State of the art in myocarditis treatment

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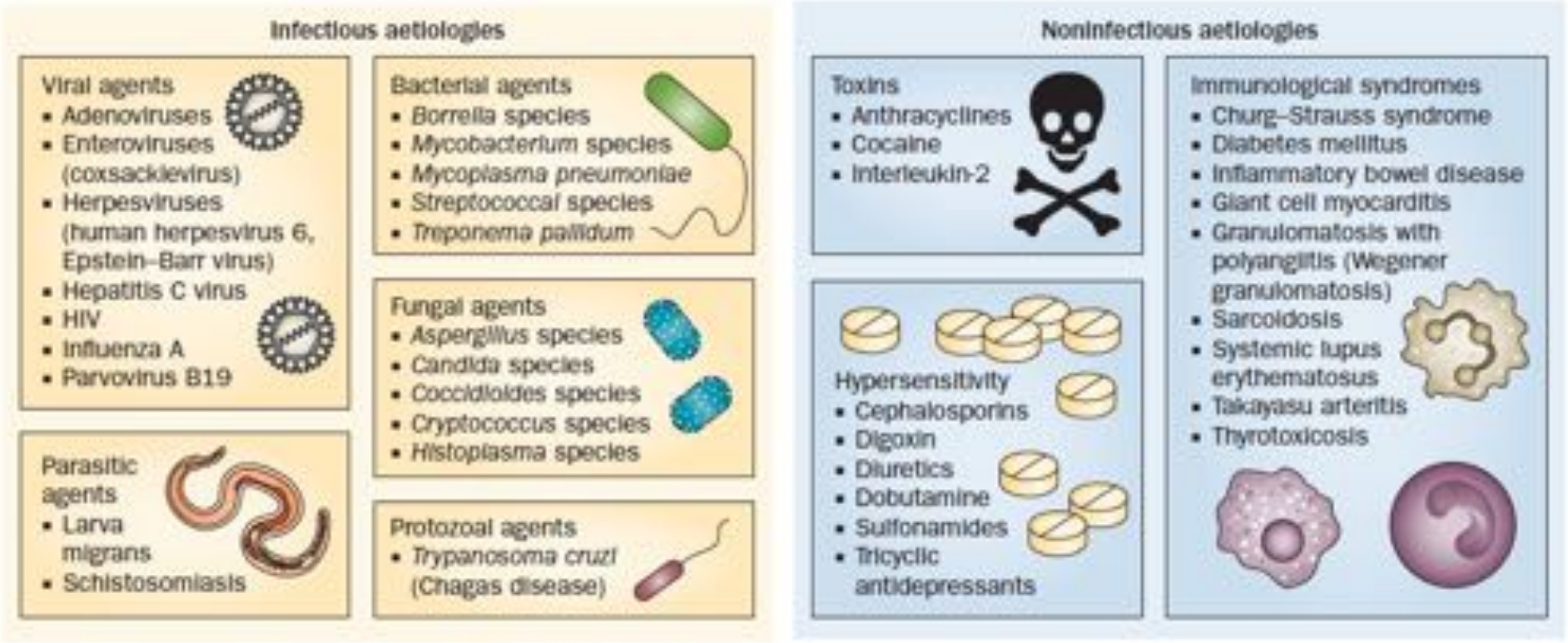
nature REVIEWS CARDIOLOGY

Viral myocarditis—diagnosis, treatment options, and current controversies

Ari Pollack, Amy R. Kontorovich, Valentin Fuster and G. William Dec



Nat. Rev. Cardiol. **12**, 670–680 (2015)

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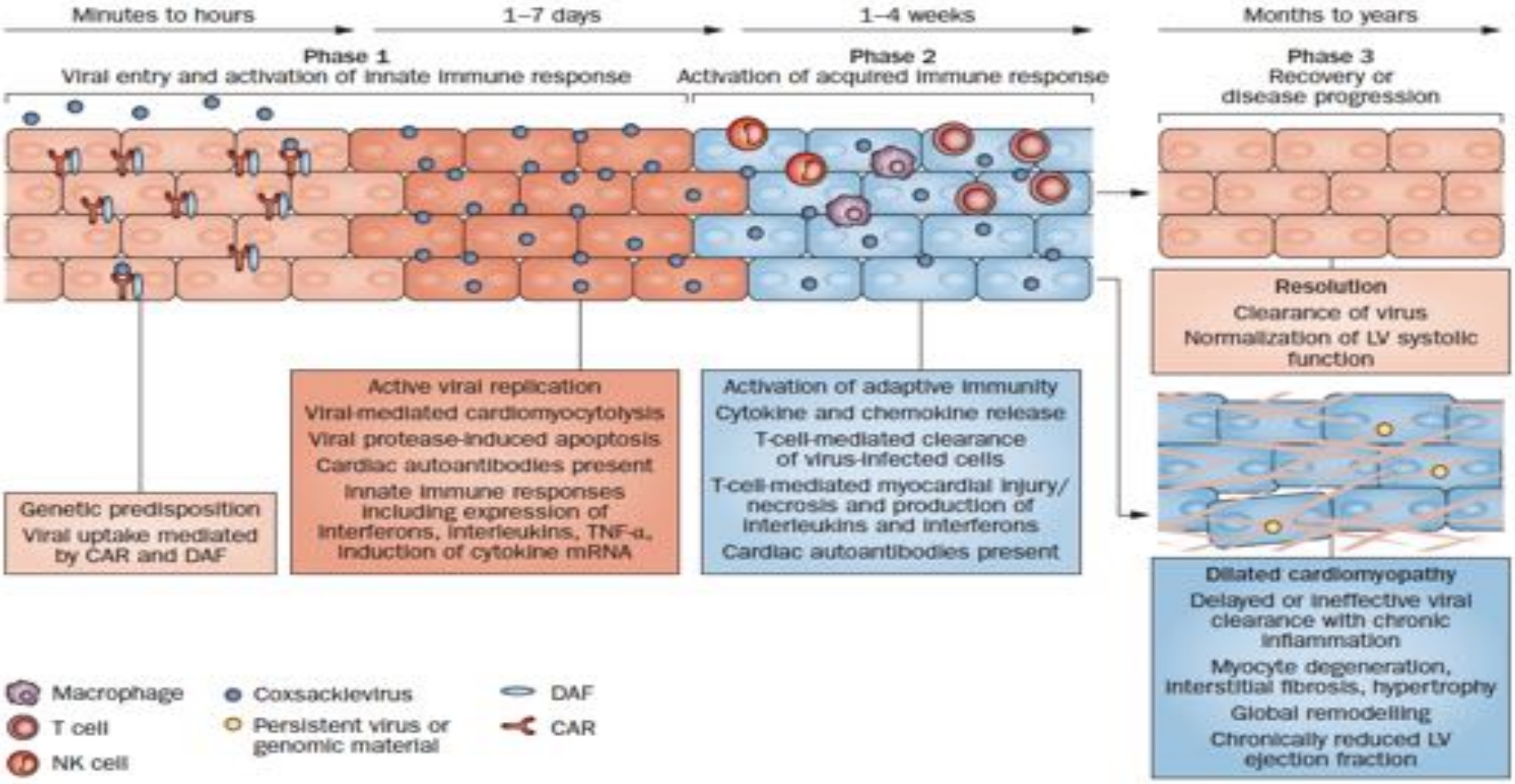

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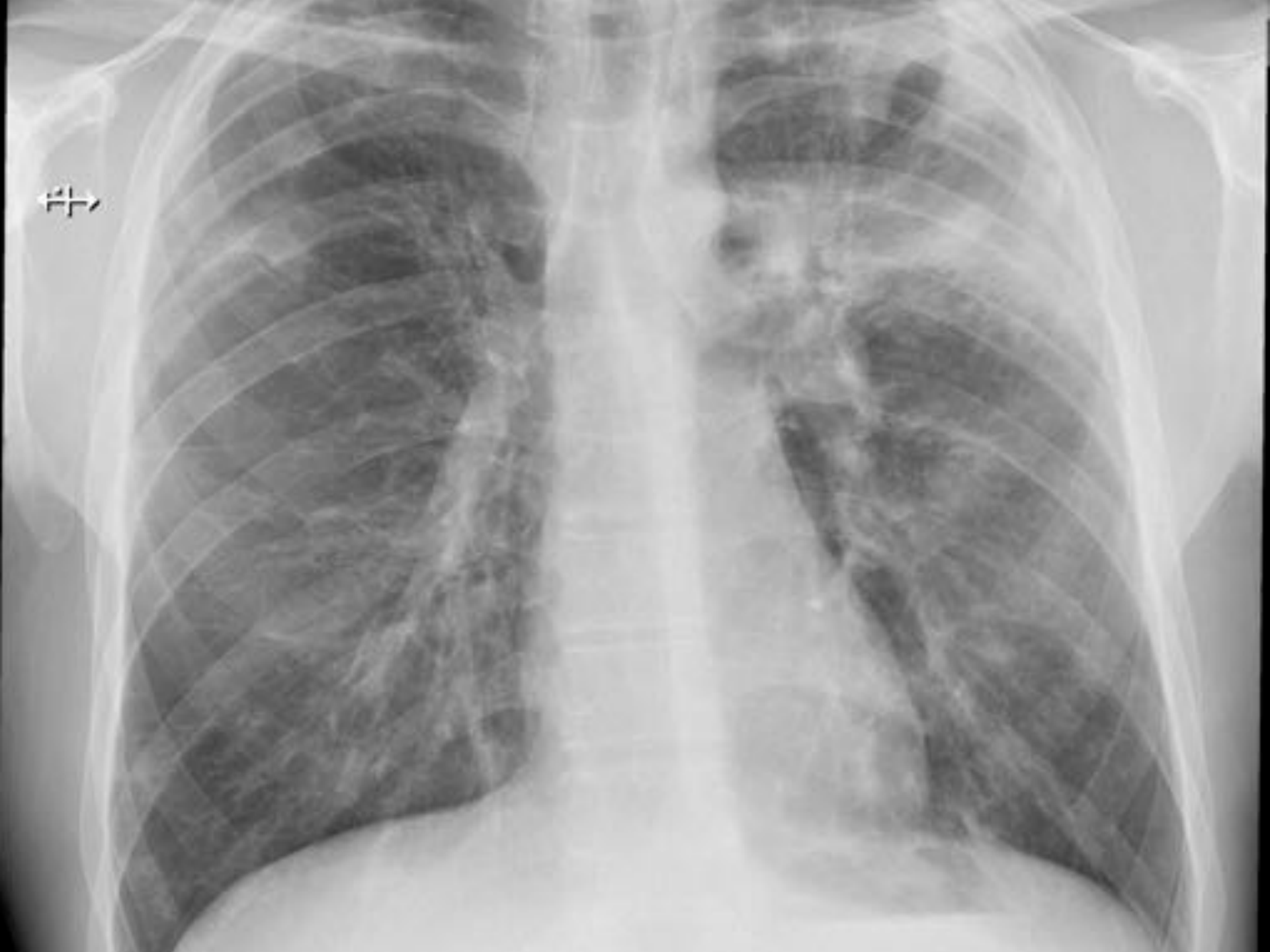
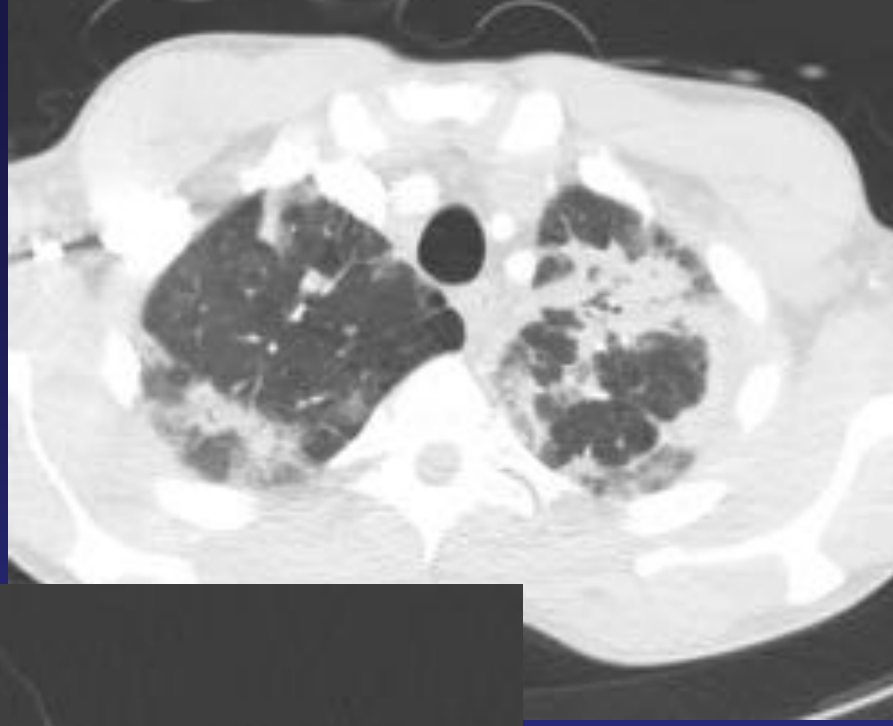
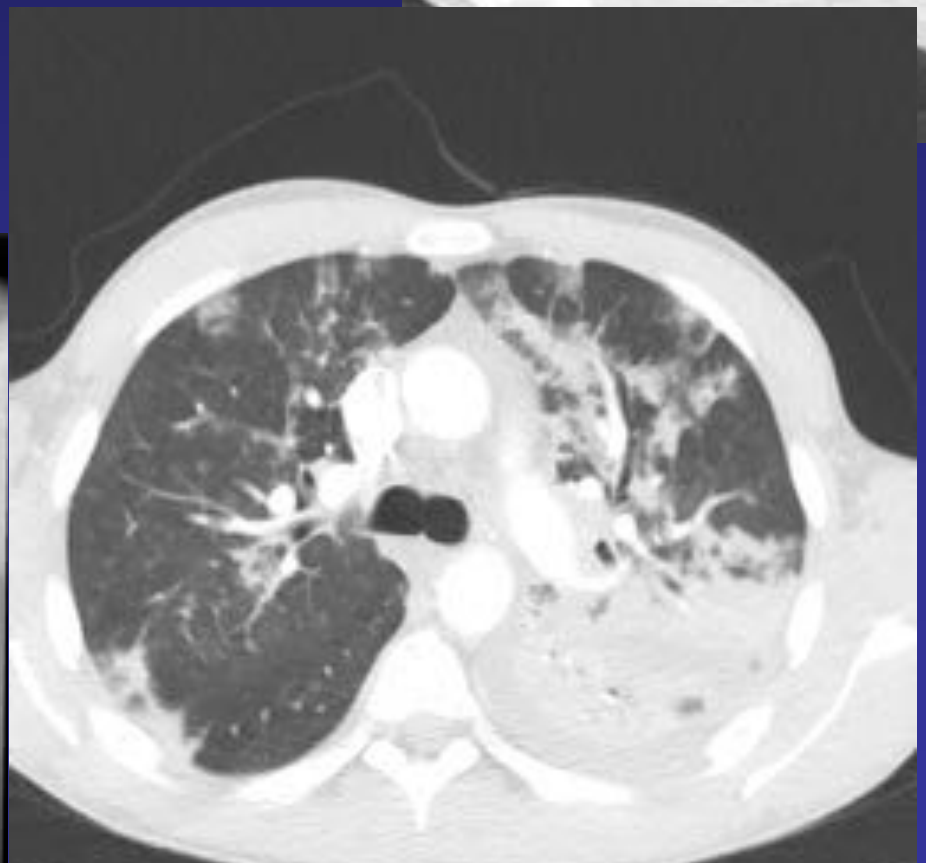
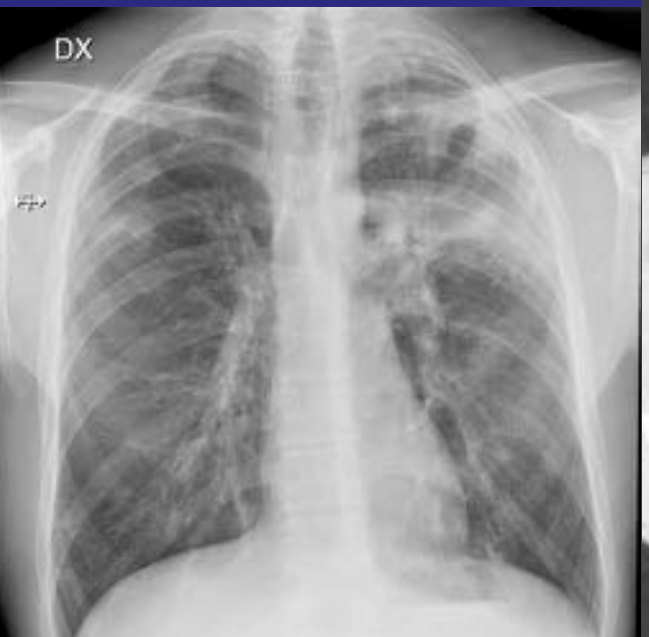
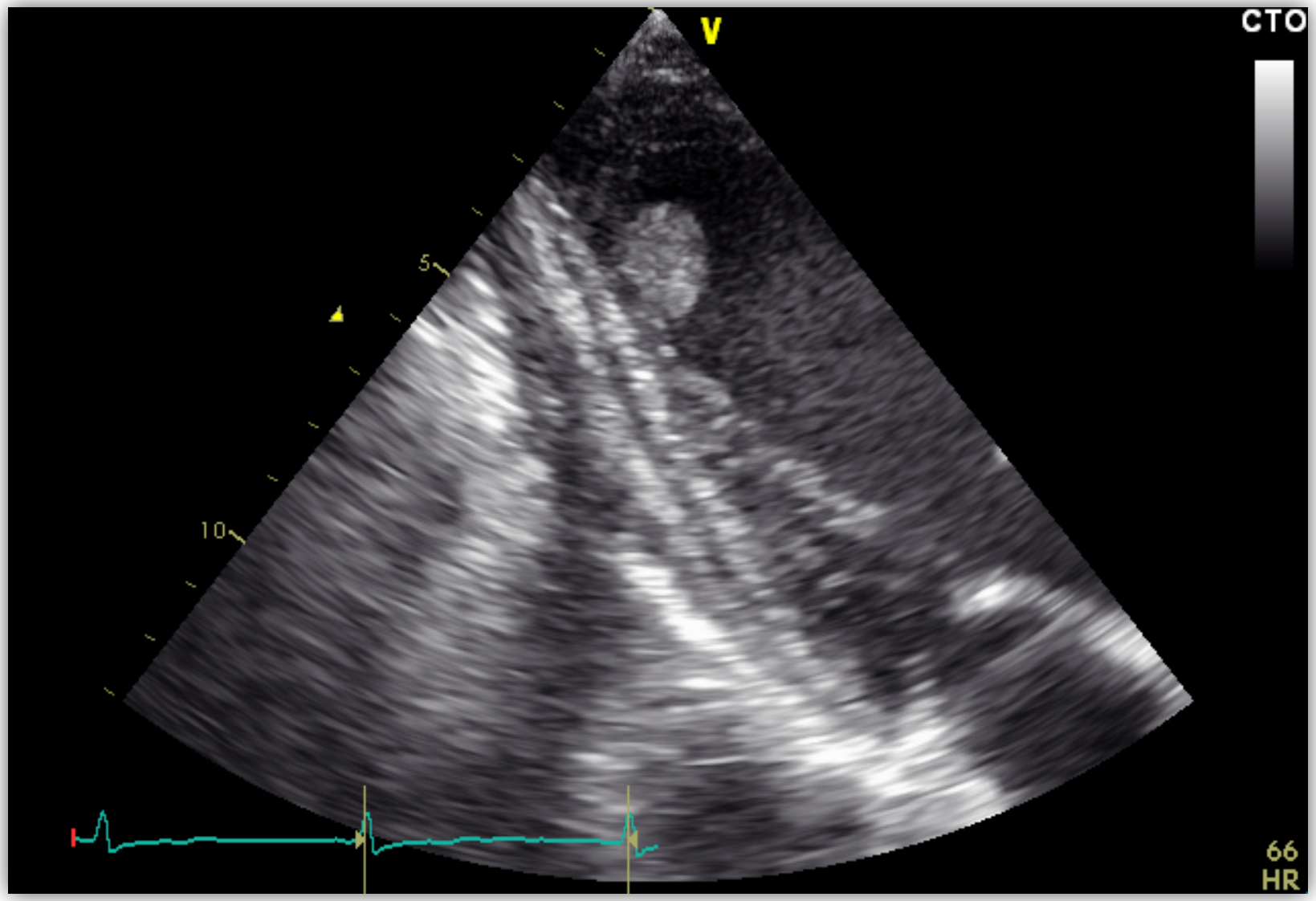


GB 21550 /mcL; N 6900;
L 2370; E 10340 /mcL
PCR 184 mg/L
BNP 1392 pg/ml





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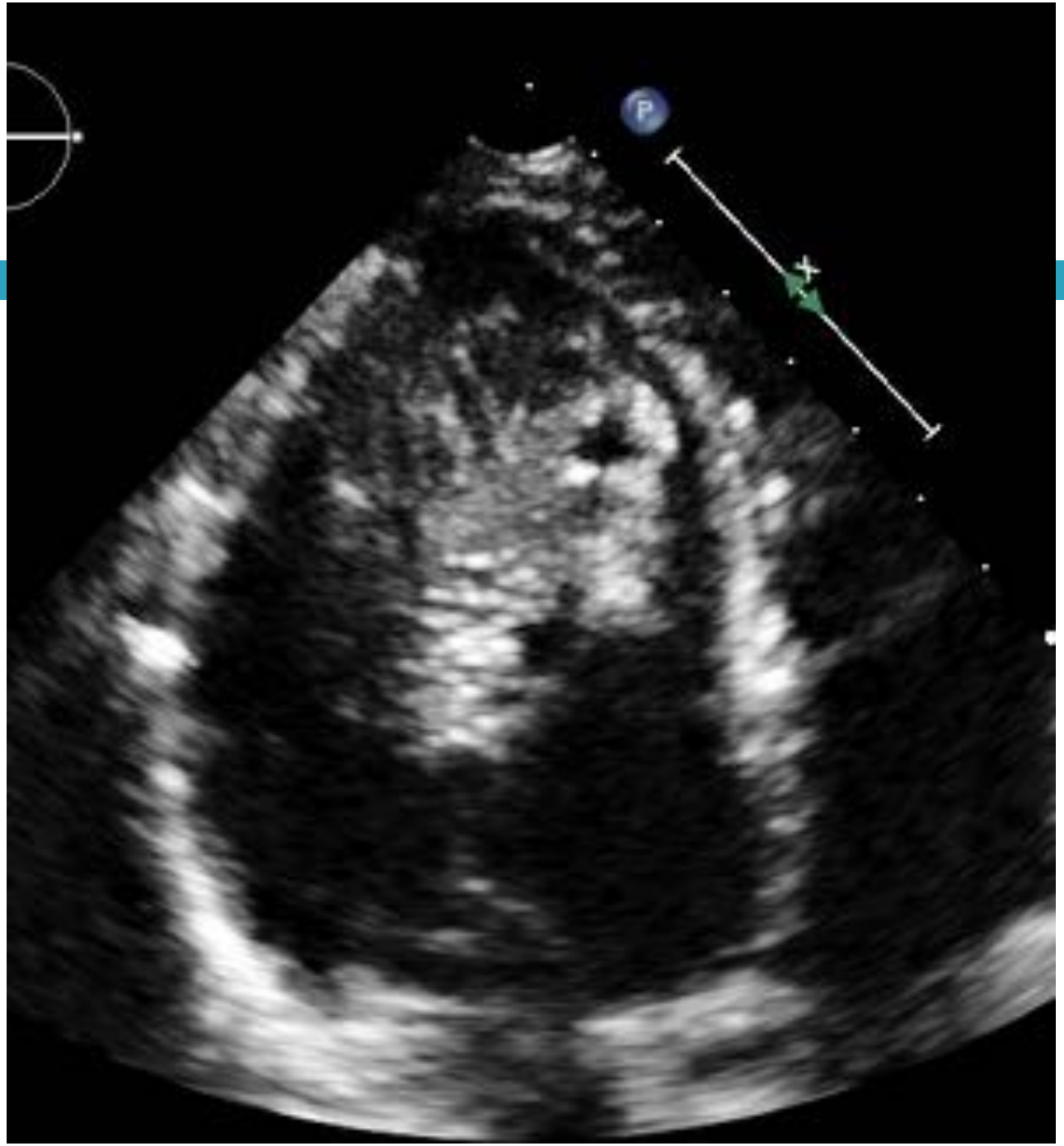
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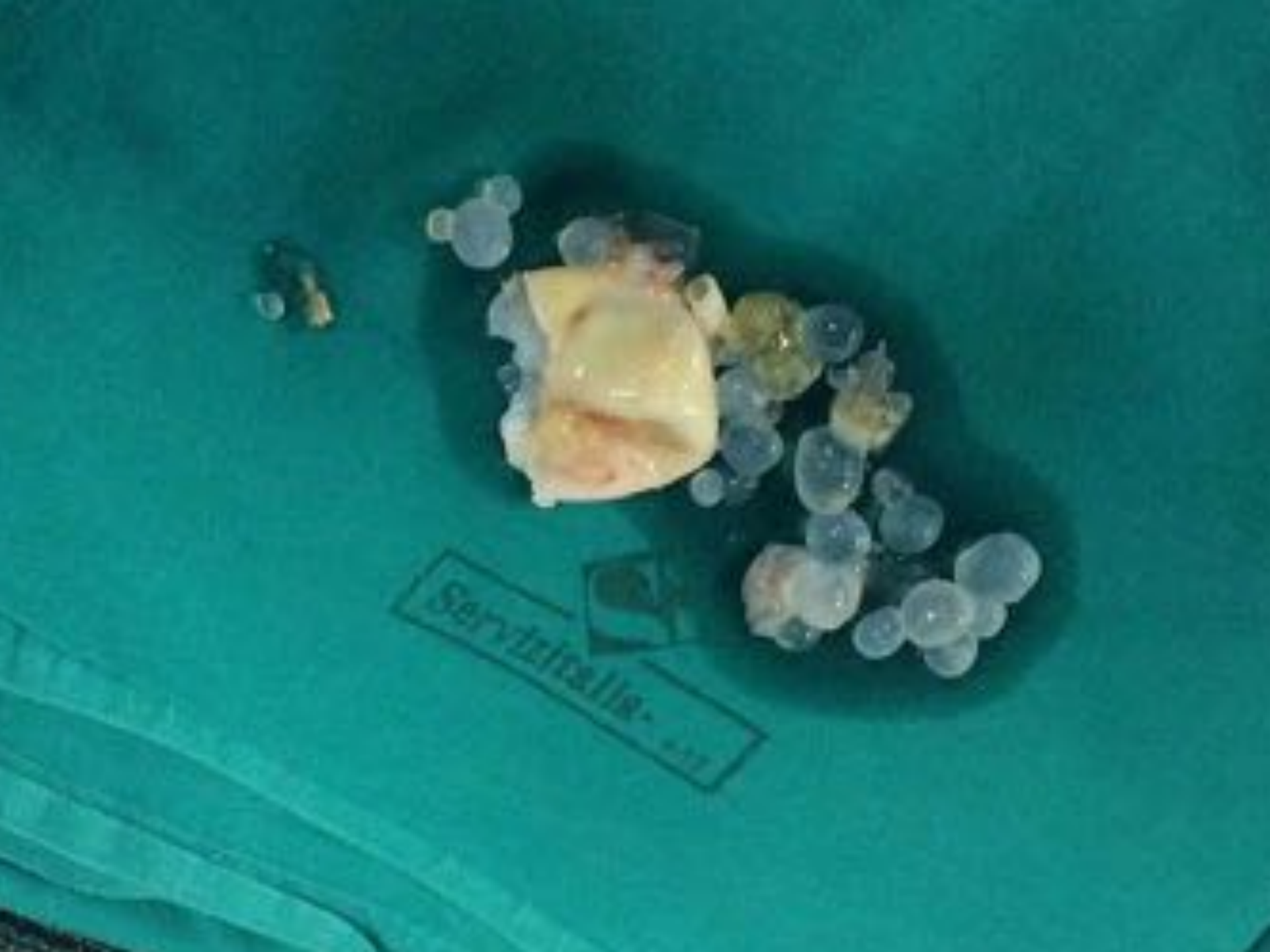
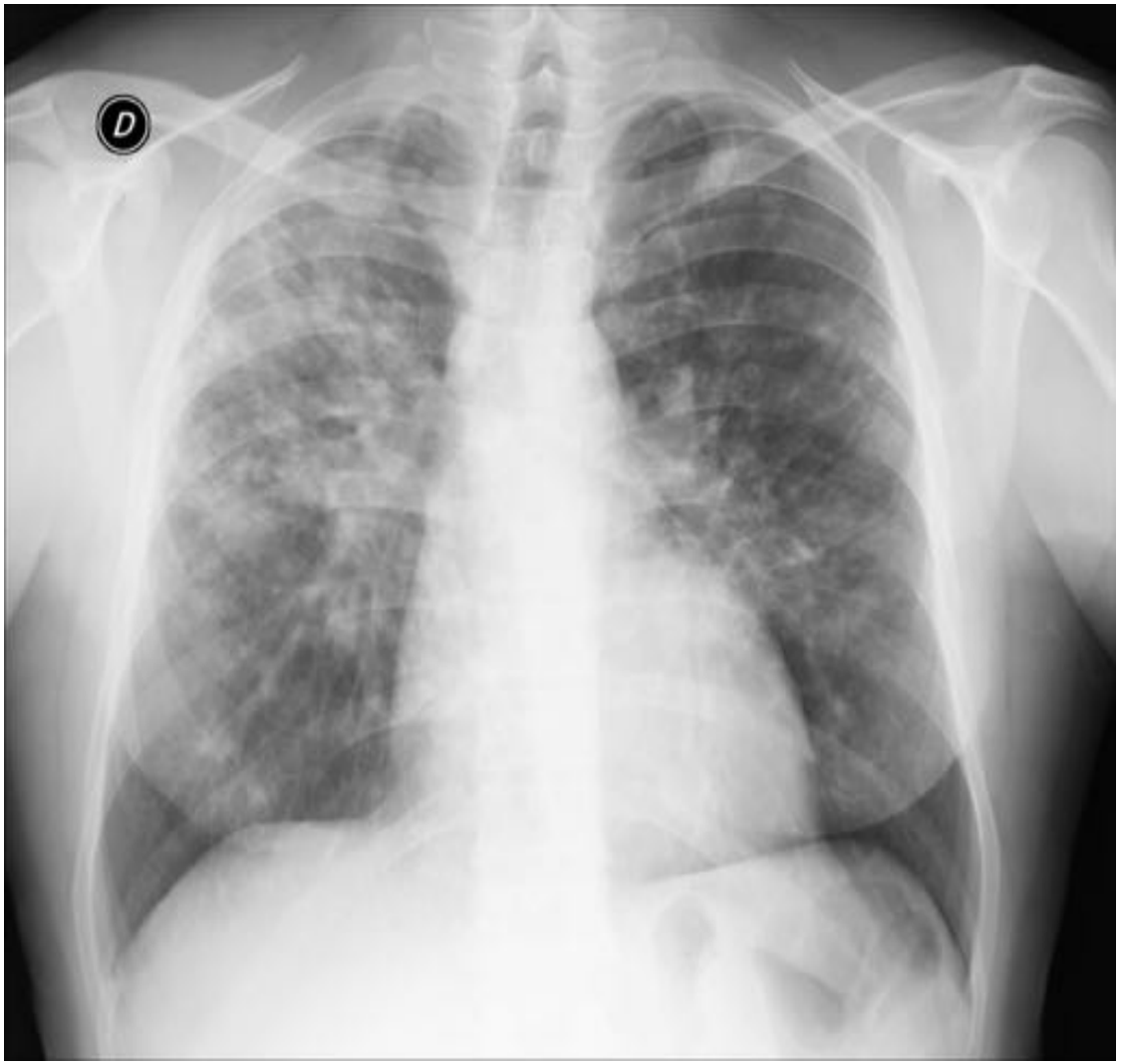
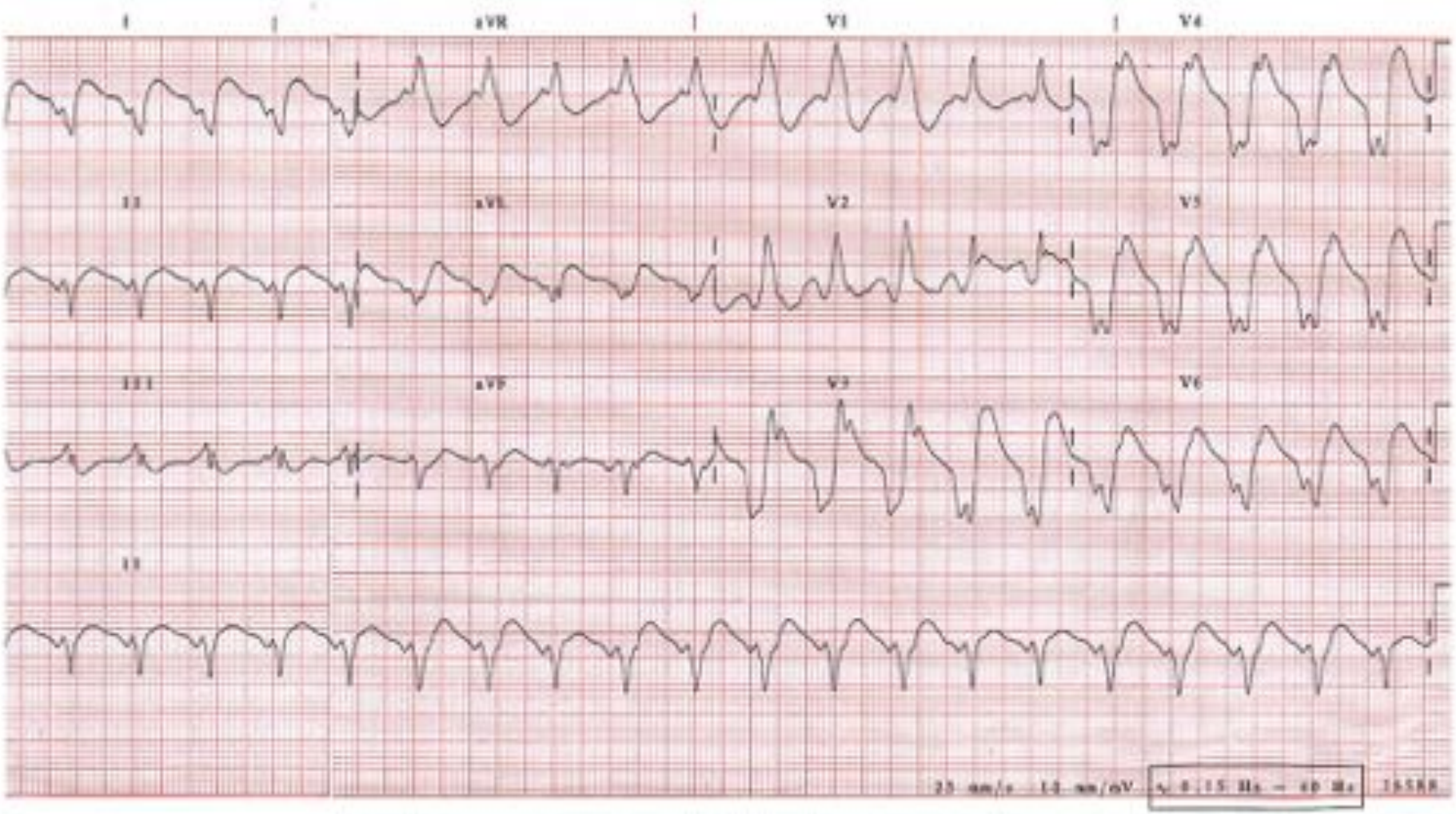


43 aa; M; palpitazioni; sincope; FEVsin 43%; asinergie++ SIV e postero-basali



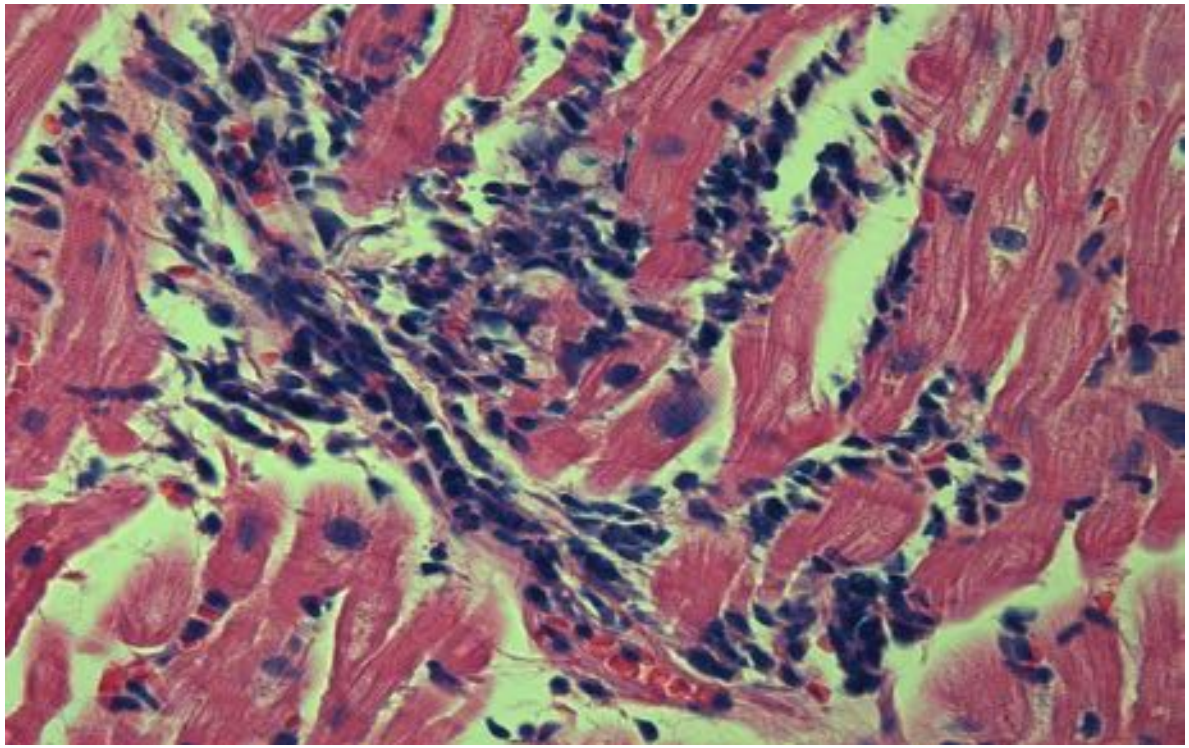
43 aa; M; palpitazioni; sincope; FEVsin 43%



Circulation. 1996 Mar 1;93(5):841-2.

Report of the 1995 World Health Organization/International Society and Federation of Cardiology Task Force on the Definition and Classification of cardiomyopathies.

Richardson P, McKenna W, Bristow M, Maisch B, Mautner B, O'Connell J, Olsen E, Thiene G, Goodwin J, Gyarfas I, Martin I, Nordet P.

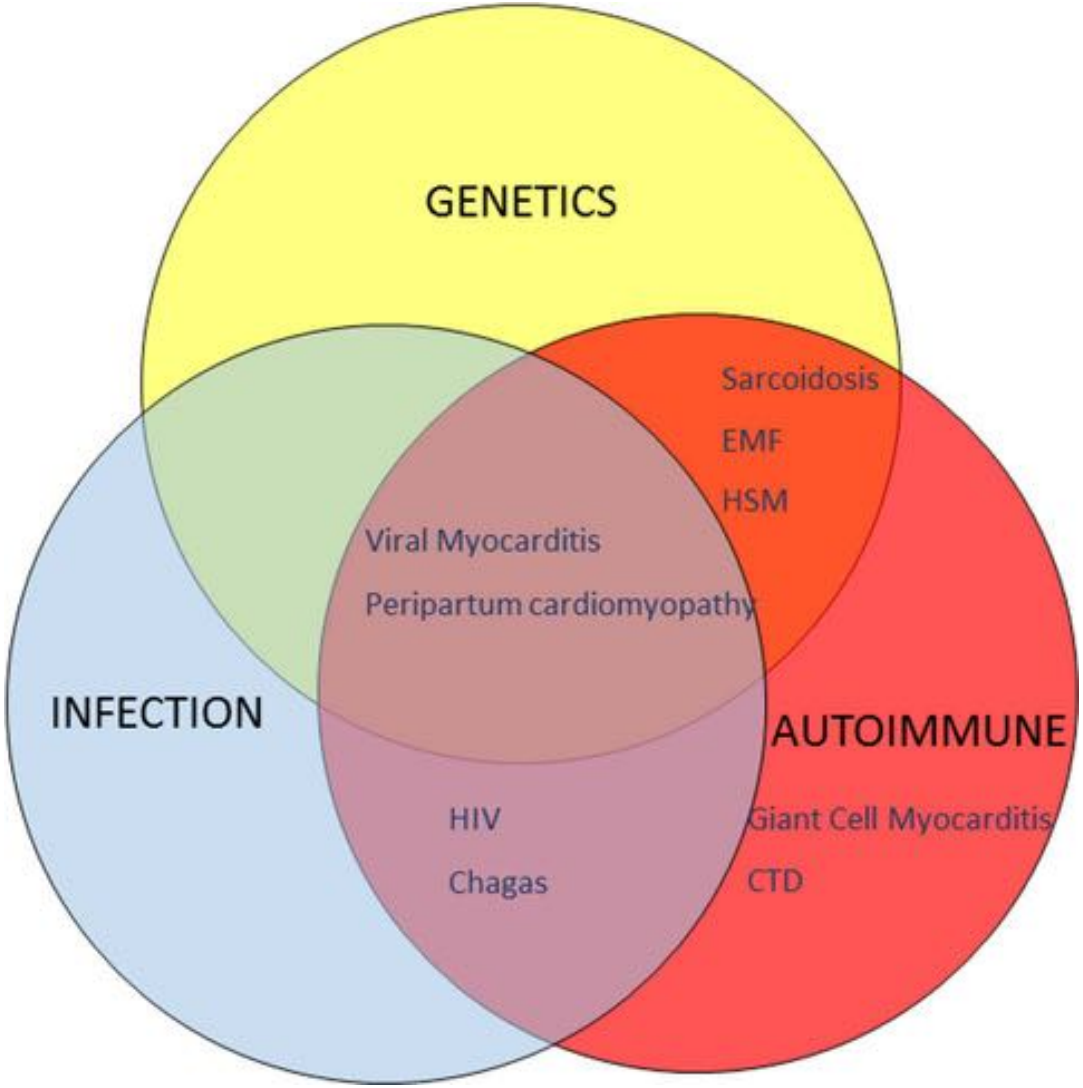


La MIOCARDITE è una malattia infiammatoria del miocardio, con coinvolgimento di miociti, interstizio ed endotelio vascolare. La diagnosi si basa su criteri istopatologici ed immunoistochimici

Inflammatory Cardiomyopathic Syndromes

Circulation
JOURNAL OF THE AMERICAN HEART ASSOCIATION

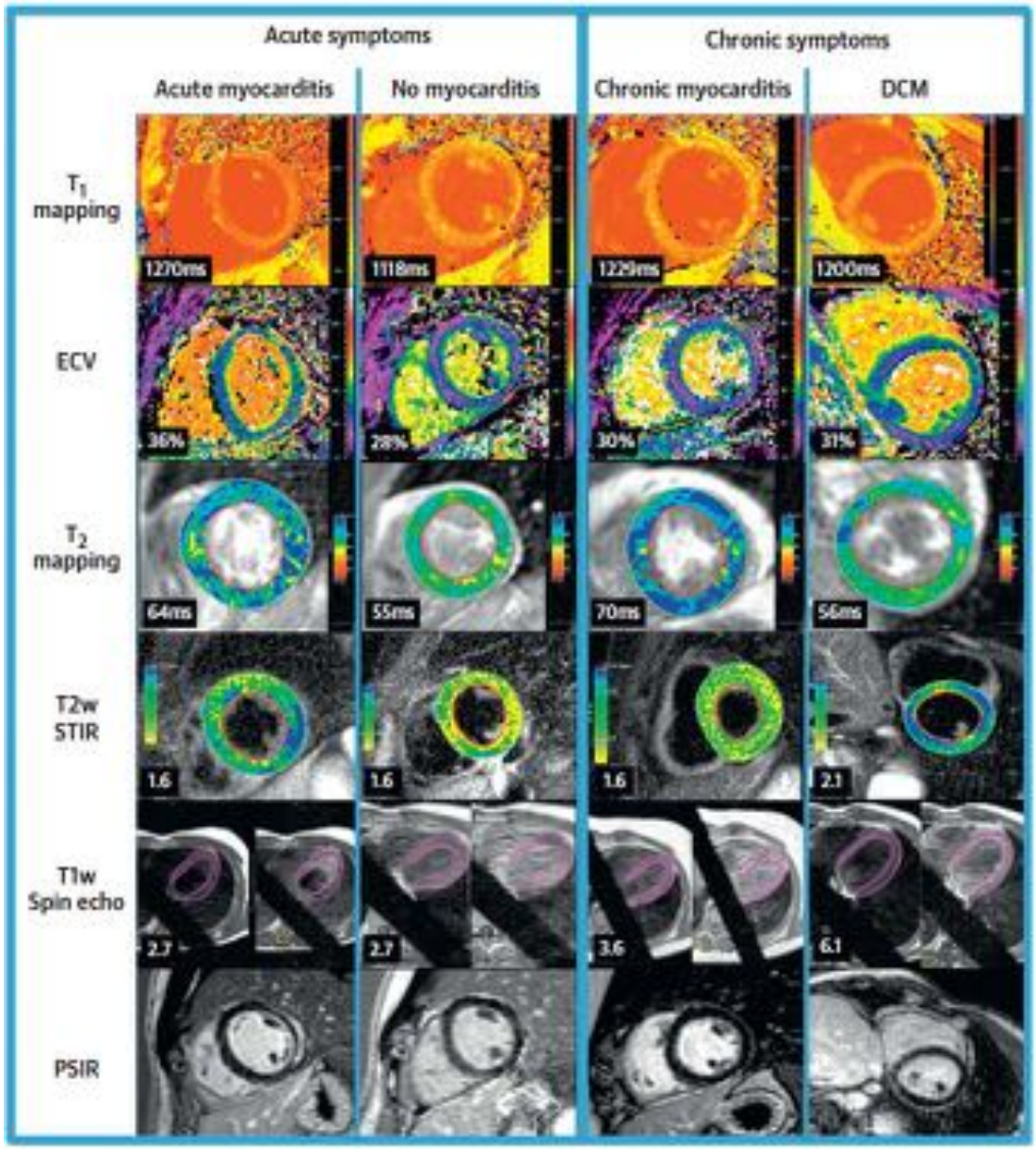
Diagram showing current evidence for overlapping theories of common causes of inflammatory cardiomyopathy



Trachtenberg, Circ Res. 2017;121:803-818

Comprehensive Cardiac Magnetic Resonance Imaging in Patients With Suspected Myocarditis

The MyoRacer-Trial



Lurz et al. J Am Coll Cardiol 2016;67:1800–11

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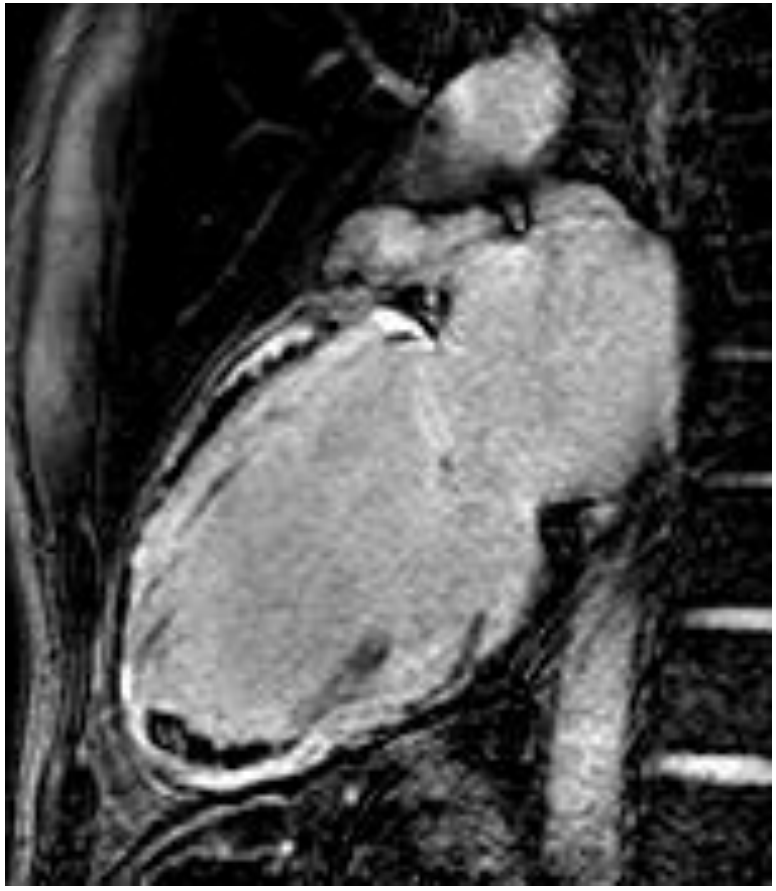
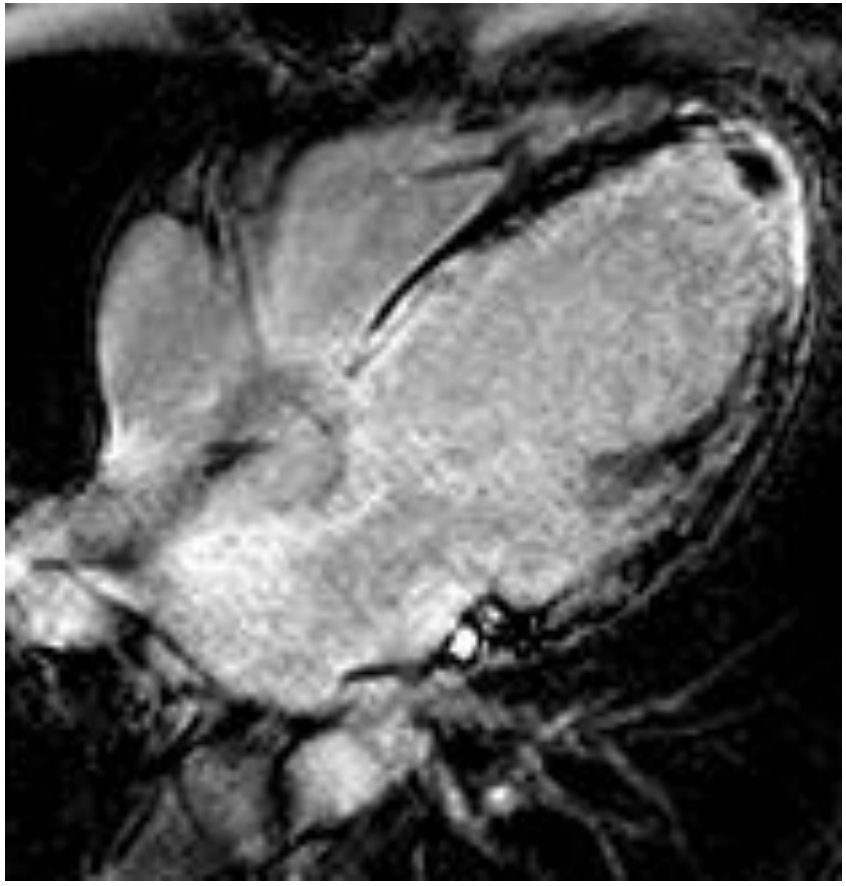
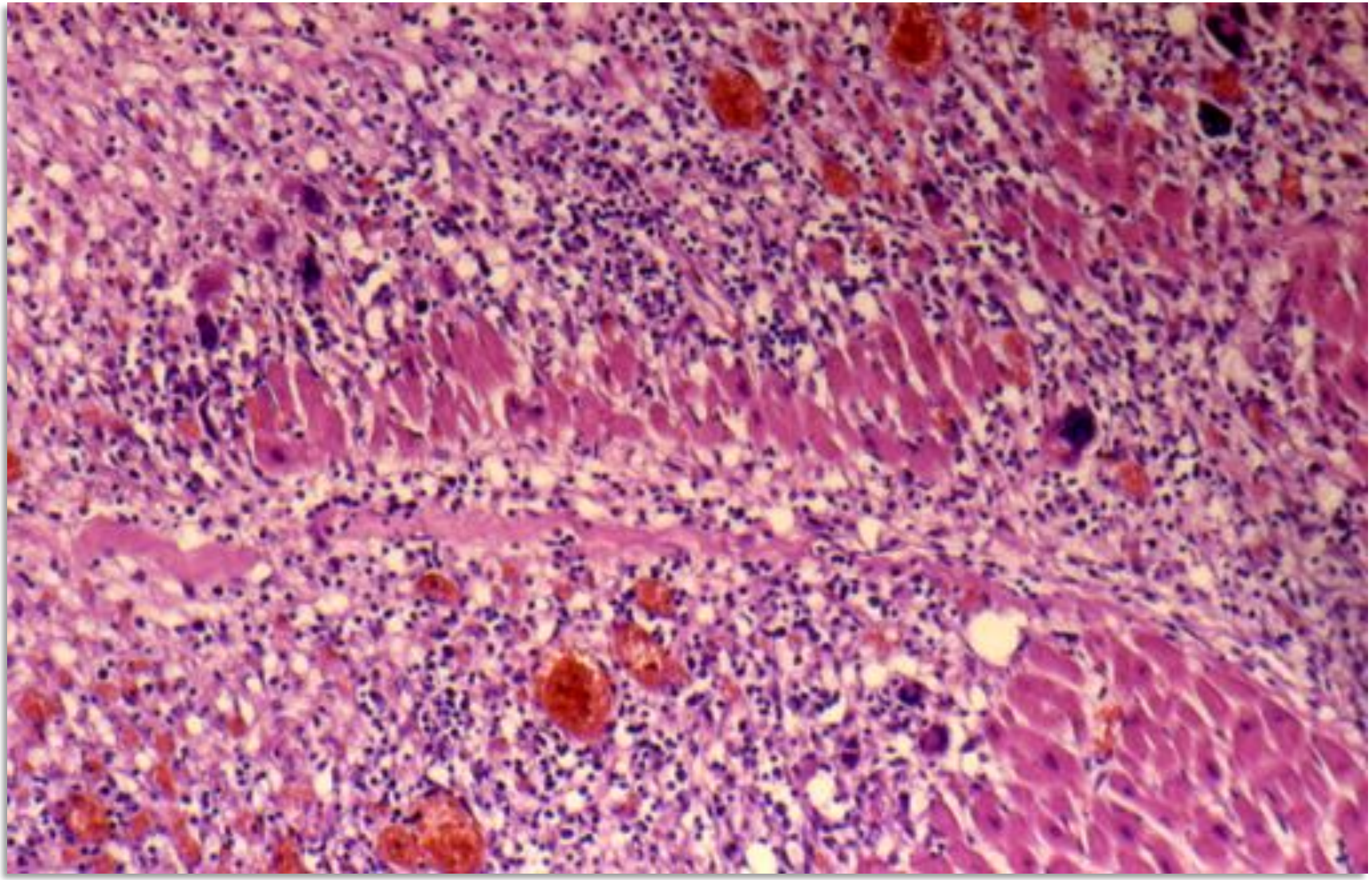
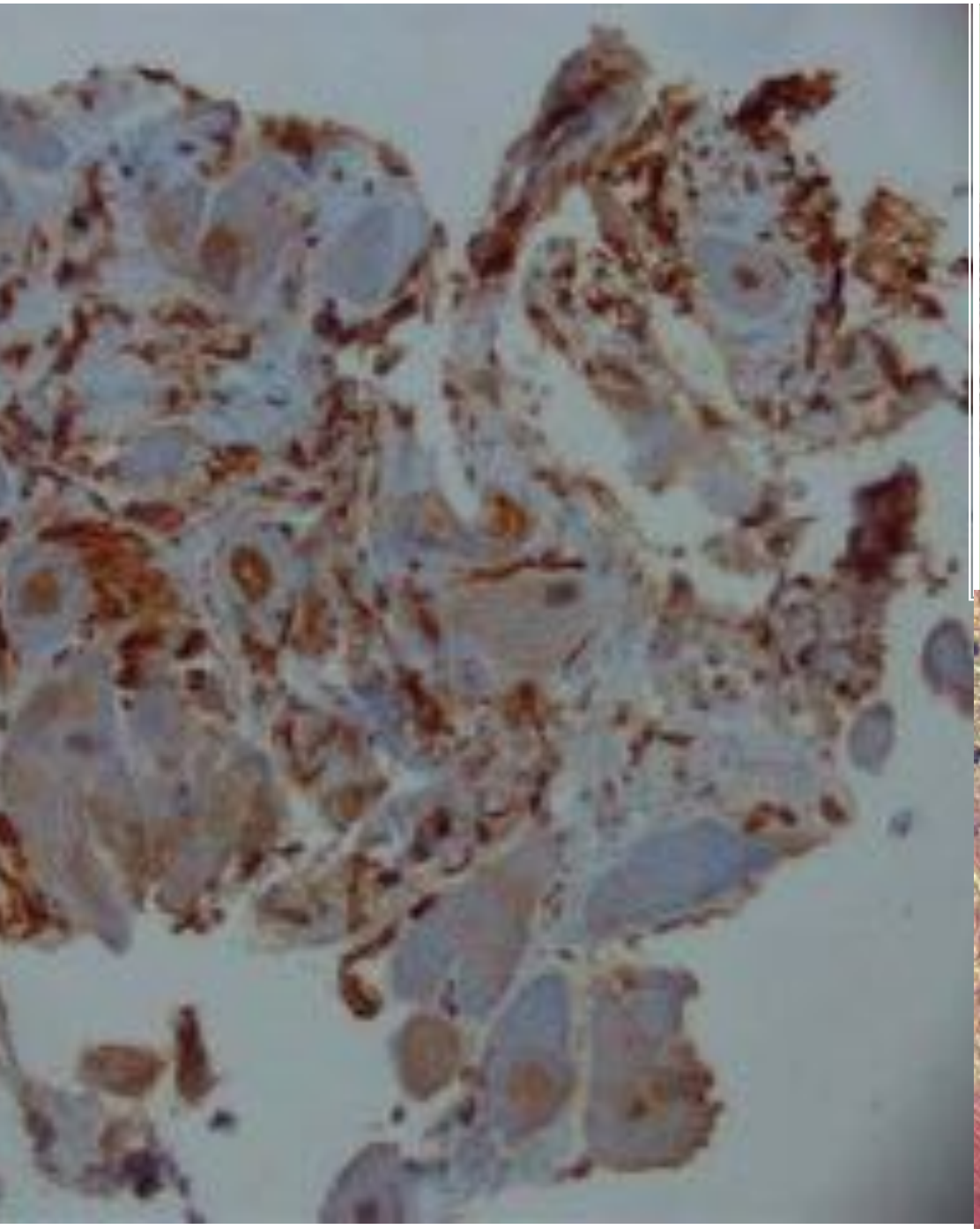
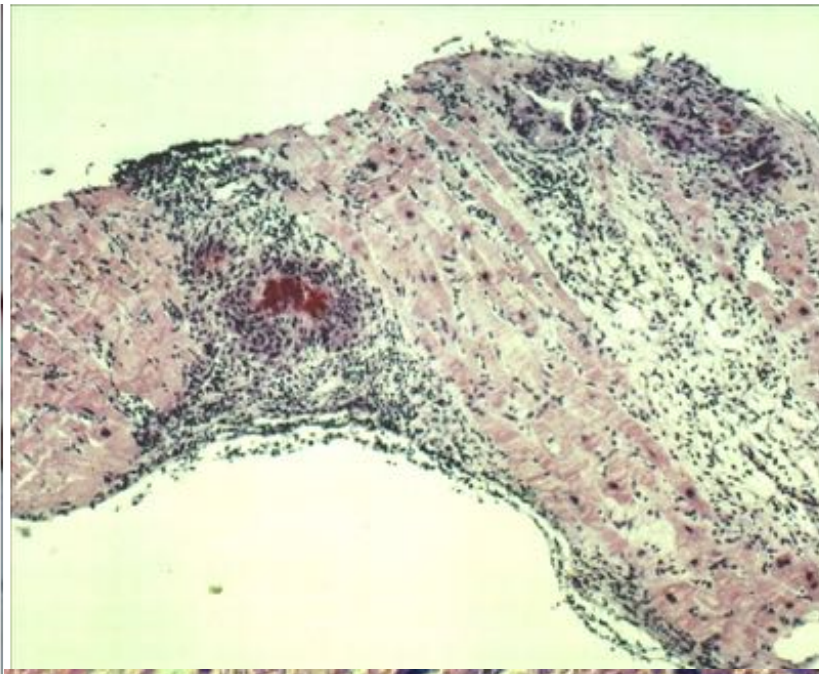
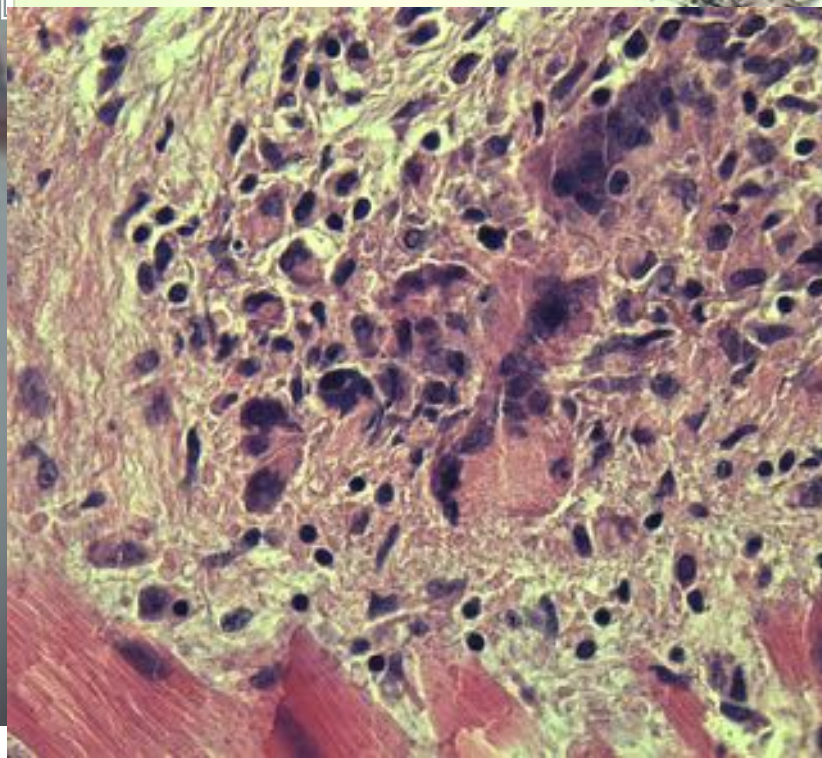






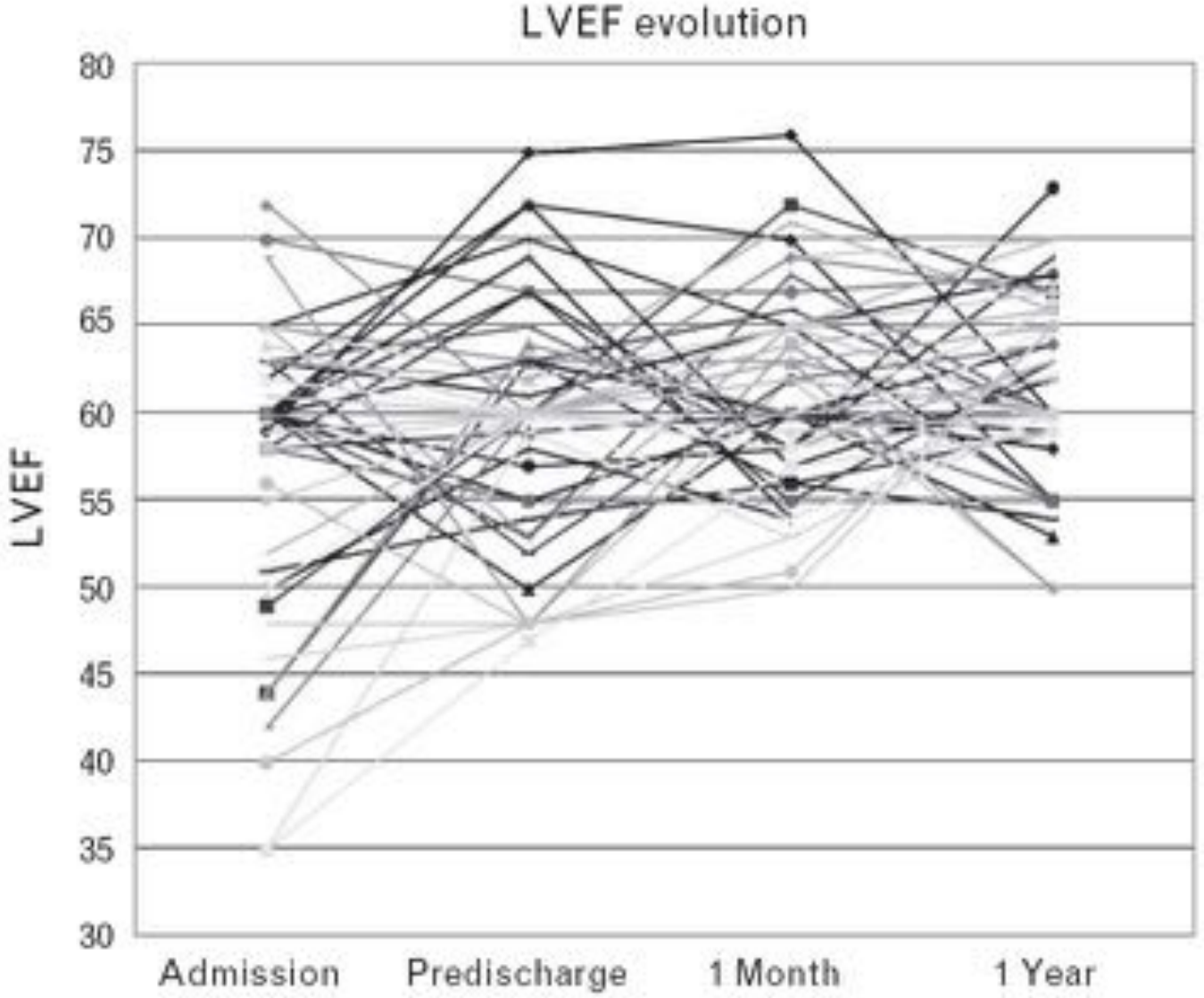






Clinical presentation and long-term follow-up of perimyocarditis

Buiatti, Sinagra et al
J Cardiovasc Med 2012, 13:000–000



Long-term follow-up on cardiac function following fulminant myocarditis requiring percutaneous extracorporeal cardiopulmonary support

Heart and Vessels

20 patients
Mean age 45 ±19
NYHA IV
13 lymphocitic
7 non lymphocitic

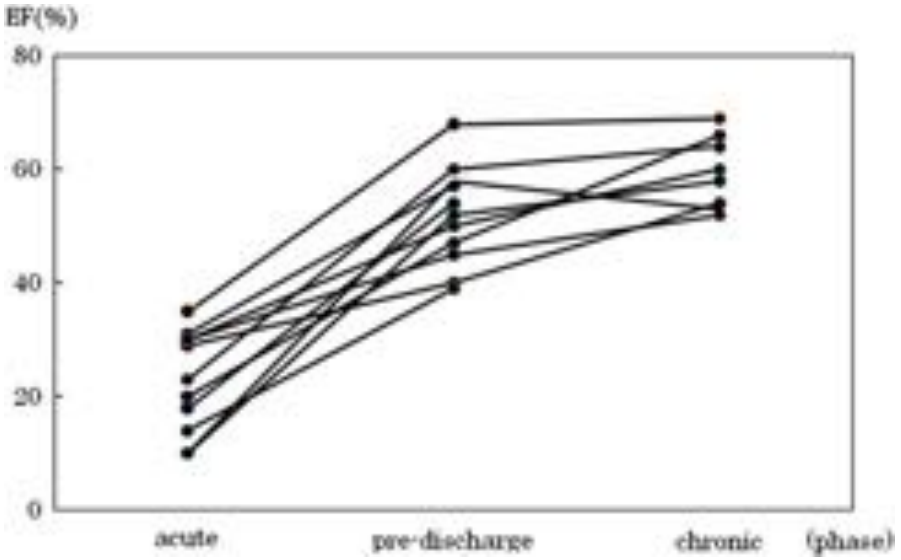


Table 2 Time course of diastolic dimension and systolic dimension by echocardiography

	Acute phase	Predischarge	Chronic phase
Dd (mm)	46.8 ± 7.4	51.3 ± 2.9	50.4 ± 1.8
Ds (mm)	41.4 ± 7.7	36.8 ± 4.0	35.2 ± 3.3
EF (%)	22.7 ± 9.8	53.1 ± 7.2	57.2 ± 9.6*
IVS (mm)	11.6 ± 2.2	8.7 ± 2.3	7.6 ± 1.6*
LVPW (mm)	12.5 ± 3.2	8.8 ± 1.8	8.1 ± 1.4*
LAD (mm)	29.3 ± 6.4	33.4 ± 4.1	36.0 ± 4.1*

The cardiac function of patients with fulminant myocarditis recovers rapidly during their stay in hospital. The cardiac function of predischarge patients remains unchanged in the chronic phase. The long-term survival of fulminant myocarditis appears favorable in the chronic phase.

Ishida et al. Heart Vessels (2013) 28:86–90

The fate of acute myocarditis between spontaneous improvement and evolution to dilated cardiomyopathy: a review

Heart

Evolution to dilated cardiomyopathy in patients with acute myocarditis

Reference	*Number of patients*	*Diagnosis*	*Evolution to DCM (%)*	*Mean follow up or range*
Bengtsson 1966[30]	90	Clinical	15	60 months
Helin 1968[31]	12	Clinical	0	7 months
Sainani 1968[32]	19	Clinical	0	3 months
Smith 1970[33]	42	Clinical	7	84 months
Gerzen 1972[34]	18	Clinical	28	12 months
Levi 1977[35]	10	Clinical	0	42–68 months
Kitaura 1979[36]	11	Histology	27	18–156 months
Edwards 1982[37]	5	Histology	20	6–12 months
Kayakawa 1983[38]	20	Clinical	30	49 months
Fenoglio 1983[39]	18	Histology	17	12 months
Kayakawa 1984[39]	10	Clinical	30	55 months
Daly 1984[40]	9 (IS 9)	Histology	22	6 months
Dec 1985[13]	18	Histology	11	6–12 months
Das 1985[41]	18	Clinical	11	54 months
Billingham 1986[42]	20 (IS 15)	Histology	40	5 months
Giesecke1987[28]	45	Clinical	11	3 months
Quigley 1987[12]	23	Histology	52	43 months
Weiss 1987[43]	13 (IS 1)	Histology	46	36 months
Anderson 1987[44]	10	Histology	20	6–12 months
Sekiguchi1988[17]	20	Histology	10	49 months
Salvi 1990[45]	38 (IS 28)	Histology	29	49 months
Davidoff 1991[46]	36 (IS 20)	Histology	19	30 months
Sekiguchi 1994[47]	90	Histology	14	45 months
Maisch 1994[48]	21	Histology	28	6 months
Mason 1995[27]	47	Histology	17	52 months
Sinagra 1997[18]	56 (IS 36)	Histology	39	48 months
Total	719		Mean 21%	Mean f/u 33 months

D'ambrosio, Sinagra et al. Heart 2001;85:499–504

Circulation

American Heart Association.

Long-Term Evolution and Prognostic Stratification of Biopsy-Proven Active Myocarditis
Marco Anzini, Marco Merlo, Gastone Sabbadini, Giulia Barbati, Gherardo Finocchiaro, Bruno Pinamonti, Alessandro Salvi, Andrea Perkan, Andrea Di Lenarda, Rossana Bussani, Jozef Bartunek and Gianfranco Sinagra

Circulation. 2013;128:2384-2394; originally published online October 1, 2013;
doi: 10.1161/CIRCULATIONAHA.113.003092

Registro Cardiomiopatie di Trieste - Miocarditi

	Pop. totale (82; 100%)	SCC (53; 65%)	Aritmie (20; 24%)	Pseudo-IMA (9; 11%)	p value *
Recente virosi – no. (%)	58(70)	39(74)	13(65)	6(67)	0.742
Puntura d'insetto – no. (%)	12(15)	4(8)	8(40)	0(0)	0.001
Durata sintomi – giorni	8[1-30]	15[5-54]	3.5[1-12]	1[1-14]	0.013
NYHA III-IV – no. (%)	39(48)	36(68)	3(15)	0(0)	<0.001
Press. Art. Sist. – mmHg	123±20	118±19	134±20	126±23	0.009
Frequenza cardiaca – bpm	88±28	98±26	64±19	84±22	<0.001
BBS – no. (%)	12(15)	10(19)	2(10)	0(0)	0.266
DASI – mm/m^2	22±5	24±5	18±3	19±3	<0.001
DTD VS I – mm/m^2	34[29-38]	36[33-40]	26[25-32]	27[26-30]	<0.001
FE VS – %	32[24-52]	28[21-32]	57[49-64]	56[53-64]	<0.001
FE VS < 50 % – no. (%)	59(72)	53(100)	5(25)	1(11)	<0.001

* p tra i gruppi (analisi della varianza)

Circulation
JOURNAL OF THE AMERICAN HEART ASSOCIATION

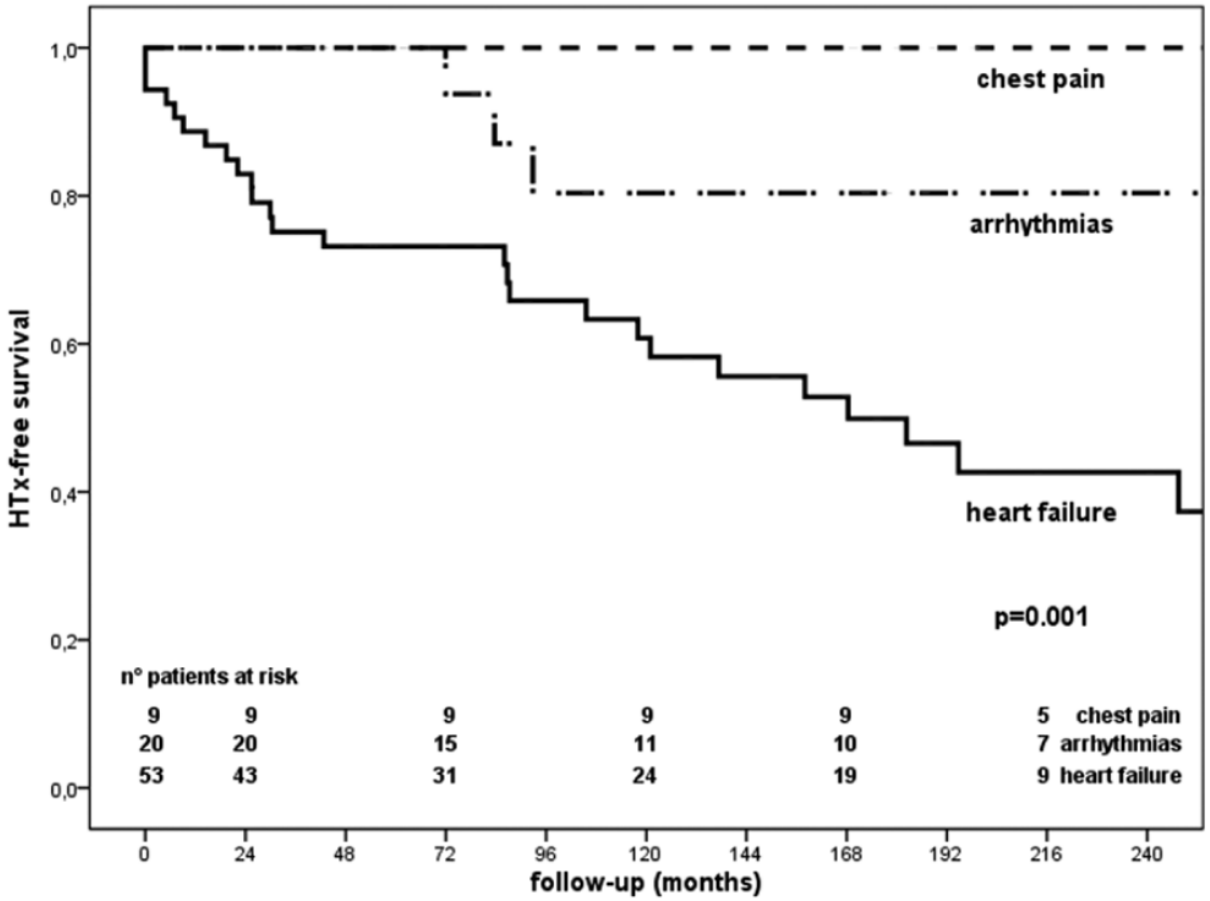
American Heart Association.

Long-Term Evolution and Prognostic Stratification of Biopsy-Proven Active Myocarditis

Marco Anzini, Marco Merlo, Gastone Sabbadini, Giulia Barbati, Gherardo Finocchiaro, Bruno Pinamonti, Alessandro Salvi, Andrea Perkan, Andrea Di Lenarda, Rossana Bussani, Jozef Bartunek and Gianfranco Sinagra

Circulation. 2013;128:2384-2394; originally published online October 1, 2013;
doi: 10.1161/CIRCULATIONAHA.113.003092

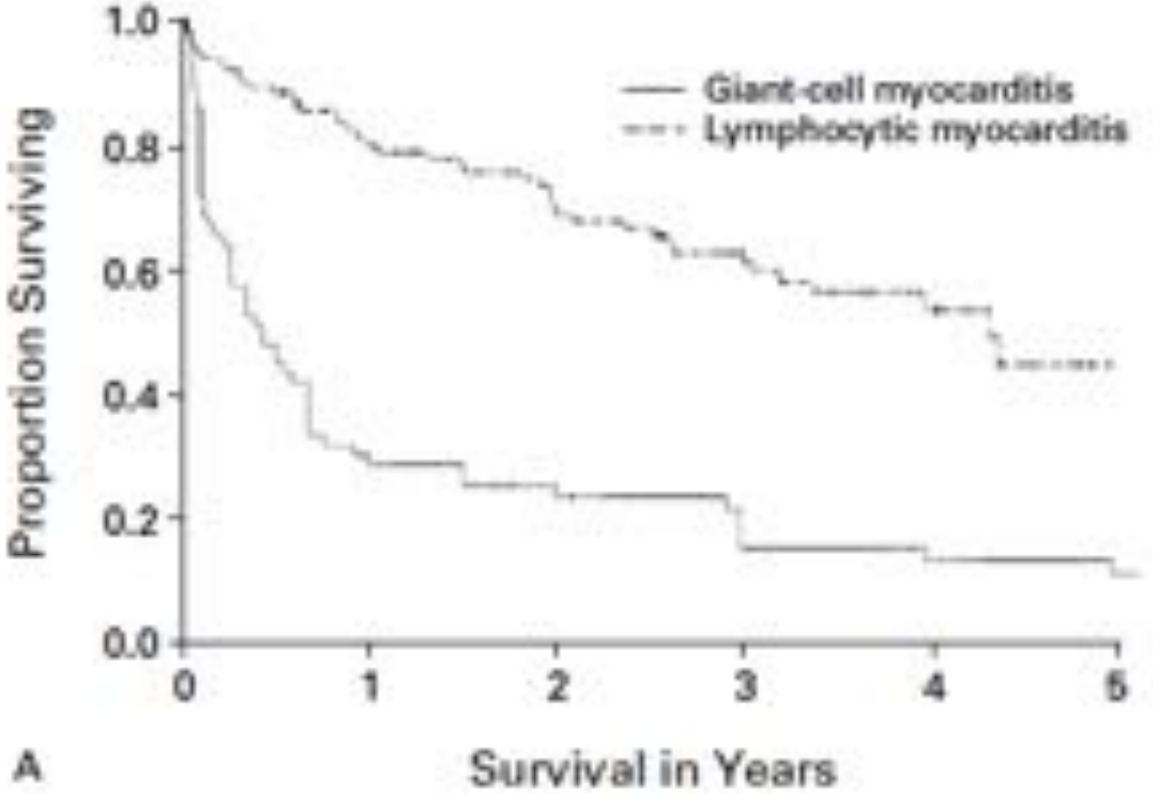
82 pts; Fup 147 mo;
53% 6 mo impr/norm LVEF



IDIOPATHIC GIANT-CELL MYOCARDITIS — NATURAL HISTORY AND TREATMENT

63 patient with biopsy proven giant cell myocarditis
mean age 42.6 ± 12.7 years

Duration of survival from the onset of symptoms



Cooper et al. N Engl J Med 1997;336:1860-6

Venoarterial Extracorporeal Membrane Oxygenation for Acute Fulminant Myocarditis in Adult Patients: A 5-Year Multi-Institutional Experience

THE ANNALS OF THORACIC SURGERY

Table 5. Review of Published Studies That Included 6 or More Adults Patients Affected by Acute Fulminant Myocarditis and Supported by ECMO

Reference	Time Span	Patients, n	ECMO Weaning, n (%)	Survival to Hospital Discharge, n (%)	Postoperative Survival, % (follow-up, years)
Kawahito et al [19]	1991–1997	6	5 (80)	5 (80)	NA
Aoyama et al [5]	1989–2000	52	42 (80.7)	31 (59.6)	NA
Chen et al [9]	1994–2001	15	14 (93)	11 (73)	NA
Asaumi et al [13]	1993–2001	6	4 (67)	4 (67)	NA
Maejima et al [14]	1991–2000	8	NA	6 (75)	100 (range, 1.4–5.9)
Sezai et al [20]	1999–2006	7	7 (100)	7 (100)	NA
Pages et al [6]	2001–2006	6	5 (83)	5 (83)	80% (1)
Thiagarajan et al [21]	1992–2007	16	NA	9 (56)	NA
Hsu et al [10]	1994–2009	51	NA	31 (61)	NA
Mirabel et al [12]	2002–2009	35	NA	24 (69)	100 (1.5)
Beurtheret et al [22]	2005–2009	14	NA	9 (65)	NA
Diddle et al [6]	1995–2014	147	101 (69)	90 (61)	NA

Cardiopulmonary support with VAECMO provides an invaluable tool in the treatment of AFM, although major complications may characterize the hospital course. Long-term outcome appears favorable with rare episodes of recurrent myocarditis or cardiac related events.

Lorusso et al. Ann Thorac Surg 2016;101:919–26

Venoarterial Extracorporeal Membrane Oxygenation for Acute Fulminant Myocarditis in Adult Patients: A 5-Year Multi-Institutional Experience

THE ANNALS OF THORACIC SURGERY

Table 2. ECMO-Related Data

Variable	Value
ECMO run, days	9.9 ± 19
Cardiac recovery	
No	14 (24.5)
Yes	43 (75.5)
Cardiac recovery time, days	9.0 ± 10.6
In-hospital major complications	
No	17 (29.9)
Yes	40 (70.1)

Table 4. Multivariate Analysis

Variable	β	SE	Exp (β)	*p* Value
pH before ECMO implantation	−14.251	7.148	.000	0.046
Lactate normalization, hours from ECMO implantation	0.029	0.012	1.029	0.013
Cardiac recovery	5.288	1.769	197.930	0.003

Lorusso et al. Ann Thorac Surg 2016;101:919–26

Contents lists available at ScienceDirect

International Journal of Cardiology

journal homepage: www.elsevier.com/locate/ijcard

Correspondence

Arrhythmic risk prediction of acute myocarditis presenting with life-threatening ventricular tachyarrhythmias

Marco Anzini, Marco Merlo *, Jessica Artico, Gianfranco Sinagra

Cardiovascular Department, "Ospedali Riuniti" and University of Trieste, Italy

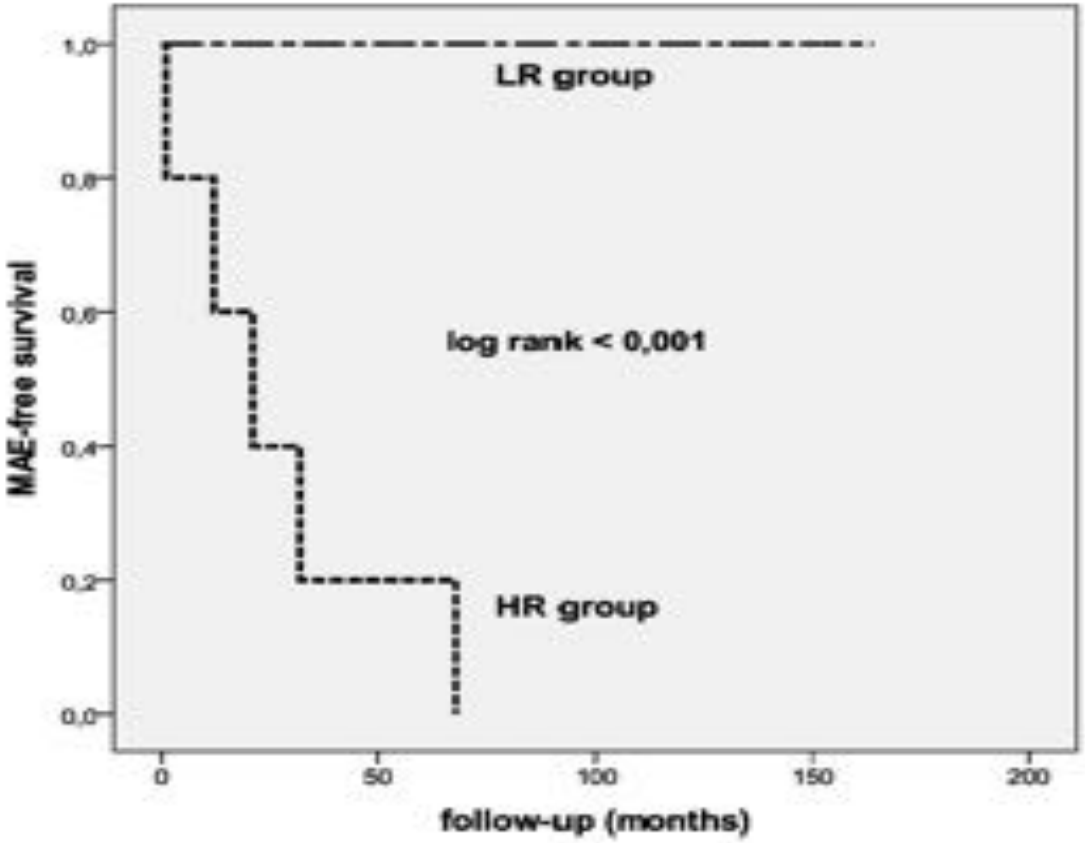


Fig. 1. Kaplan–Meier curves — MAE-free survival in HR vs LR patients. HR: high risk; LR: low risk; MAE: major arrhythmic events.

ALTO RISCHIO, se 2 tra

Esordio con SCC clinico

EF basale < 35%

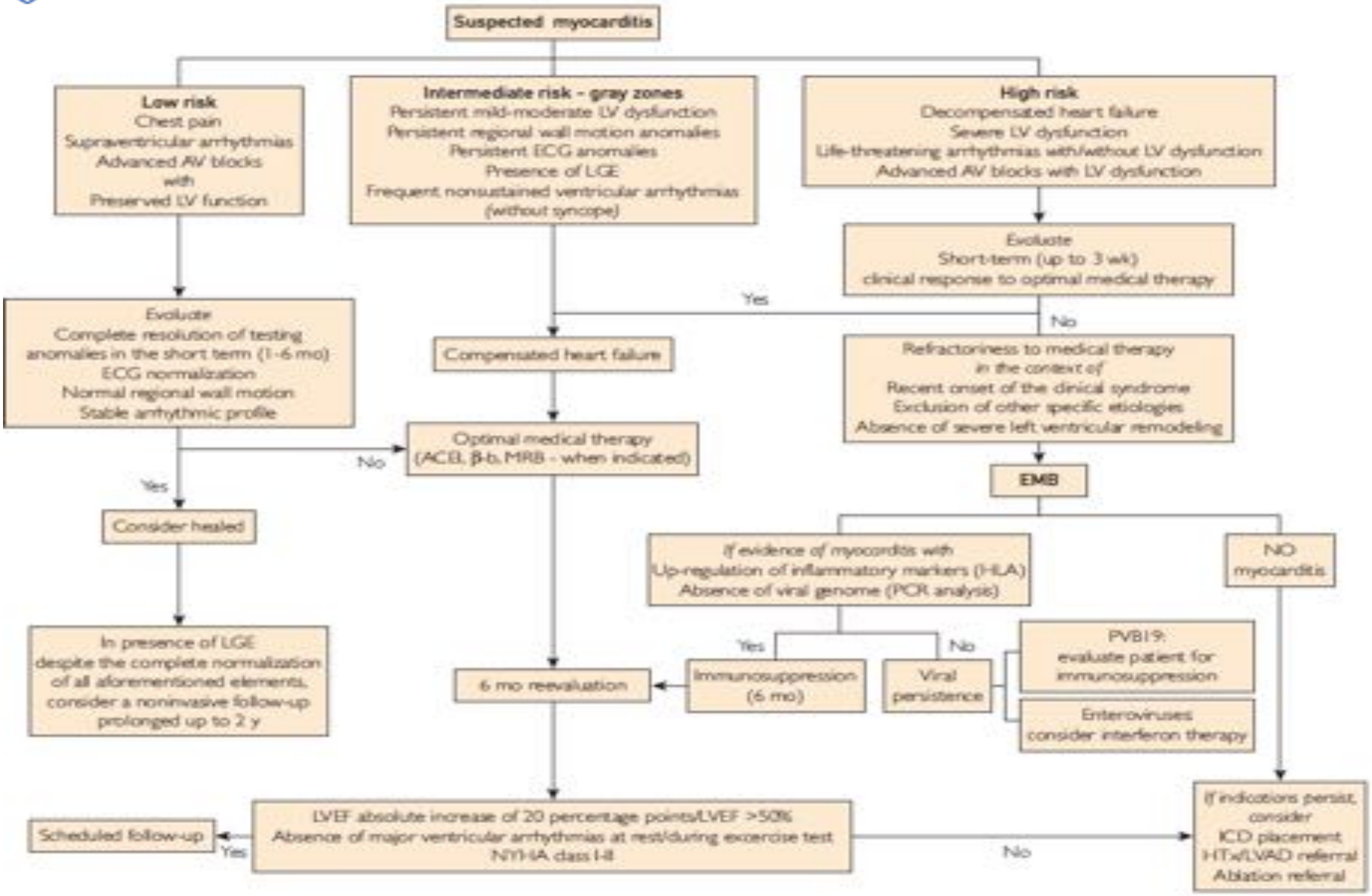
Segmenti acinetici / Evoluzione aneurismatica

LGE +

Int J Cardiol 2016; 212:169-170

MAYO CLINIC

Myocarditis in Clinical Practice

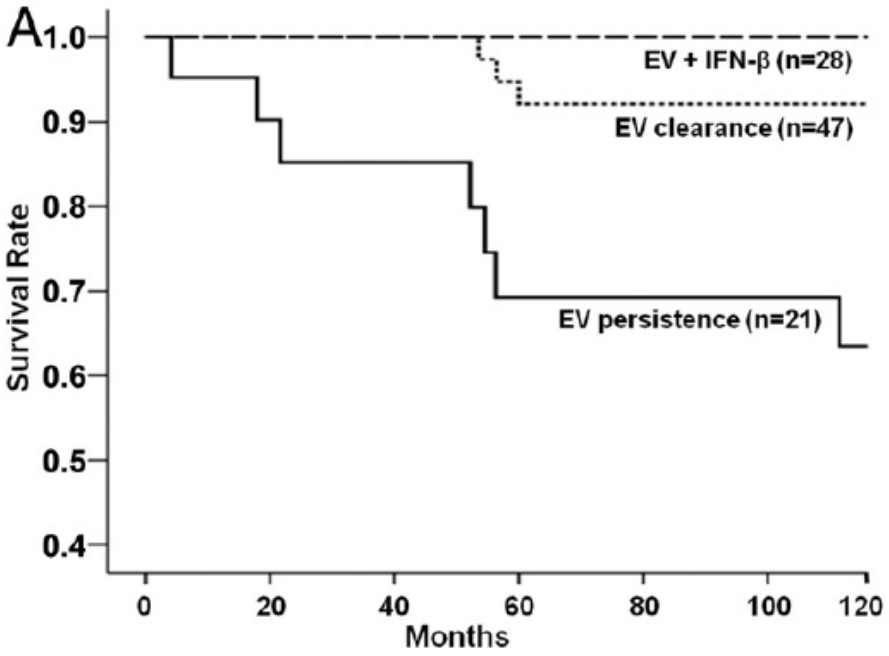


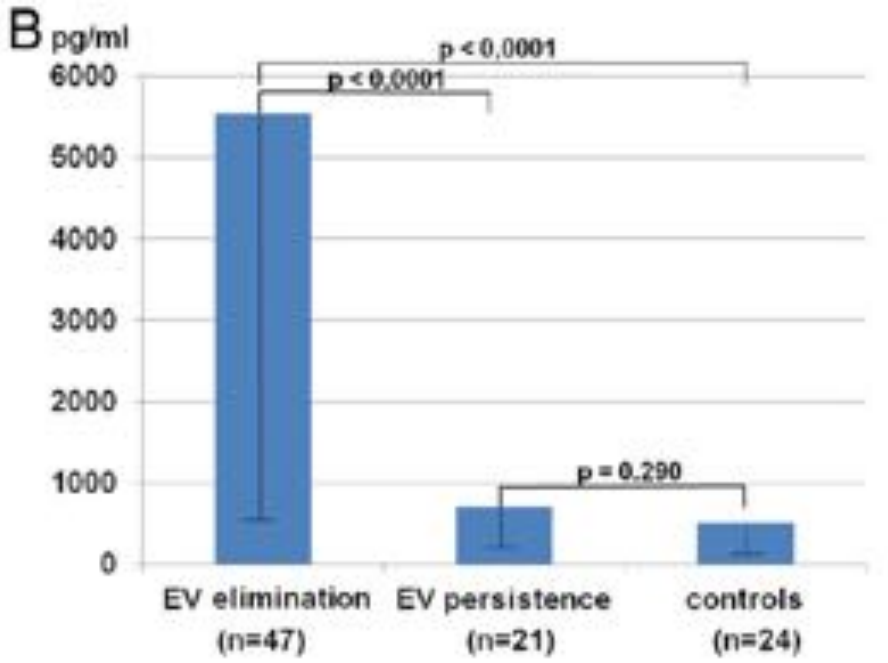
Sinagra et al Mayo Clin Proc. ■ September 2016;91(9):1256-1266

Research Correspondence

JACC Vol. 60, No. 14, 2012
October 2, 2012:1295–6

Interferon-Beta Improves Survival in Enterovirus-Associated Cardiomyopathy





Anti-viral therapies: interferon-beta

Betaferon in chronic viral cardiomyopathy (BICC) trial: Effects of interferon-β treatment in patients with chronic viral cardiomyopathy

Response variable	Adeno/enterovirus (stratum 1, $n = 15$)		Parvovirus (stratum 2, $n = 128$)		Differences between strata	
	Placebo ($n = 6$)	IFN-β-1b ($n = 9$)	Placebo ($n = 42$)	IFN-β-1b ($n = 86$)	p (two-sided)	p treatment effect (interaction test)
Overall response (virus elimination/reduction, primary)	1 (16.7 %)	4 (44.4 %)	8 (19.0 %)	29 (33.7 %)	0.646	0.652
NYHA improvement	($n = 5$)	($n = 7$)	($n = 37/39$)	($n = 80$)		
Week 0–12	1 (20.0 %)	6 (85.7)	7 (18.9 %)	29 (36.3 %)	**0.039**	0.160
Week 0–24	1 (20.0 %)	6 (85.7)	11 (28.2 %)	31 (38.8 %)	0.100	0.094
Randomised untreated patients	3		3			

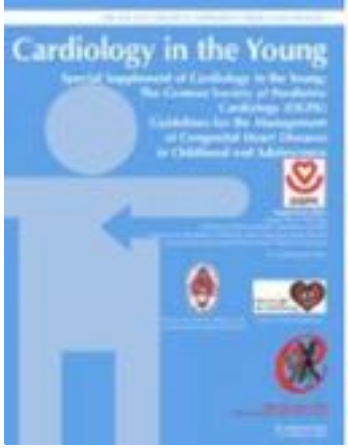
Differences were considered to be statistically significant at a value of <0.05 (bold value)

Response variable	Treatment effect	
	Odds ratio IFN-β-1b vs. Placebo (adjusted for strata)	p (two-sided)
Overall response (virus elimination, primary)	**2.33**	**0.0487**
NYHA improvement		
Week 0–12	**3.19**	**0.013**
Week 0–24	2.08	0.073

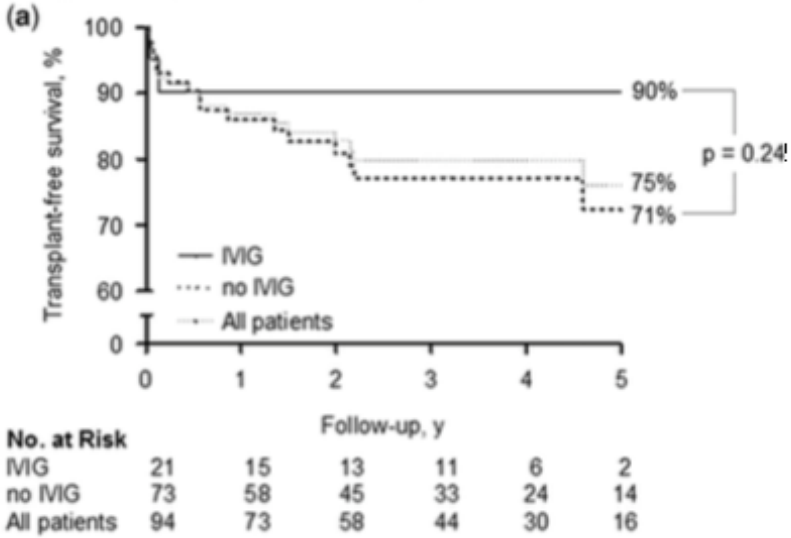
Differences were considered to be statistically significant at a value of <0.05 (bold values)

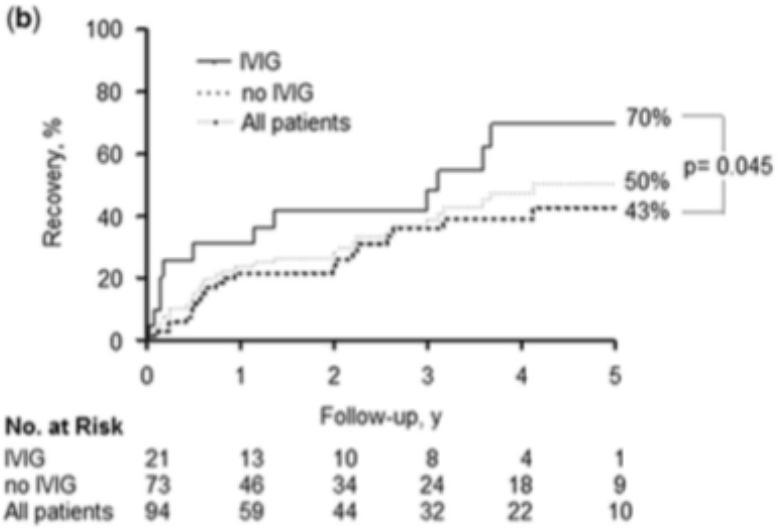
Schultheiss et al. Clin Res Cardiol. 2016 Sep;105(9):763-73

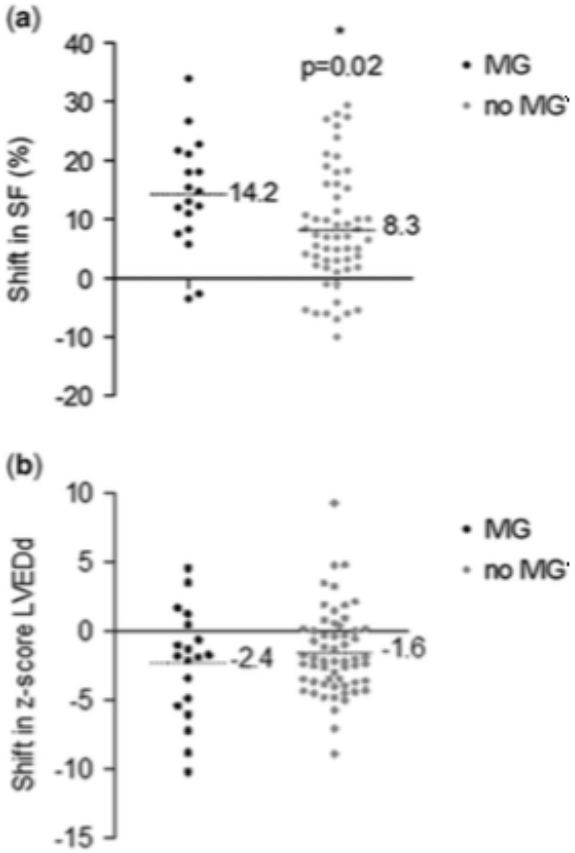
High dose intravenous immunoglobulin



Intravenous immunoglobulins in children with new onset dilated cardiomyopathy





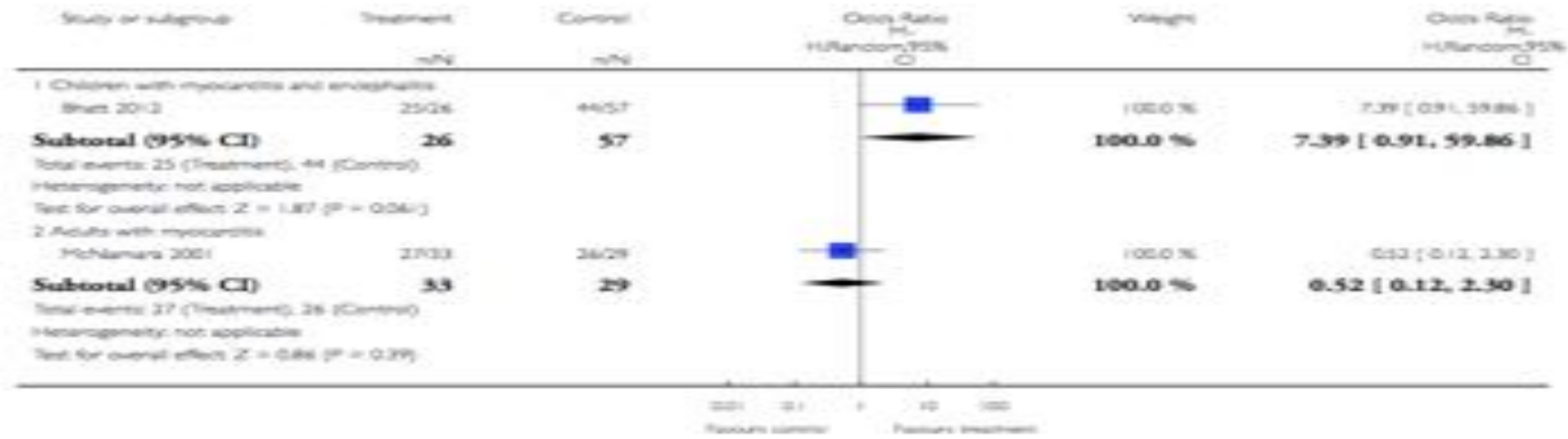


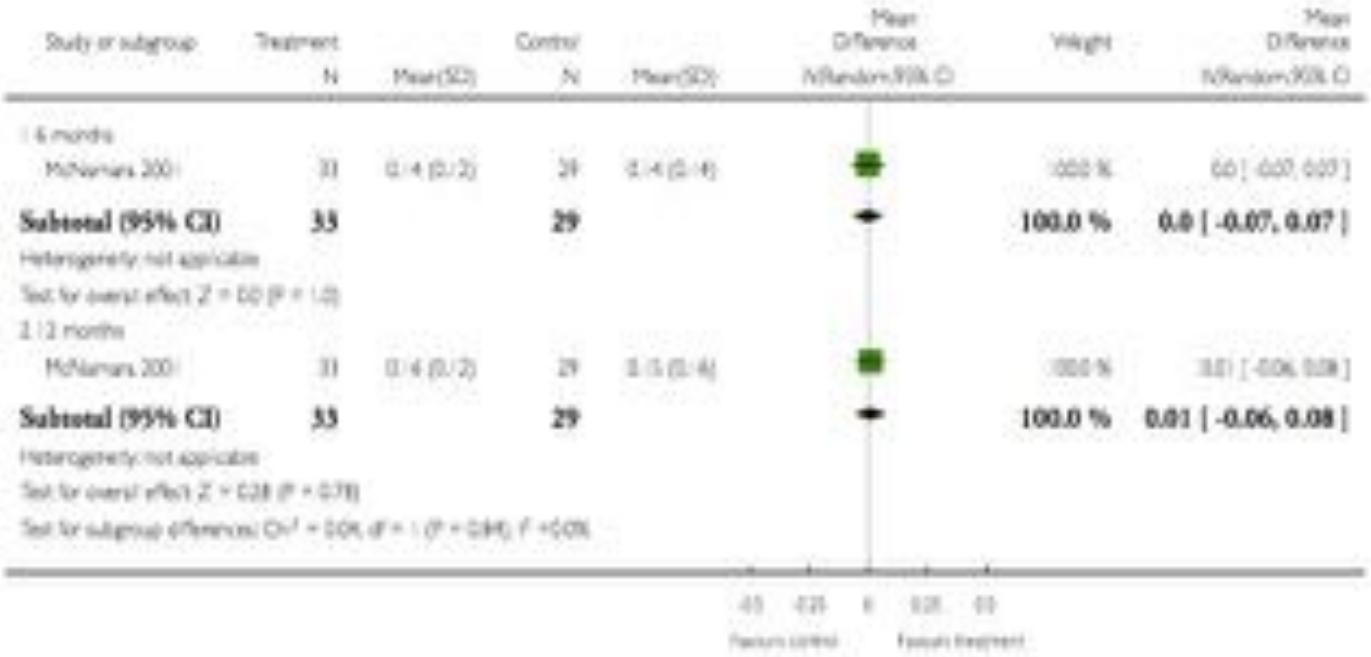
Heidendael et al, Cardiology in the Young 2017

High dose intravenous immunoglobulin

Cochrane Library
Cochrane Database of Systematic Reviews

Intravenous immunoglobulin for presumed viral myocarditis in children and adults





Change in LVEF

Robinson et. Al Cochrane Database Syst Rev. 2015 May 20;(5)

Apheresis in the Treatment of Idiopathic Dilated Cardiomyopathy

Jeffrey L. Winters*

Studies examining patients with iDCM have found the presence of viral genome in endomyocardial biopsies in 20–67% of patients. Viruses identified include parvovirus B19 (36.6%), enterovirus (32.6%), human herpes virus 6 (10.5%), and adenovirus (8.1%).

TABLE I. Cardiac Autoantibodies Identified in Idiopathic Dilated Cardiomyopathy [10,11,12,18]

Antigen	Frequency in iDCM (%)	Frequency in ischemic cardiomyopathy (%)	Frequency in healthy controls (%)	Clinical associations
α-Myosin	23–66	4–21	0–2.5	Worsening LVEF and increased diastolic stiffness
β1-Adrenoreceptor	26–46	10–13	1–10	Depressed myocardial function, greater incidence of ventricular arrhythmia, and greater incidence of sudden cardiac death. Presence predicts increased risk of all-cause and cardiovascular mortality
Troponin-I	50	NA	NA	
Na-K-ATPase	26	NA	2	Independent predictor of poor systolic function, ventricular tachycardia, and sudden cardiac death
M2—muscarinic acetylcholine receptor	40	NA	8	Greater incidence of atrial fibrillation

The available evidence suggests that apheresis may be an effective treatment

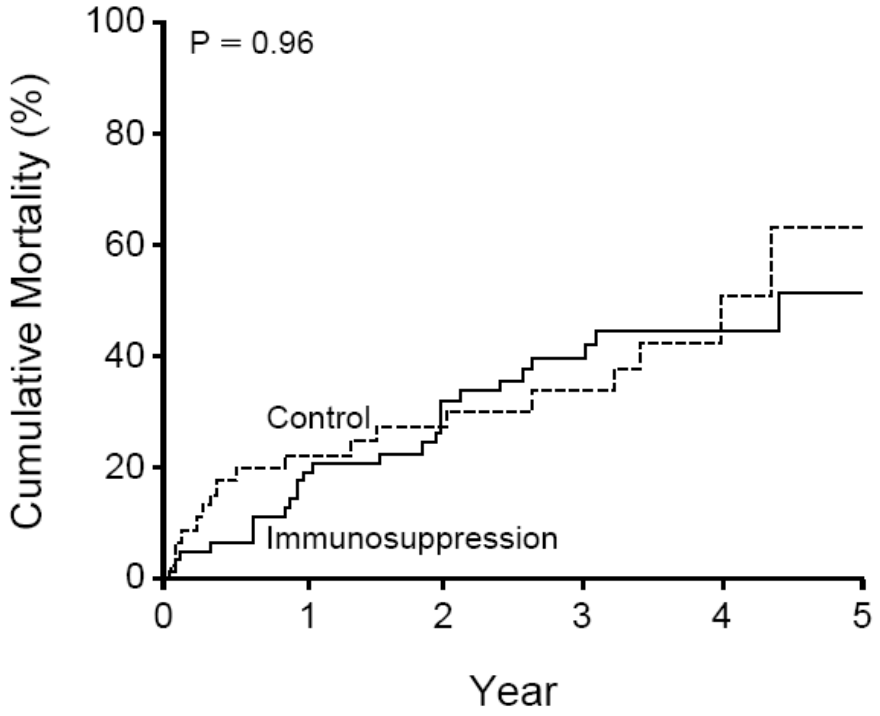
Journal of Clinical Apheresis 2012

The New England
Journal of Medicine

Volume 333 AUGUST 3, 1995 Number 5

A CLINICAL TRIAL OF IMMUNOSUPPRESSIVE THERAPY FOR MYOCARDITIS

JAY W. MASON, M.D., JOHN B. O'CONNELL, M.D., AHVIE HERSKOWITZ, M.D., NOEL R. ROSE, M.D., PH.D., BRUCE M. MCMANUS, M.D., PH.D., MARGARET E. BILLINGHAM, M.D., THOMAS E. MOON, PH.D., AND THE MYOCARDITIS TREATMENT TRIAL INVESTIGATORS*



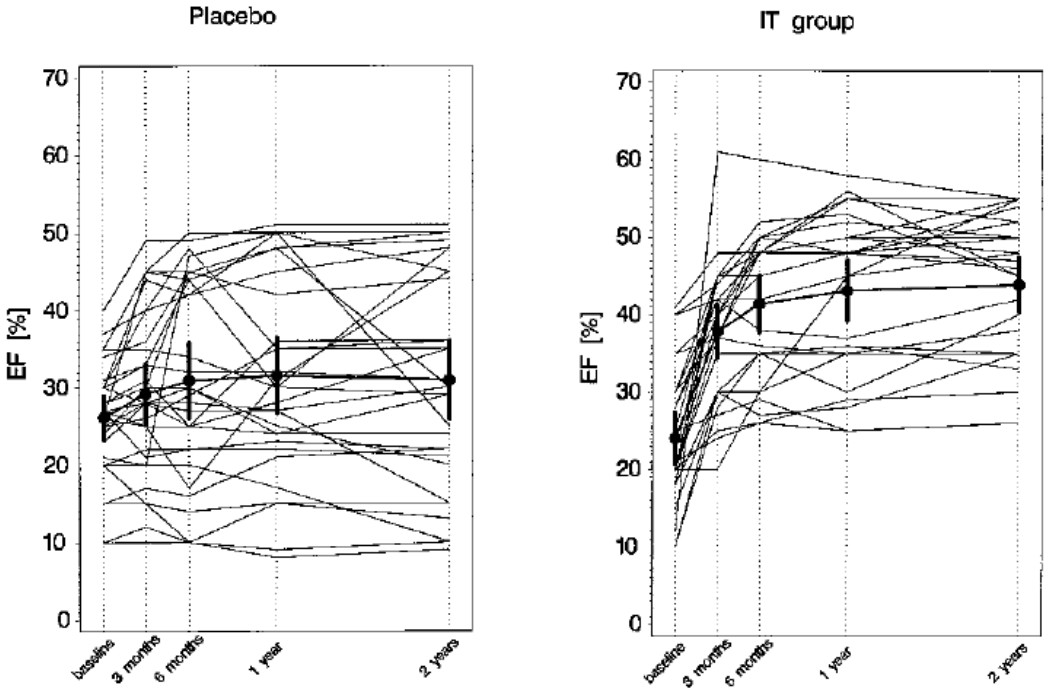
Pazienti con durata di malattia fino a 2 anni

Arruolameno in base a istopatologia tradizionale (Dallas)

Randomized, Placebo-Controlled Study for Immunosuppressive Treatment of Inflammatory Dilated Cardiomyopathy : Two-Year Follow-Up Results

Romuald Wojnicz, Ewa Nowalany-Kozielska, Celina Wojciechowska, Grazyna Glanowska, Przemyslaw Wilczewski, Tomasz Niklewski, Marian Zembala, Lech Polonski, Marius M. Rozek and Jan Wodniecki

Circulation 2001, 104:39-45



Studio Randomizzato Controllato

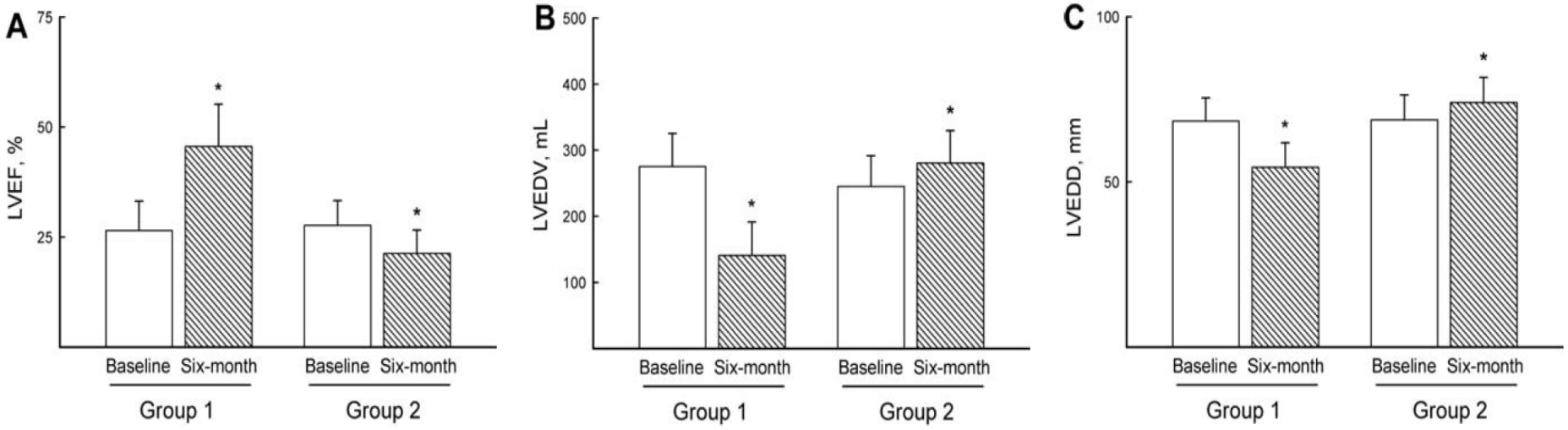
Pazienti con sintomi di SCC da almeno 6 mesi

Arruolameno in base a Immunoistochimica (Immunoattivazione)

Randomized study on the efficacy of immunosuppressive therapy in patients with virus-negative inflammatory cardiomyopathy: the TIMIC study

European Heart Journal (2009) **30**, 1995–2002

Andrea Frustaci[1,2]*, Matteo A. Russo[3,4], and Cristina Chimenti[1,2,4]

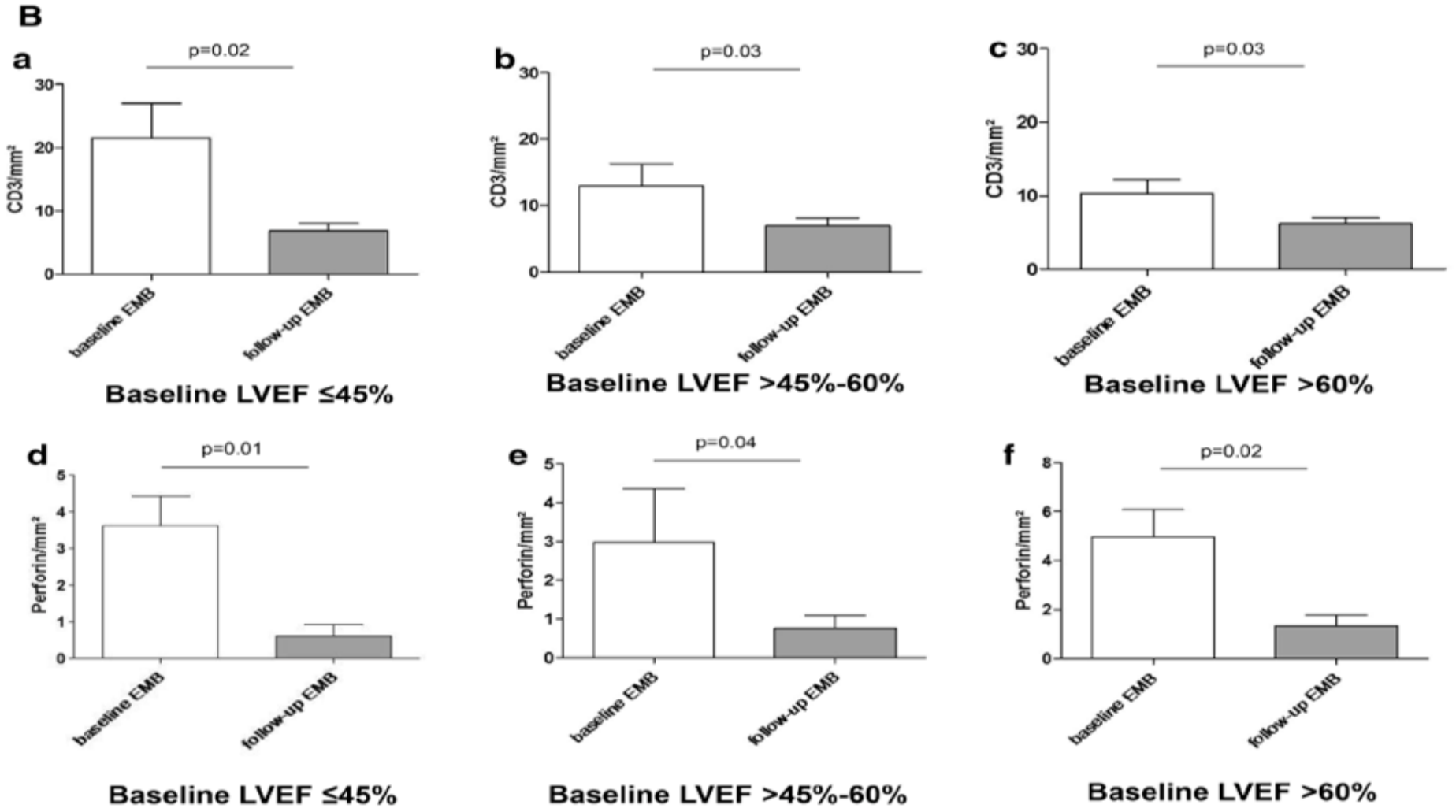


Studio Randomizzato Controllato

Pazienti con sintomi di SCC da almeno 6 mesi

Eleggibilità se Immunoistochimica positiva e Virologia negativa

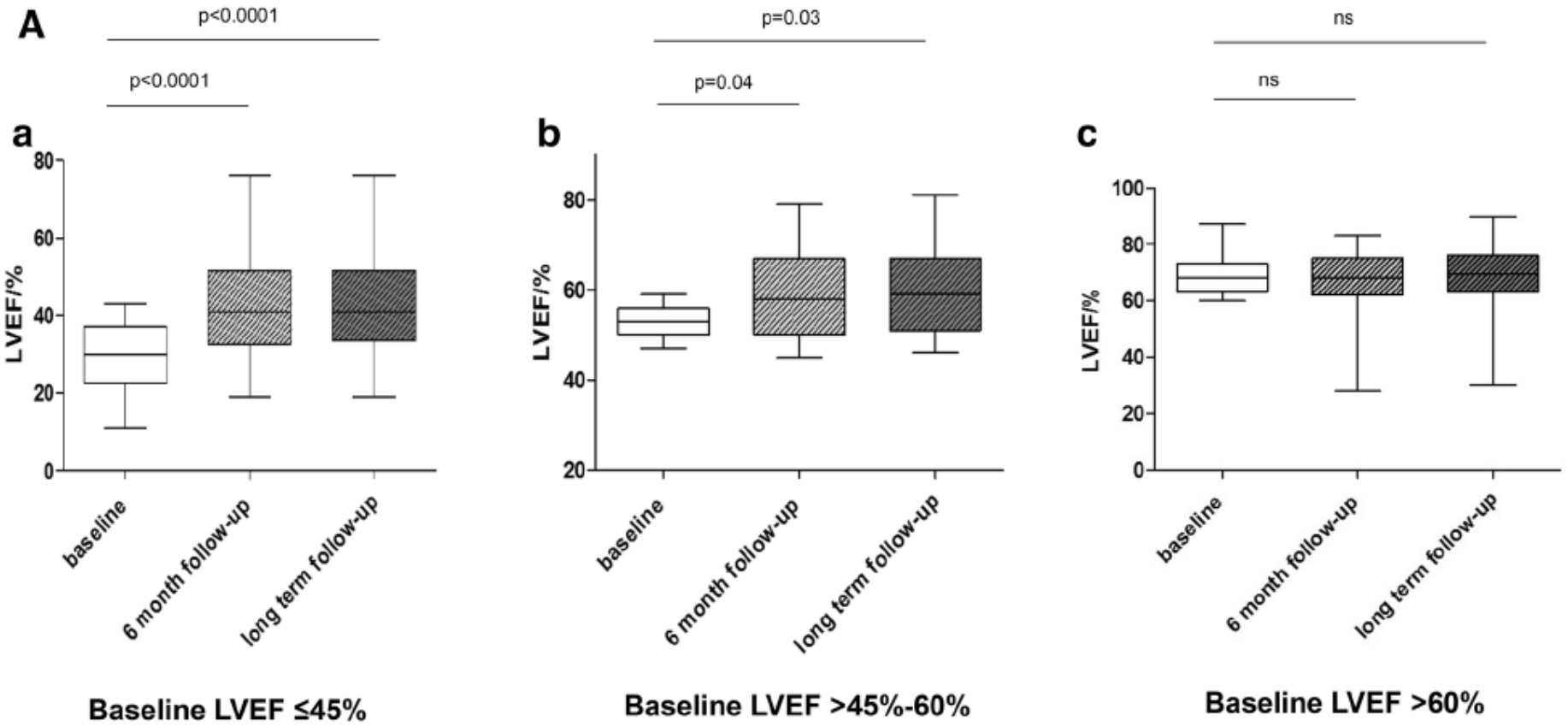
Long-term outcome of patients with virus-negative chronic myocarditis or inflammatory cardiomyopathy after immunosuppressive therapy



Immunohistochemical detection of intramyocardial Inflammation according to baseline LVEF

Echer et al. Clin Res Cardiol. 2016

Long-term outcome of patients with virus-negative chronic myocarditis or inflammatory cardiomyopathy after immunosuppressive therapy



Hemodynamic course at 6 months and at a long-term follow-up period according to baseline LVEF

Echer et al. Clin Res Cardiol. 2016

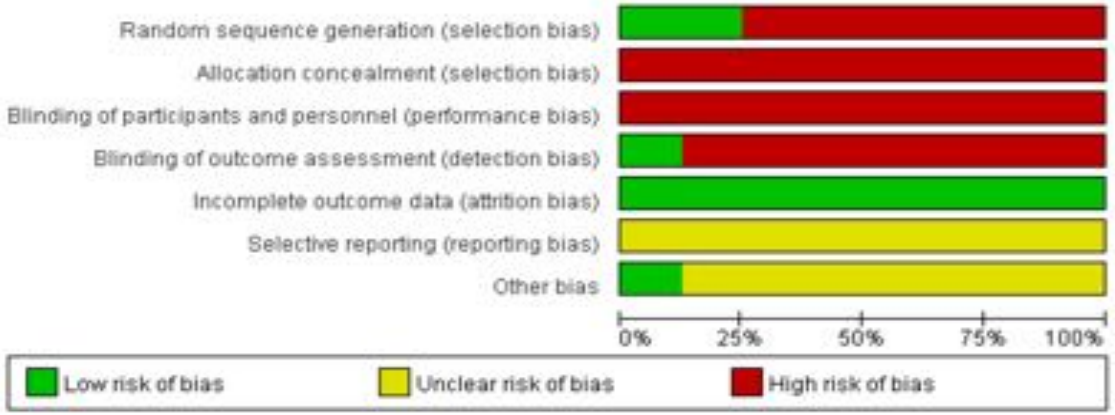
[Intervention Review]

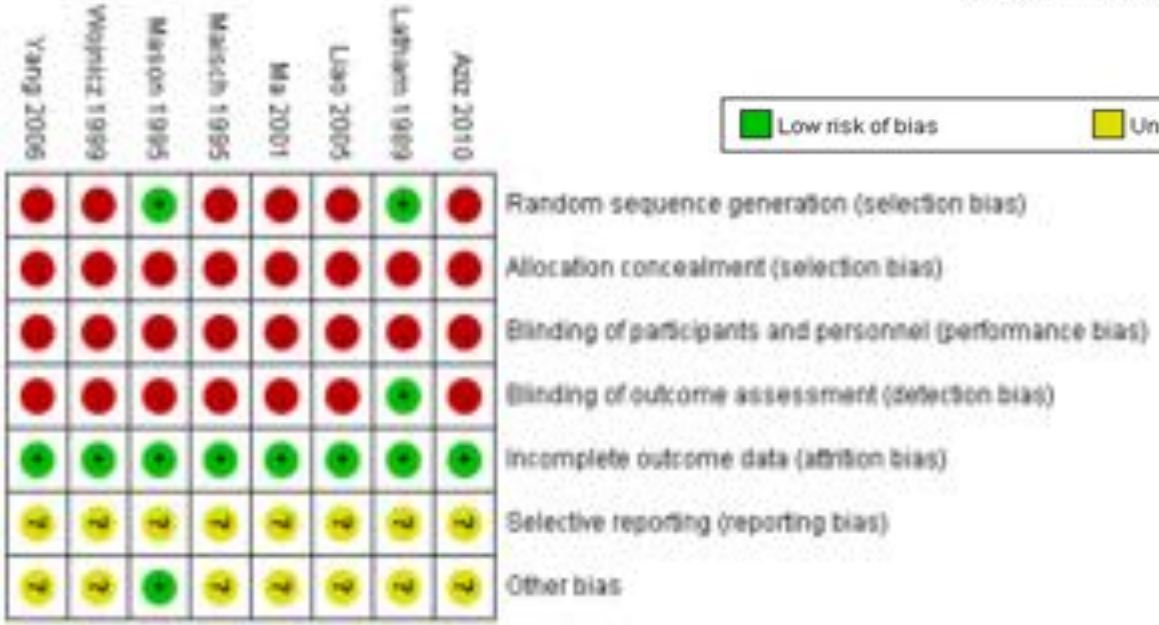
Corticosteroids for viral myocarditis

Huai Sheng Chen[1], Wei Wang[2], Sheng Nan Wu[3], Jian Ping Liu[4]

[1]Intensive Care Unit, Shenzhen People's Hospital, The Second Affiliated Hospital of Ji Nan University, Shenzhen City, China. [2]Endocrinology, Shenzhen People's Hospital, Shenzhen City, China. [3]Intensive Care Unit, Shenzhen People's Hospital, The Second Affiliated Hospital of Ji Nan University, Shenzhen City, China. [4]Centre for Evidence-Based Chinese Medicine, Beijing University of Chinese Medicine, Beijing, China

THE COCHRANE COLLABORATION®





Chen, et al. Cochrane Database of Systematic Reviews 2013, Issue 10.

[Intervention Review]

Corticosteroids for viral myocarditis

Huai Sheng Chen[1], Wei Wang[2], Sheng Nan Wu[3], Jian Ping Liu[4]

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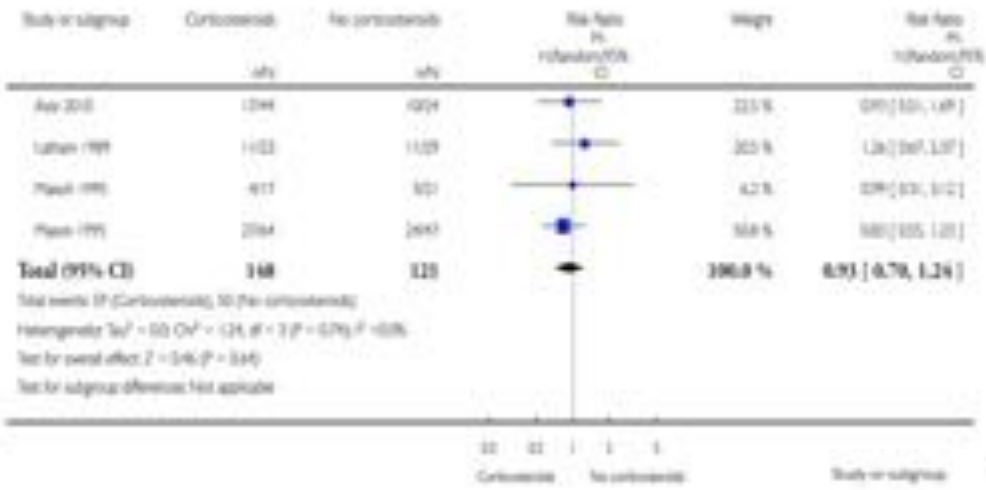
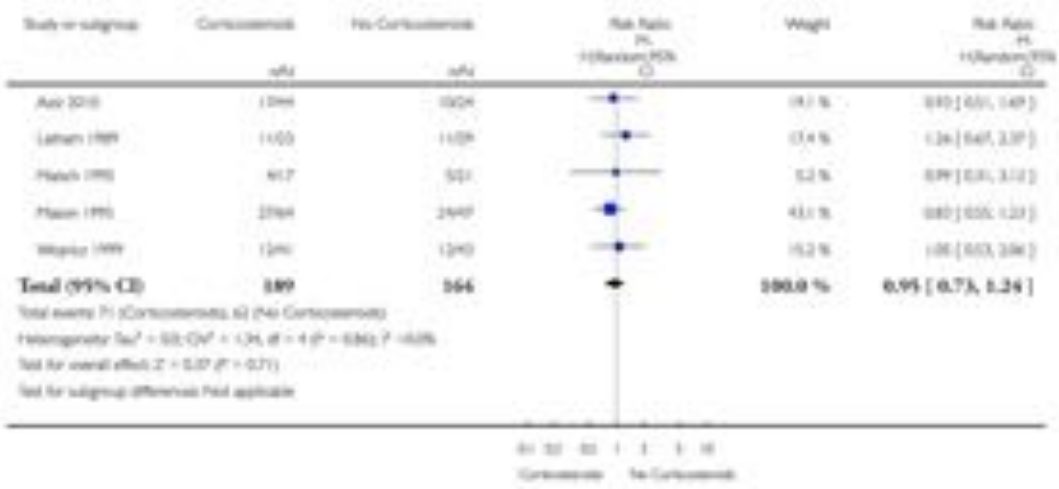


All cause mortality

CV death or Cardiac trasplant



Chen et al. Cochrane Database of Systematic Reviews 2013, Issue 10

nature REVIEWS CARDIOLOGY

Viral myocarditis—diagnosis, treatment options, and current controversies

Ari Pollack, Amy R. Kontorovich, Valentin Fuster and G. William Dec

Box 1 | Current recommendations for immunosuppressive therapy from the ESC

1. Immunosuppression should be started only after ruling out active infection on EMB by PCR
2. Based on experience with noncardiac autoimmune disease, consideration of immunosuppression in proven autoimmune (for example, infection-negative) forms of myocarditis, should be made if no contraindications to immunosuppression are present, including giant-cell myocarditis, cardiac sarcoidosis, and myocarditis associated with known extracardiac autoimmune disease
3. Steroid therapy is indicated in cardiac sarcoidosis in the presence of ventricular dysfunction and/or arrhythmia and in some forms of infection-negative eosinophilic or toxic myocarditis with heart failure and/or arrhythmia
4. Immunosuppression can be considered, on an individual basis, in infection-negative lymphocytic myocarditis refractory to standard therapy in patients with no contraindications to immunosuppression
5. Follow-up EMB can be required to guide the intensity and the length of immunosuppression

Abbreviations: EMB, endomyocardial biopsy; ESC, European Society of Cardiology. Adapted from Caforio, A. L. P. *et al.* Current state of knowledge on aetiology, diagnosis, management, and therapy of myocarditis: a position statement from the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. *Eur. Heart J.* **34** (33), 2636–2648 © (2013), with permission from Oxford University Press and the European Society of Cardiology.

Protocolli di immunosoppressione

Protocollo utilizzato da Wojnicz R et al. Circulation 2001

FARMACO	DOSAGGIO
Prednisone	1 mg/kg/die per 12 giorni, ndi riduzione della dose di 5 mg/die ogni 5 giorni fino alla dose di 0,2 mg/kg/die, per un totale di 90 giorni
Azatioprina	1 mg/kg/die per un totale di 100 giorni

Protocollo utilizzato da Frustaci et al. Eauropean Heart Journal 2009

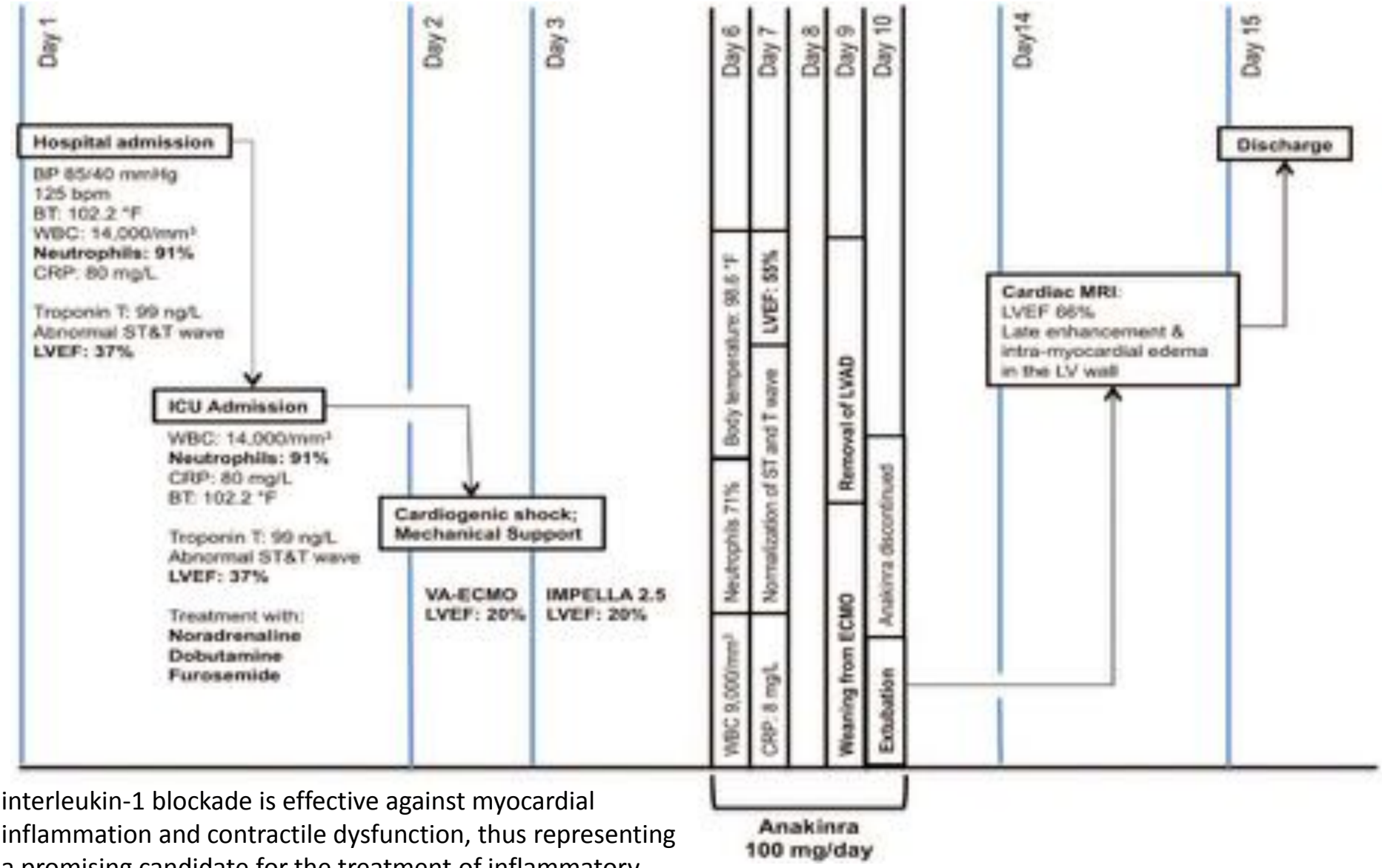
FARMACO	DOSAGGIO
Prednisone	1 mg/kg/die per 4 settimane, quindi 0.33 mg/kg/die per 5 mesi
Azatioprina	2 mg/kg/die per 6 mesi

Protocollo utilizzato presso la SC Cardiologia di Trieste

FARMACO	DOSAGGIO
Prednisone	50 mg/m^2/die per 2 settimane indi scalo di 0,3 mg/kg per due mesi, indi scalo gradualmente fino allo stop (6°mese)
Azatioprina	75 mg/m^2/die per 6 mesi
Ciclosporina*	10 mg/kg/die (2 somministrazioni) per 6 mesi

Controllo con biopsia endomiocardica a 2 e 6 mesi.
*** In casi selezionati (es. miocardite a cellule giganti) o in caso di persistente attività infiammatoria nonostante terapia con prednisone.**

Treating Life-Threatening Myocarditis by Blocking Interleukin-1



interleukin-1 blockade is effective against myocardial inflammation and contractile dysfunction, thus representing a promising candidate for the treatment of inflammatory heart failure

Cavalli et al. Crit Care Med 2016;

MAYO CLINIC

Proposal for the Scheduled Follow-up of Patients With Myocarditis

Variable	Low risk	Intermediate risk	High risk
Time of clinical reevaluations	1 mo, 6 mo, 2 y	3 mo, 6 mo, 12 mo, then yearly	3 wk, 3 mo, 6 mo, 12 mo, then yearly
Noninvasive testing	Assess ECG and echocardiographic normalization between 1 and 6 mo. Cardiac MRI is recommended	Periodic evaluation of LVEF and LV remodeling (ECG) Periodic evaluation of the arrhythmic burden (Holter monitoring) Annual evaluation of arrhythmia induction during exercise test Cardiac MRI with LGE evaluation, if not assessed at disease presentation	
Exercise restriction	Yes, for 2 y	Yes, lifetime	Yes, lifetime
Lifetime follow-up	No, if normalization at 2 y	Yes	Yes
Lifetime therapy	No, if normalization at 2 y	Yes	Yes

ECG = electrocardiography; LGE = late gadolinium enhancement; LV = left ventricular; LVEF = LV ejection fraction; MRI = magnetic resonance imaging.

Myocarditis in Clinical Practice

Sinagra G et al, Mayo Clin Proc- September 2016: 91(9):1256-1266

Take home message

- modello ad alta reversibilità del quadro d'esordio e/o della disfunzione;
- spazio per terapia convenzionale ed osservazione evoluzione (prime 2-4 settimane);
- CRM nelle prime 2 sett;
- nei casi con SC progressivo o refrattario e/o con disfunzione Vsin persistente <40% o progressiva o con aritmie VE maggiori, vi sono indicazioni a BEM;
- se BEM + con immunoistochimica+ e virologia molecolare -, vi sono indicazioni a tp immunosoppressiva (steroide+AZA);
- dati controversi su IgEv, aferesi, beta-IFN;
- dati promettenti su inibitori IL per casi selezionati;
- indicazioni e modalità defibrillatore (ICS, sICD, LifeVEST) da discutere caso per caso;
- importanza del Fup per la gestione e rimodulazione tp