Table 1. Baseline Characteristics of the Study Group According to the Presence (RVSD+) or Absence (RVSD−) of Right Ventricular Systolic Dysfunction

Characteristics	All Patients (n=250)	RVSD− (n=164)	RVSD+ (n=86)	*P* Value
Age, y	50.8±14.0	51.9±13.5	48.7±14.6	0.081
Male sex, n (%)	171 (68.4)	108 (65.9)	63 (73.3)	0.232
History of VF or sustained VT, n (%)	15 (6.0)	9 (5.5)	6 (7.0)	0.638
History of AF, n (%)	37 (14.8)	20 (12.2)	17 (19.8)	0.109
Diabetes mellitus, n (%)	21 (8.4)	13 (7.9)	8 (9.30	0.710
Smoker, n (%)	47 (18.8)	27 (16.5)	20 (23.3)	0.192
History of alcohol excess, n (%)	32 (12.8)	17 (10.4)	15 (17.4)	0.112
Family history of DCM, n (%)	18 (7.2)	15 (9.2)	3 (3.5)	0.100
Heart rate, beats/min	75.6±14.9	72.4±13.4	81.9±15.7	<0.001
Systolic blood pressure, mm Hg	119.1±18.9	123.0±17.4	111.6±19.4	<0.001
Diastolic blood pressure, mm Hg	72.6±11.6	73.9±10.9	70.2±12.6	0.017
LBBB, n (%)	66 (26.4)	51 (31.1)	15 (17.4)	0.020
NYHA functional class, n (%)				
I	103 (41.2)	84 (51.2)	19 (22.1)	<0.001
II	96 (38.4)	63 (38.4)	33 (38.4)	
III	45 (18.0)	16 (9.8)	29 (33.7)	
IV	6 (2.4)	1 (0.6)	5 (5.8)	
Medications at baseline, n (%)				
Aspirin	82 (32.8)	58 (35.4)	24 (27.9)	0.233
Warfarin	64 (25.6)	28 (17.1)	36 (41.9)	<0.001
β-blocker	162 (64.8)	98 (59.8)	64 (74.4)	0.021
ACE-inhibitor or ARB	231 (92.4)	146 (89.0)	85 (98.8)	0.005
Amiodarone	26 (10.4)	19 (11.6)	7 (8.1)	0.397
Digoxin	42 (16.8)	18 (11.0)	24 (27.9)	0.001
Statin	58 (23.2)	39 (23.8)	19 (22.1)	0.764
Loop diuretic	128 (51.2)	66 (40.2)	62 (72.1)	<0.001
Aldosterone antagonist	82 (32.8)	38 (23.2)	44 (51.2)	<0.001
Cardiovascular magnetic resonance measurements				
LVEDV index, mL/m²	132.9±43.7	122.7±36.1	152.3±50.2	<0.001
LVESV index, mL/m²	87.2±45.0	73.4±34.7	113.6±50.5	<0.001
LV stroke volume, mL	90.6±29.1	97.5±25.3	77.5±31.5	<0.001
LVEF, %	37.1±13.2	42.2±10.7	27.4±12.1	<0.001
LV mass index, g/m²	105.3±31.8	98.8±26.1	117.7±37.7	<0.001
RVEDV index, mL/m²	89.8±26.4	80.8±17.1	107.1±32.0	<0.001
RVESV index, mL/m²	48.5±25.5	35.4±9.9	73.5±27.6	<0.001
RV stroke volume, mL	82.0±27.5	90.0±24.6	66.8±26.3	<0.001
RVEF, %	48.2±13.9	56.6±7.2	32.3±8.9	<0.001
Midwall fibrosis, n (%)	71 (28.4)	39 (23.8)	32 (37.2)	0.025
RVEF subgroup, n (%)				
RVEF>60%	49 (20)			
RVEF 45%–60%	115 (46)			
RVEF 30%–45%	55 (22)			
RVEF<30%	31 (12)			





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Outcome	RVSD− (n=164)	RVSD + (n=86)	Hazard Ratio (95% CI)	*P* Value
Primary end point, No. of patients (%)				
All-cause mortality or cardiac transplantation	17 (10.4)	42 (48.8)	5.90 (3.35–10.37)	<0.001
All-cause mortality	16 (9.8)	36 (41.9)	5.51 (3.06–9.94)	<0.001
Cardiac transplantation	1 (0.6)	6 (7.0)	13.01 (1.56–108.26)	0.018
Secondary end points, No. of patients (%)				
Cardiovascular mortality or cardiac transplantation	15 (9.2)	35 (40.7)	5.62 (3.07–10.30)	<0.001
Cardiovascular mortality	14 (8.5)	29 (33.7)	5.12 (2.70–9.70)	<0.001
Heart failure death, heart failure hospitalization, or cardiac transplantation*	13 (7.9)	32 (37.2)	6.13 (3.21–11.70)	<0.001
Heart failure death	3 (1.8)	17 (19.8)	14.19 (4.15–48.45)	<0.001
Heart failure hospitalization	12 (7.3)	27 (31.4)	5.61 (2.84–11.10)	<0.001

Conclusions

RVSD is common in DCM with a prevalence of 34% in our cohort. Detection of RVSD by CMR represents a powerful independent predictor of transplant-free survival and adverse HF outcomes in DCM. Routine functional evaluation of the right ventricle is therefore warranted in the CMR examination of DCM patients for comprehensive phenotypic characterization and risk stratification. Further study is required to evaluate whether amelioration of RVSD, with treatments that target RV performance, may yet improve prognosis.

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