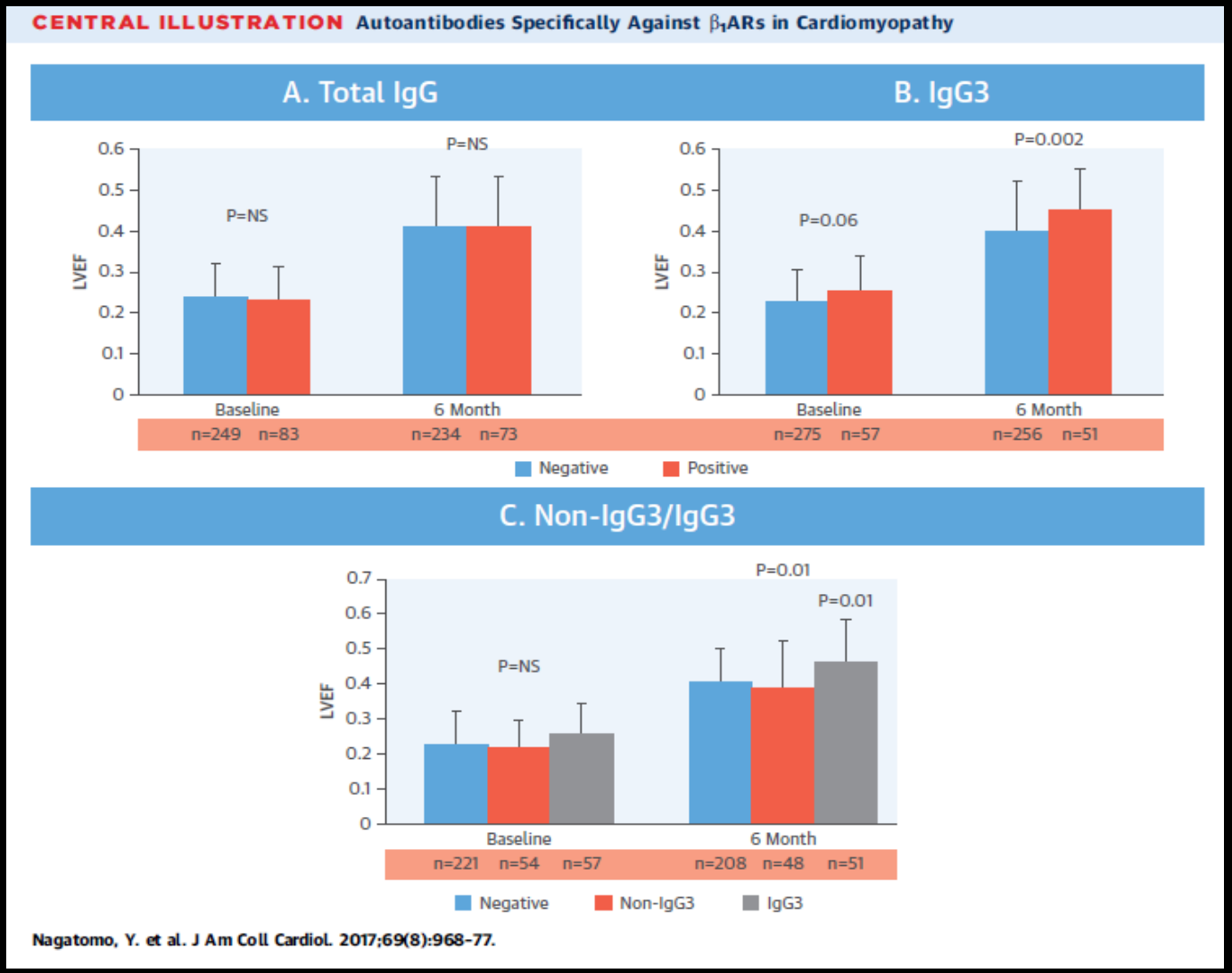
ΘΕΡΑΠΕΥΤΙΚΕΣ ΕΞΕΛΙΞΕΙΣ 2017

Ιωάννης Α. Παρασκευαΐδης

Myocardial Recovery in Pts With Systolic HF and Autoantibodies Against b1-Adrenergic Receptors

The IMAC-2 Study Y. Nagatomo et al. J Am Coll Cardiol 2017;69:968–77

Peripheral blood samples were drawn at enrollment in patients with recent-onset cardiomyopathy (LVEF<0.40%, <6 months). The presence of IgG and IgG3-b1AR-AAb was determined, and echo were assessed, at baseline and 6 months. Patients were followed up for 48 months



Nagatomo, Y. et al. J Am Coll Cardiol. 2017;69(8):968-77.

Myocardial Recovery in Pts With Systolic HF and Autoantibodies Against b1-Adrenergic Receptors

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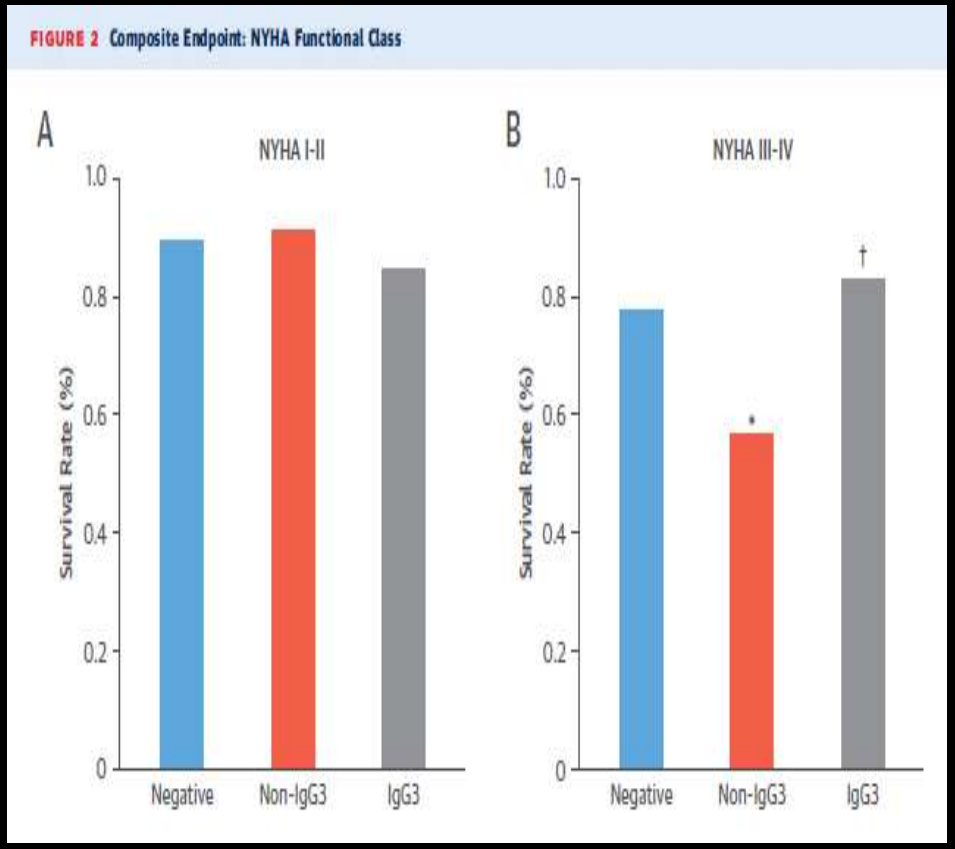
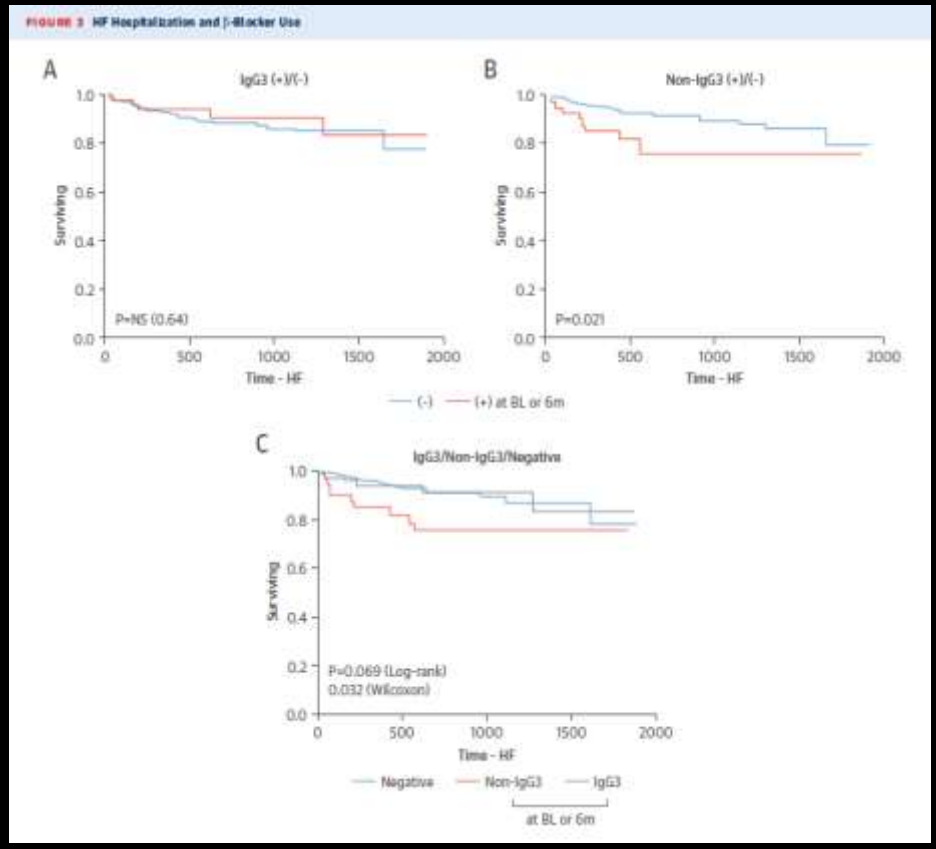
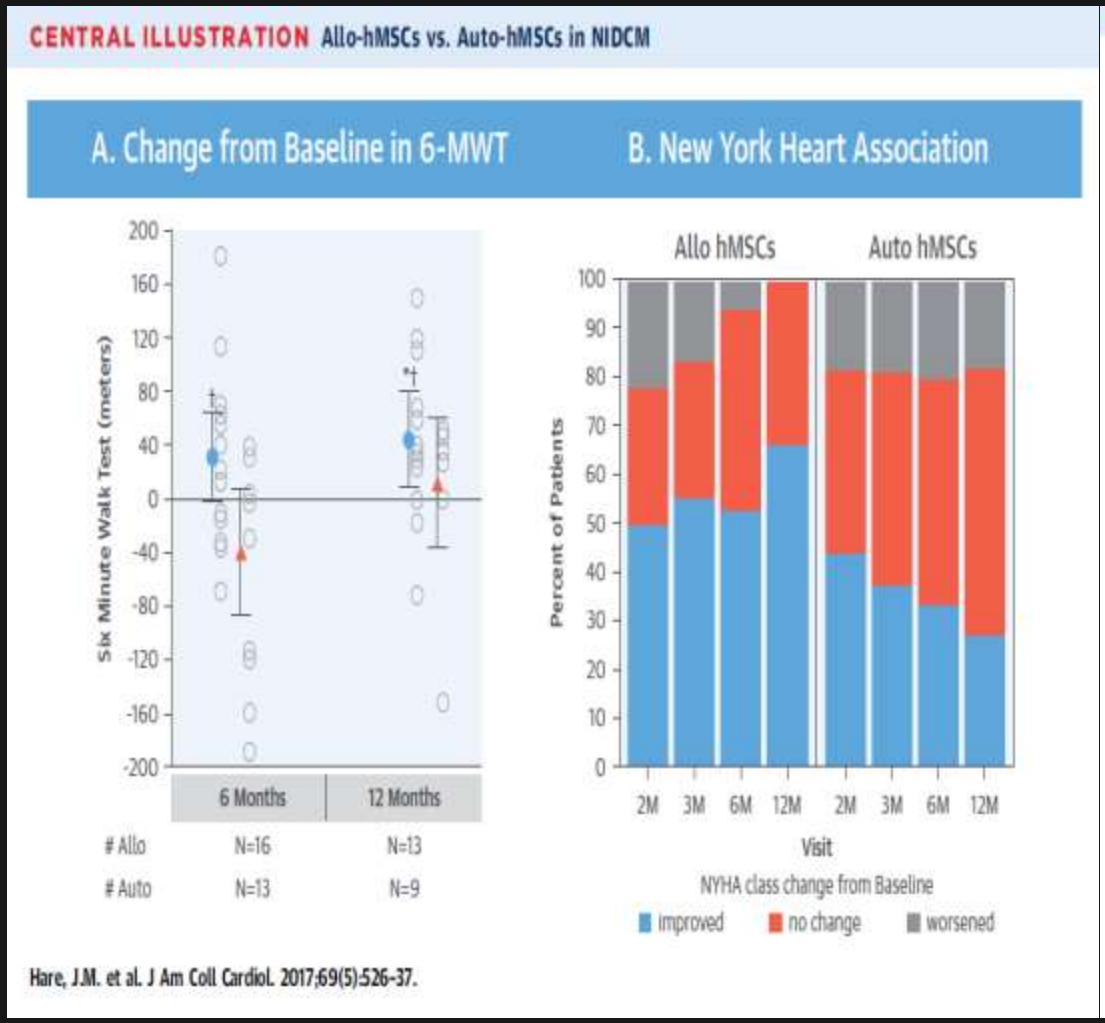


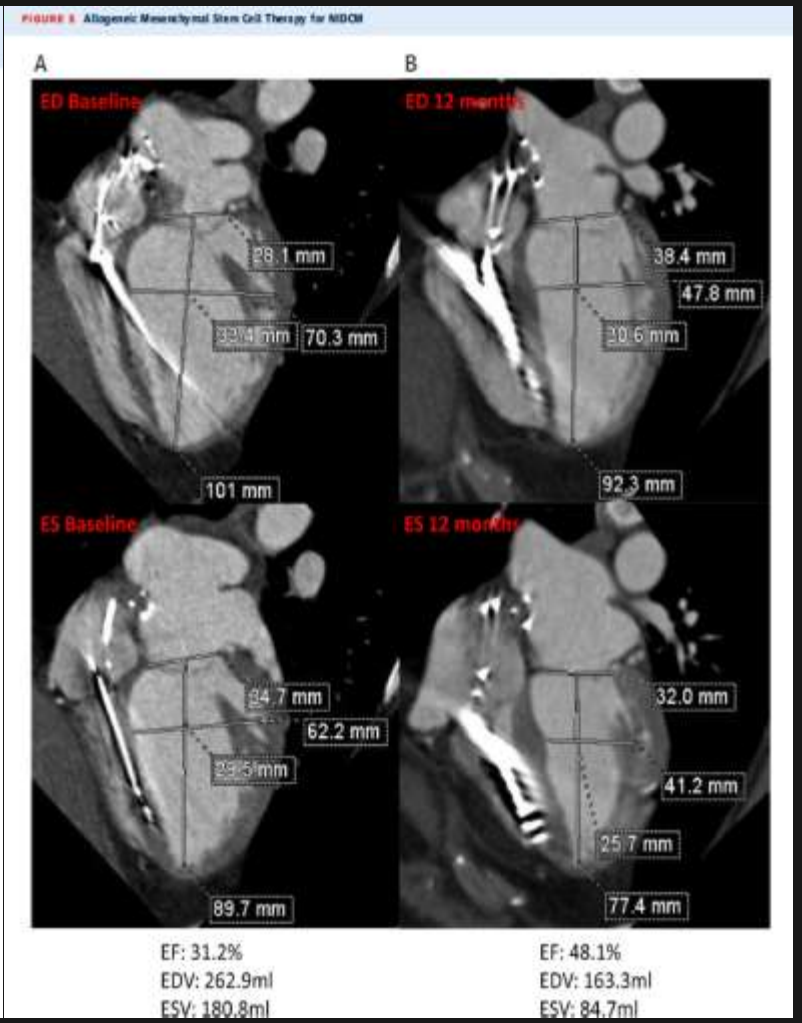


IgG3-b1AR-AAbs were associated with more favorable myocardial recovery in patients with recent-onset cardiomyopathy.

Randomized Comparison of Allogeneic Versus Autologous Mesenchymal Stem Cells for Non-ischemic DCM

POSEIDON-DCM Trial J.M. Hare J Am Coll Cardiol 2017;69:526–37

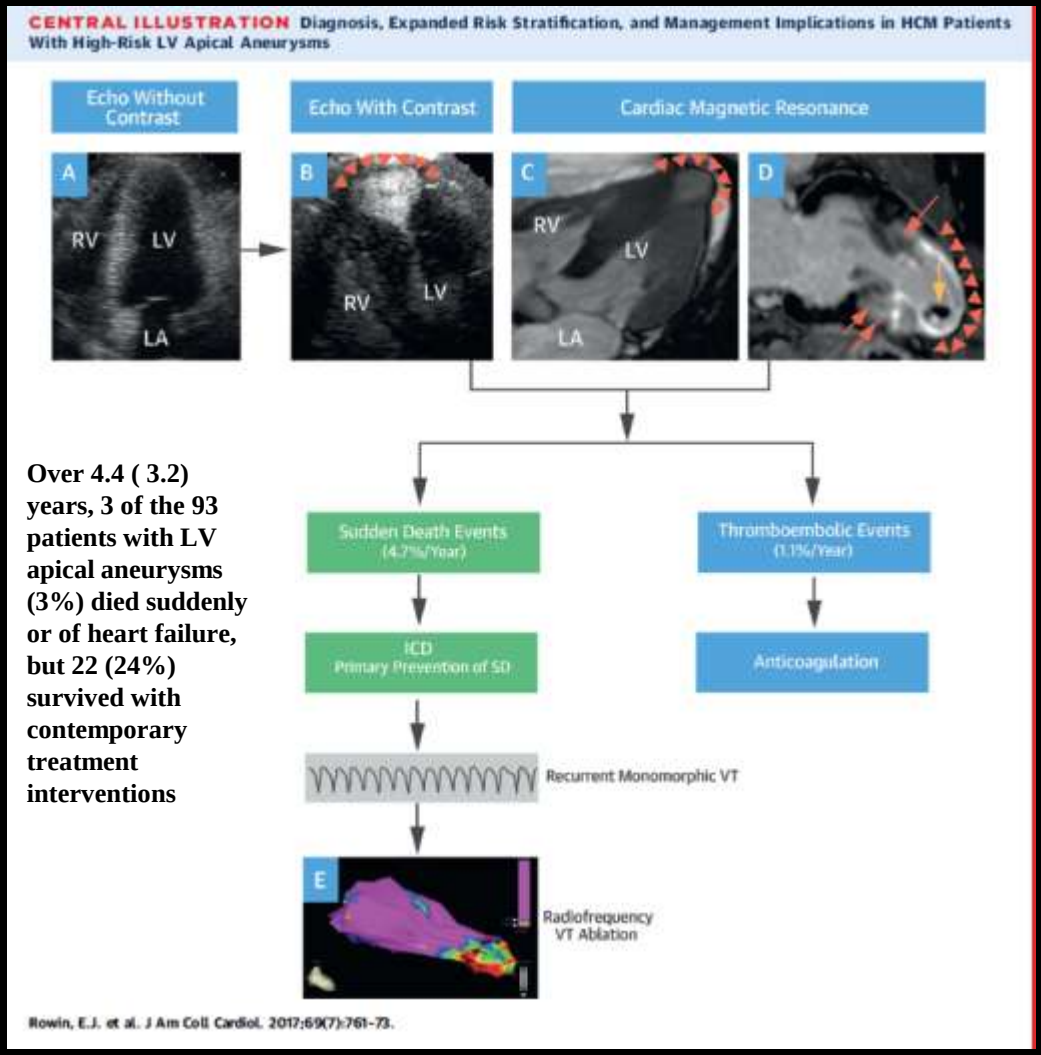




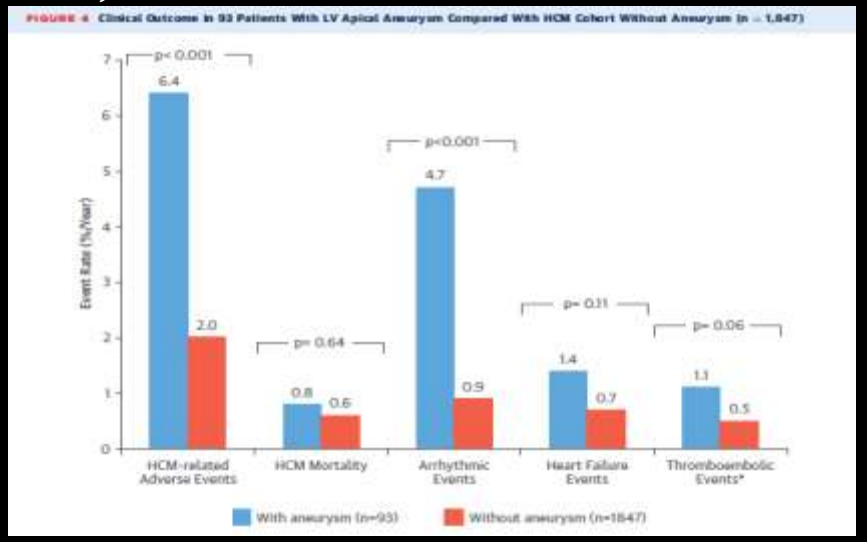
These findings demonstrated safety and clinically meaningful efficacy of allo-hMSC versus auto-hMSC in NIDCM patients.

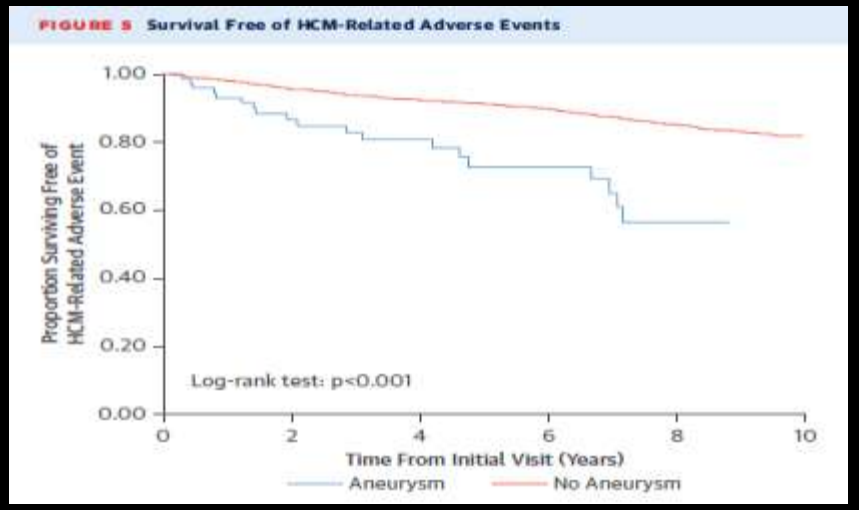
Hypertrophic Cardiomyopathy With Left Ventricular Apical Aneurysm

E.J. Rowin J Am Coll Cardiol 2017;69:761–73



Over 4.4 (3.2) years, 3 of the 93 patients with LV apical aneurysms (3%) died suddenly or of heart failure, but 22 (24%) survived with contemporary treatment interventions





HCM patients with LV apical aneurysms are at high risk for arrhythmic sudden death and thromboembolic events. Identification of this phenotype expands risk stratification and can lead to effective treatment interventions for potentially life-threatening complications

Cardiovascular causes of maternal sudden death. Sudden Arrhythmic Death Syndrome is leading cause in UK

D. Krexi et al. Eur J Obstet Gynecol Reprod Biol. 2017 Oct;217:177

80 cases mean age was 30±7 years of sudden unexpected death due to cardiac causes in relation to pregnancy and postpartum period in a database of 4678 patients were found and examined macroscopically and microscopically.

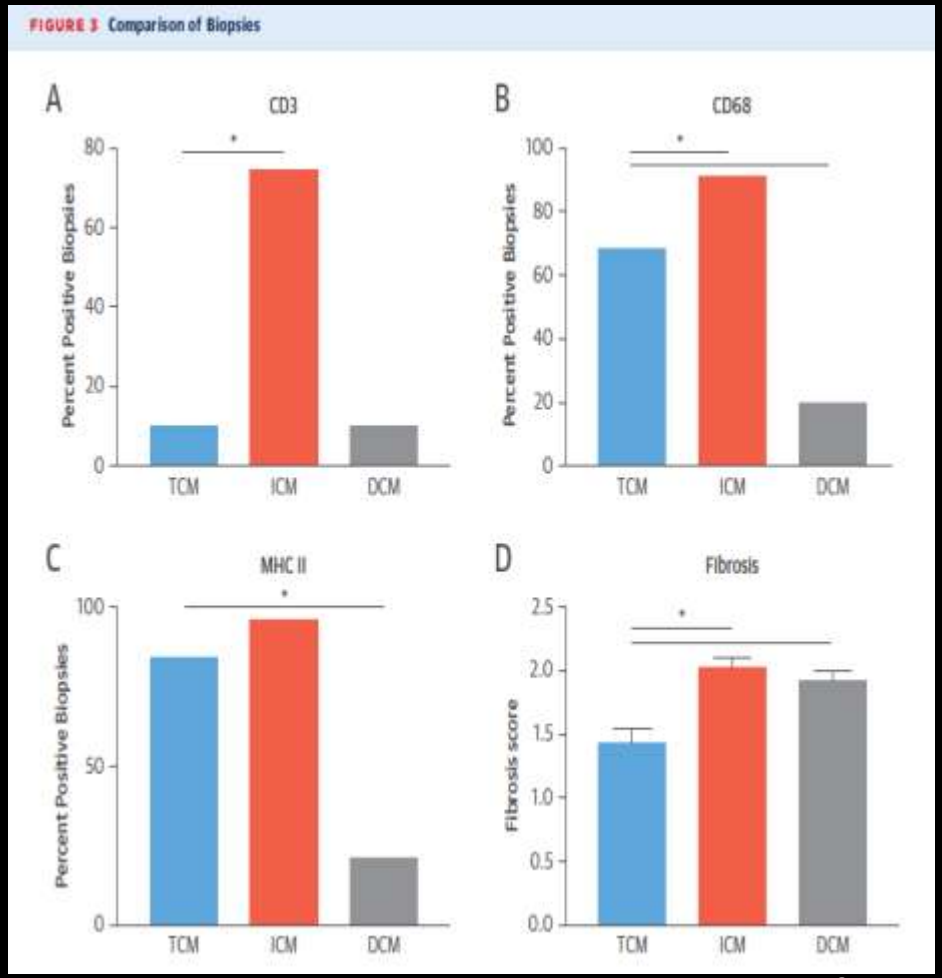
Cause of Death	Total	% cohort
Morphological Normal Heart	43	53.75
Cardiomyopathy	11	13.80
Dissection of Aorta or its	7	8.75
Congenital Heart Disease	2	2.50
Valvular Diseases	3	3.75
Floppy Mitral Valve	2	2.50
Mitral Stenosis	1	1.25
Miscellaneous Causes	14	17.50

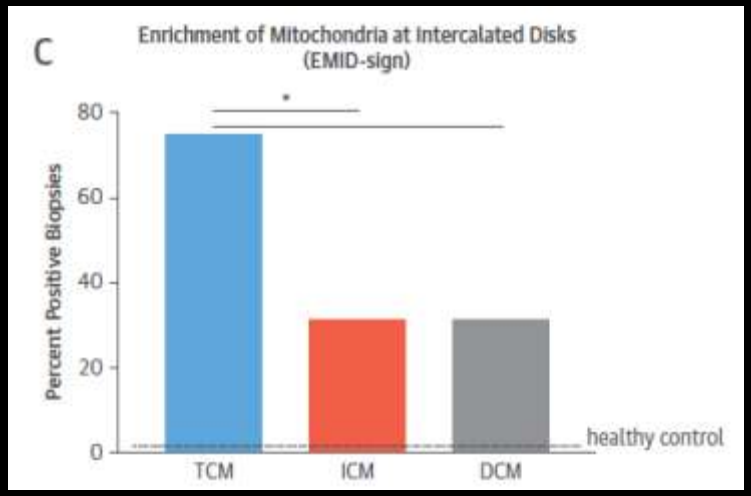
This study highlights sudden cardiac deaths in pregnancy or in the postpartum period, which is mainly due to Sudden Arrhythmic Deaths with underlying channelopathies and cardiomyopathy. We wish to raise awareness of the need of cardiological screening of the family as a result of the diagnosis

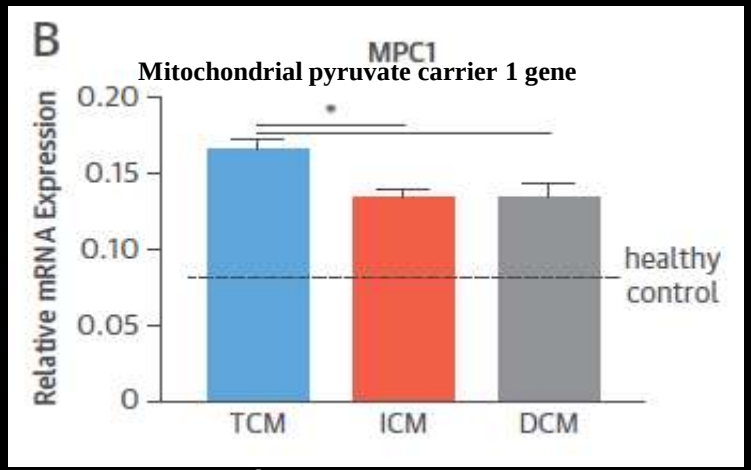
Histopathological and Immunological Characteristics of Tachycardia-Induced Cardiomyopathy

K.A.L. Mueller et al J Am Coll Cardiol 2017;69:2160–72

Nineteen patients with Tachycardia induced Cardiomyopathy (TCM), 79 with Dilated Cardiomyopathy (DCM) , and 91 with Inflammatory Cardiomyopathy (ICM).







MHCII= major histocompatibility complex class II: a set of cell surface proteins essential for the acquired immune system to recognize foreign molecules

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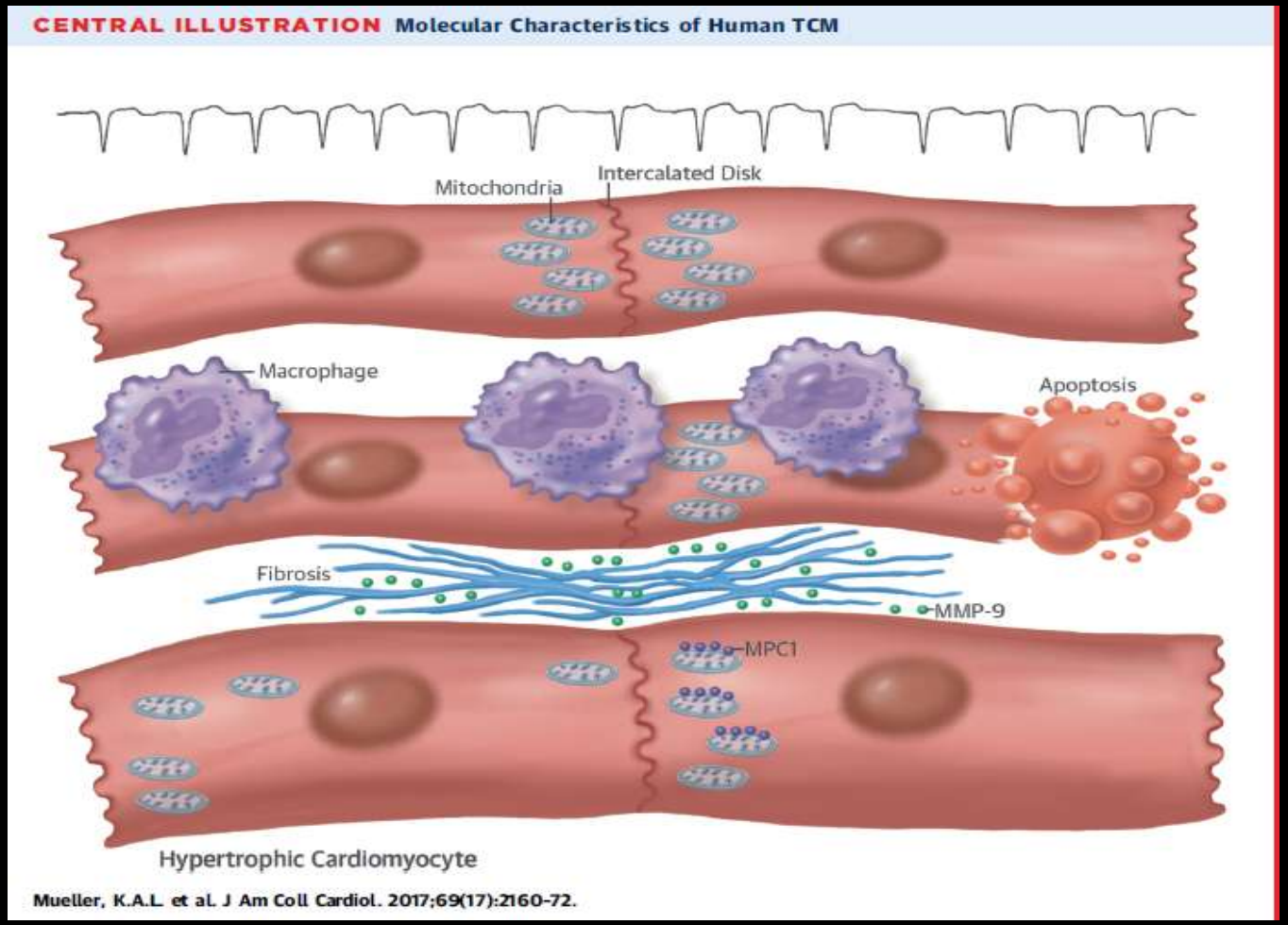
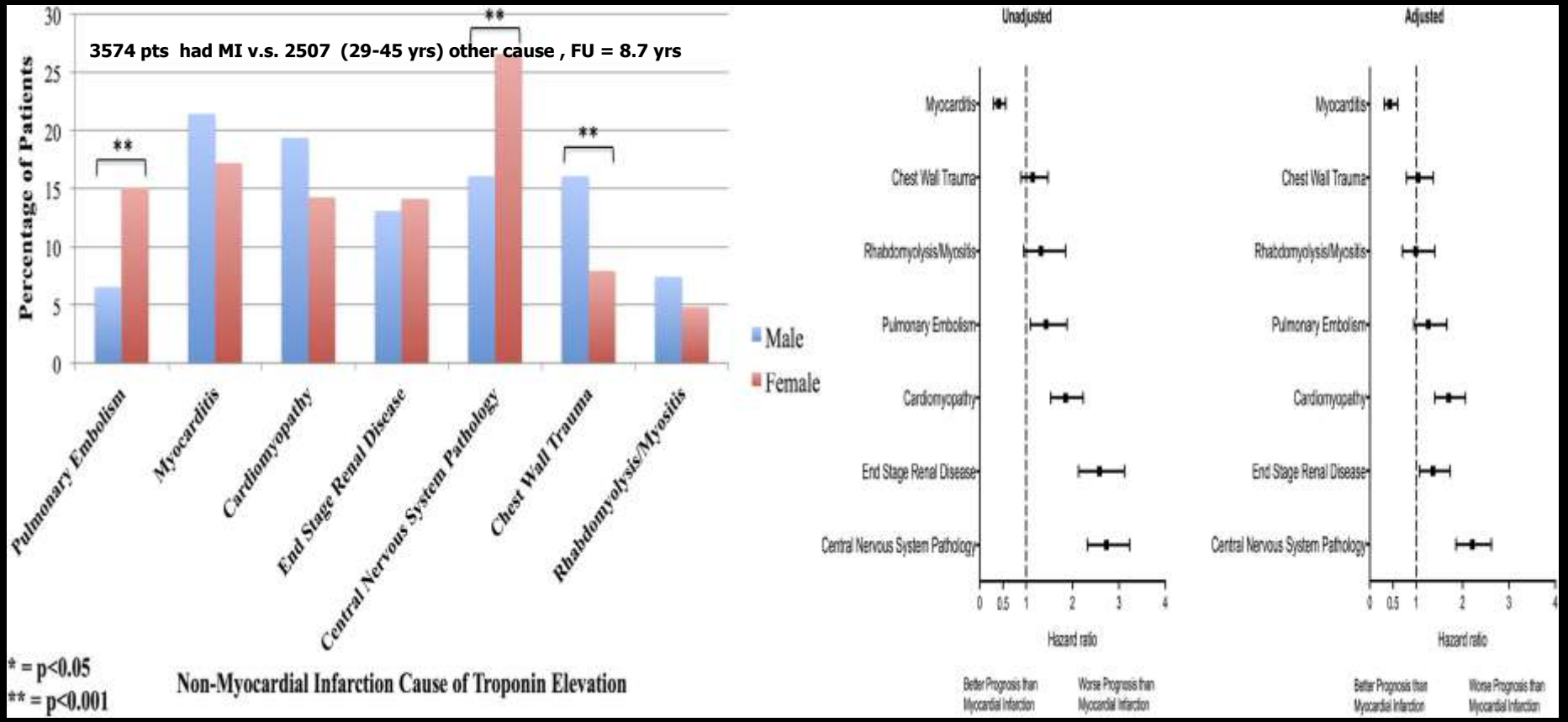


TCM is characterized by changes in cardiomyocyte and mitochondrial morphology accompanied by a macrophage-dominated cardiac inflammation. Further prospective studies are warranted for better characterization of patients with TCM by EMB, which could help identify patients with TCM

Causes of Troponin Elevation and Associated Mortality in Young Patients

C. Wu et al. Am J Med. 2018 Mar;131(3):284-292

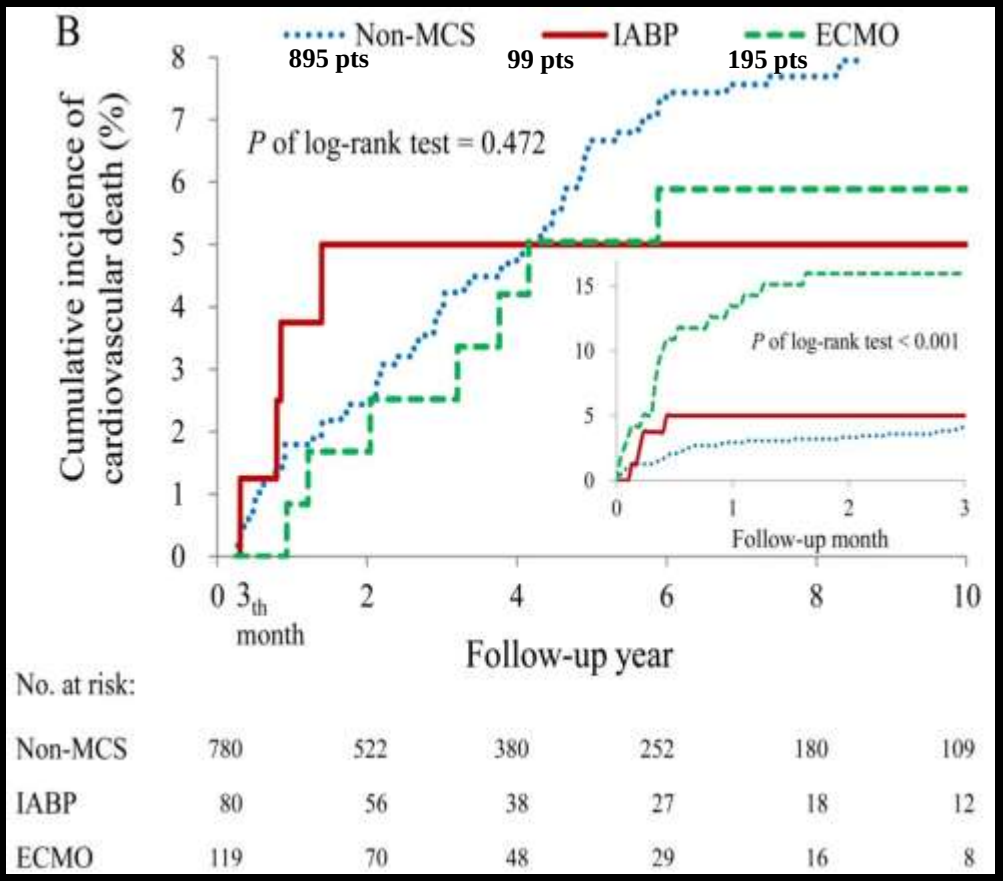


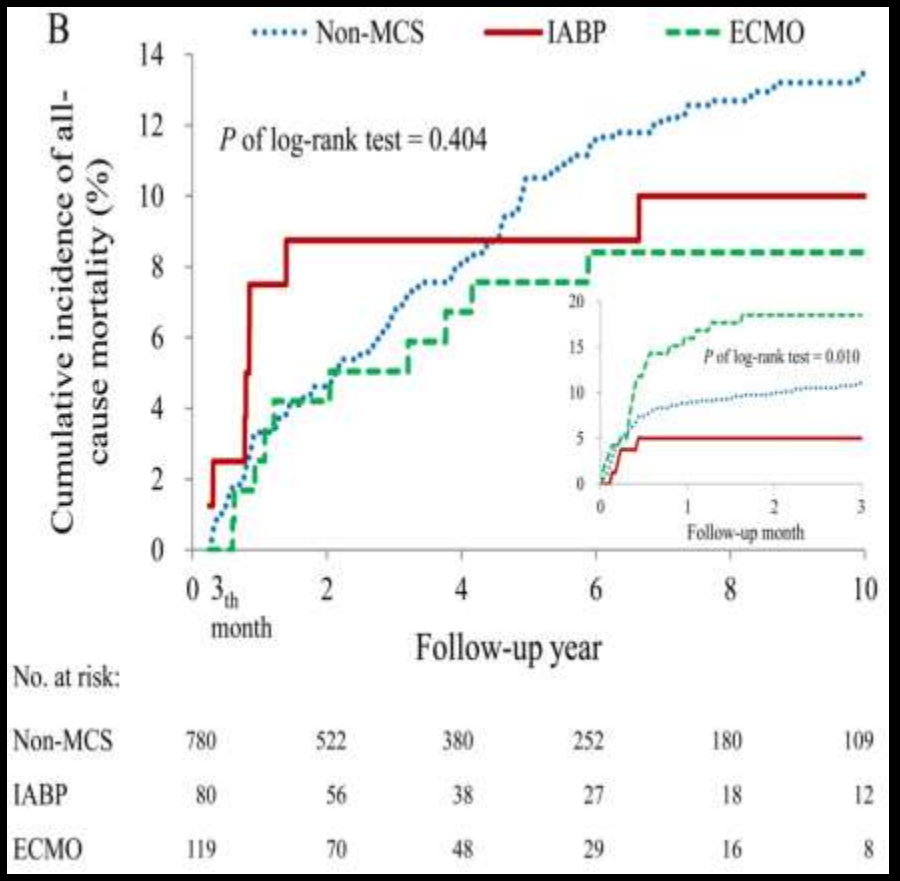
Most non-myocardial infarction causes of troponin elevation are associated with higher all-cause mortality compared with acute myocardial infarction

HF and Mortality of Adult Survivors from Acute Myocarditis Requiring Intensive Care Treatment

A Nationwide Cohort Study (Taiwan)

J.J Chang. et al. Int. J. Med. Sci. 2017, 14(12): 1241-1250





The severity of acute myocarditis did not affect long-term outcomes, however, it was associated with cardiovascular/all-cause death within 3 months after discharge

Extracorporeal membrane oxygenation in adult pts with acute fulminant myocarditis.

X. Liao et al. Herz 2017 doi.org/10.1007/s00059-017-4617-7

The indications

a) high-dose inotropic support comprising dopamine >15 μg × kg–1BWT ×min–1; adrenaline >0.15 μg × kg–1BWT × min–1; and norepinephrine >0.15 μg × kg–1BWT × min–1; mean arterial pressure of <60mm Hg;

b)LVEF < 30%

c) progressively elevated metabolic acidosis and lactic acid level (>3.0mmol/l), with or without IABP support;

d) severe arrhythmia (ventricular tachycardia and ventricular fibrillation) despite multiple antiarrhythmic drugs, hemodynamic instability; and

e) continuous cardiopulmonary resuscitation without recovery of effective spontaneous circulation because of cardiac arrest.

Variable	Group	Total ($n = 33$)	Survivors ($n = 26$)	Nonsurvivors ($n = 7$)	Z	p
ECMO support time (h)	–	72.0 (44.0)	75.0 (42.0)	72.0 (152.0)	−0.26	0.791
Ventilation time (h)	–	120.0 (73.0)	120.0 (54.0)	80.0 (240.0)	−1.26	0.208
IABP support	Yes	6	5 (83.3)	1 (16.7)	–	1.000
BPTV (ml)						
Red blood cell	–	1200.0 (1650.0)	1000.0 (625.0)	3200.0 (4200.0)	−3.94	<0.001*
Plasma	–	1200.0 (1350.0)	1000.0 (625.0)	3500.0 (2500.0)	−3.74	<0.001*
Platelet	–	200.0 (250.0)	200.0 (200.0)	1000.0 (800.0)	−2.64	0.008*
Complication						
Renal failure	Yes	16	9 (56.2)	7 (43.8)	–	0.003*
Cerebral hemorrhage	Yes	3	0 (0.0)	3 (100.0)	–	0.006*
GI complications	Yes	6	0 (0.0)	6 (100.0)	–	<0.001*
MOF	Yes	6	0 (0.0)	6 (100.0)	–	<0.001*
Sepsis	Yes	7	6 (85.7)	1 (14.3)	–	1.000
Pulmonary infection	Yes	6	5 (83.3)	1 (16.7)	–	1.000
Limb ischemia	Yes	4	0 (0.0)	4 (100.0)	–	0.001*
Peak bilirubin (μmol/l)	–	36.0 (50.0)	35.0 (27.0)	112.0 (89.0)	−3.44	0.001*

*$p < 0.01$

BPTV blood product transfusion volume, *GI* gastrointestinal, *MOF* multiple organ failure

[a] *M(Q)/n(%)*

Table 1 Complications during ECMO

Complication	Total (%)
Renal failure	16 (48.4)
Cerebral hemorrhage	3 (9)
GI complications	6 (18.1)
MOF	6 (18.1)
Sepsis	7 (21.2)
Pulmonary infection	6 (18.1)
Limb ischemia	4 (12.1)

GI gastrointestinal, *MOF* multiple organ failure

ECMO is an effective auxiliary tool in the treatment of AFM. Acute renal failure is the most common complication during ECMO. Improving patient tissue perfusion, reducing blood transfusions and preventing acute kidney failure may improve patient outcomes.

Eosinophilic Myocarditis Characteristics, Treatment, and Outcomes

M. Brambatti et al. J Am Coll Cardiol 2017;70:2363–75

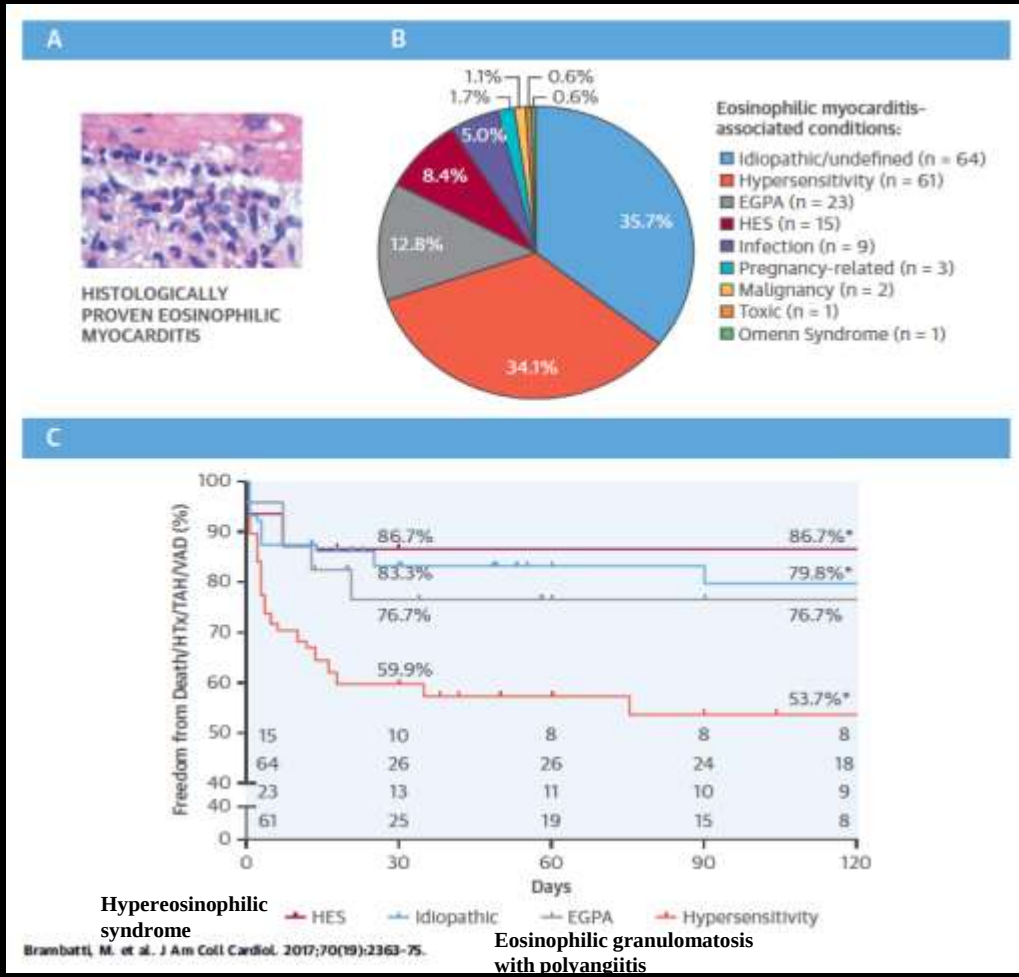


TABLE 4 In-Hospital Events and Treatment and Post-Discharge Events According to EM Different Etiologies

	Idiopathic/Undefined		Hypersensitivity		EGPA		HES		Others	
	No. of Patients With Available Data	Value	No. of Patients With Available Data	Value	No. of Patients With Available Data	Value	No. of Patients With Available Data	Value	No. of Patients With Available Data	Value
n	64		61		23		15		16	
In-hospital events and treatments										
Deaths*	64	8 (12.5)†	61	22 (36.1)†	23	5 (21.7)	15	2 (13.3)	16	3 (18.7)
TAH	64	1 (1.6)	61	0 (0.0)	23	0 (0.0)	15	0 (0.0)	16	0 (0.0)
Long-term VAD	64	2 (3.1)	61	2 (3.3)	23	0 (0.0)	15	0 (0.0)	16	0 (0.0)
Temporary MCS	64	10 (15.6)	61	12 (19.7)	23	5 (1.7)	15	1 (6.7)	16	2 (2.5)
Use of inotrope	64	22 (34.4)	61	25 (41.0)	23	5 (21.7)	15	2 (13.3)	16	2 (12.5)
Use of steroids	64	53 (82.8)	61	42 (68.8)	23	20 (87.0)	15	13 (86.7)	16	11 (68.8)
Use of other immunosuppressive agent	54	6 (11.1)	53	8 (15.1)	21	7 (33.3)	12	5 (41.7)	14	1 (7.1)
Post-discharge events										
Cardiac deaths	49	0 (0.0)	39	1 (2.6)	13	0 (0.0)	10	1 (10.0)	11	0 (0.0)
Noncardiac deaths	49	0 (0.0)	39	0 (0.0)	13	0 (0.0)	10	0 (0.0)	11	0 (0.0)
HTx	49	1 (2.0)	39	0 (0.0)	13	0 (0.0)	10	0 (0.0)	11	0 (0.0)
Long-term VAD	49	0 (0.0)	39	0 (0.0)	13	0 (0.0)	10	0 (0.0)	11	0 (0.0)

Hypereosinophilic syndrome

Eosinophilic granulomatosis with polyangiitis

EM has a poor prognosis during the acute phase. Associated conditions are identified in approximately 65% of cases. Specific trials and multicenter registries are needed to provide evidence-based treatments to improve in-hospital outcome

Myocardial expression of Toll-like receptor 4 (TLR4) predict the response to immunosuppression in patients with virus-negative chronic inflammatory cardiomyopathy.

C. Chimenti et al, European Journal of Heart Failure 2017, doi:10.1002/ejhf.796

TLR4 is a transmembrane protein. Its activation leads to an inflammatory cytokine production responsible for activating the innate immune system

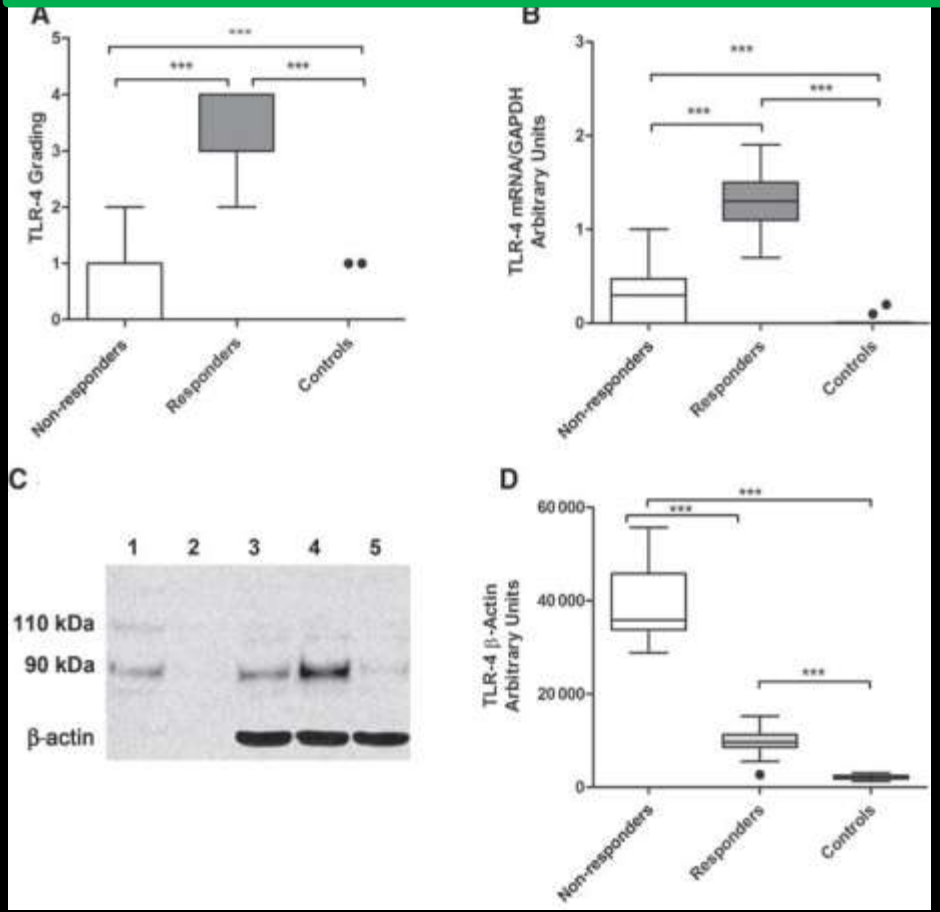


Table 2 TLR4 grading as a predictor of treatment response according to sex

TLR4 grading	All		Females		Males	
	Responders $n=193$	Non-responders $n=44$	Responders $n=110$	Non-responders $n=14$	Responders $n=83$	Non-responders $n=30$
4	126	–	76	–	50	–
3	55	–	27	–	28	–
2	12	4	7	–	5	4
1	–	25	–	8	–	17
0	–	15	–	6	–	9
2+ cut-off						
Sensitivity	100.0%		100.0%		100.0%	
Specificity	90.9%		100.0%		86.7%	
PPV	98.0%		100.0%		95.4%	
NPV	100.0%		100.0%		100.0%	
3+ cut-off						
Sensitivity	93.8%		93.6%		94.0%	
Specificity	100.0%		100.0%		100.0%	
PPV	100.0%		100.0%		100.0%	
NPV	78.6%		66.7%		85.7%	

TLR4 is highly expressed in human myocarditis responding to immunosuppression. It can be considered as a new sensitive marker in patient selection predicting a good response to immunosuppressive therapy.