Μυοκαρδίτιδα- Περικαρδίτιδα

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ΝΟΣΟΚΟΜΕΙΟ ΑΤΤΙΚΟΝ

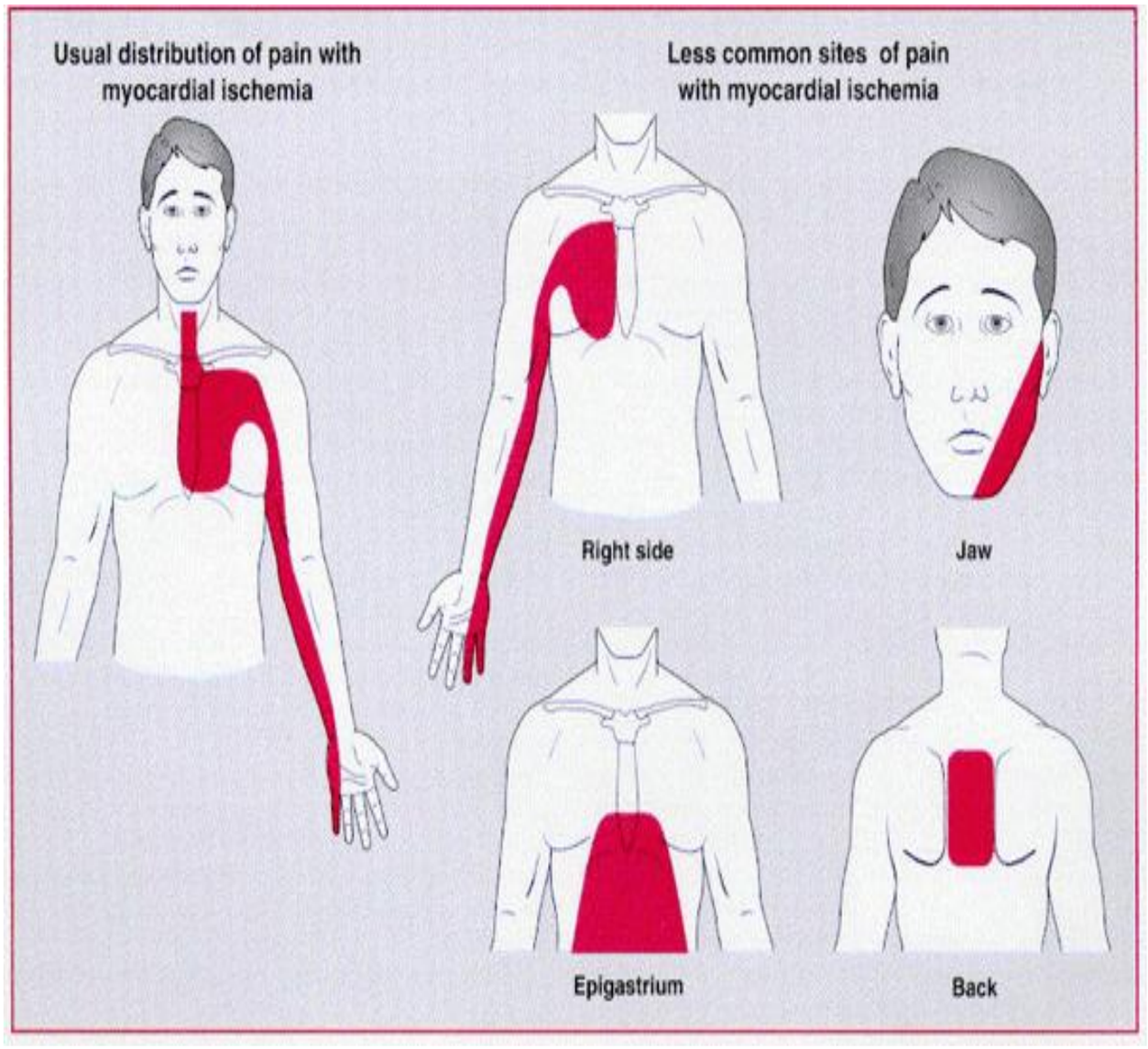
Διερεύνηση θωρακικού άλγους- Καρδιακά αίτια

Καρδιακά αίτια

- Στεφανιαία νόσος
  - Στηθάγχη προσπάθειας
  - Οξέα στεφανιαία σύνδρομα
- **Περικαρδίτιδα-μυοκαρδίτιδα**
- Διαχωρισμός Αορτής
- Στένωση Αορτής
- Πνευμονική εμβολή

Κλινική ταξινόμηση του προκάρδιου άλγους

- Τυπική στηθάγχη: πληρεί και τα 3 χαρακτηριστικά
  - Οπισθοστερνικο άλγος– χαρακτηριστικές επεκτάσεις-διάρκεια
  - έκλυση με άσκηση η συναισθηματική φόρτιση
  - βελτίωση με ανάπαυση η νιτρογλυκερίνη
- Πιθανή στηθάγχη: πληρεί δυο από τα παραπάνω κριτήρια
- Μη καρδιακής προέλευσης θωρακικό άλγος : Πληρεί ένα η κανένα από τα παραπάνω κριτήρια



Stable Angina guidelines ESC 2005

Definition

- **Myocarditis (WHO):** i**nflammatory disease of the heart muscle**, diagnosed by established histological, immunological, and immunohistochemical criteria

- **Inflammatory cardiomyopathy (WHO):** myocarditis and cardiac dysfunction

- **Viral myocarditis:** histological evidence of myocarditis and positive viral PCR

- **Autoimmune myocarditis:** histological evidence of myocarditis and negative viral PCR, (with or without serum cardiac autoantibodies)

Richardson et al, Circulation 1996
Caforio et al, Eur Heart J 2013

Etiology

Causes	Examples
Infectious	viral, bacterial, fungal, parasitic, protozoal, richettsial, spirochetal
Immune-mediated	- auto-antigens (Giant cell, sarcoidosis, SLE etc) - allo-antigens (transplant rejection) - allergens (penicillin etc)
Toxic	chemotherapy, heavy metals, scorpion sting, radiation, pheochromocytoma etc

Caforio et al, Eur Heart J 2013

Etiology: key points

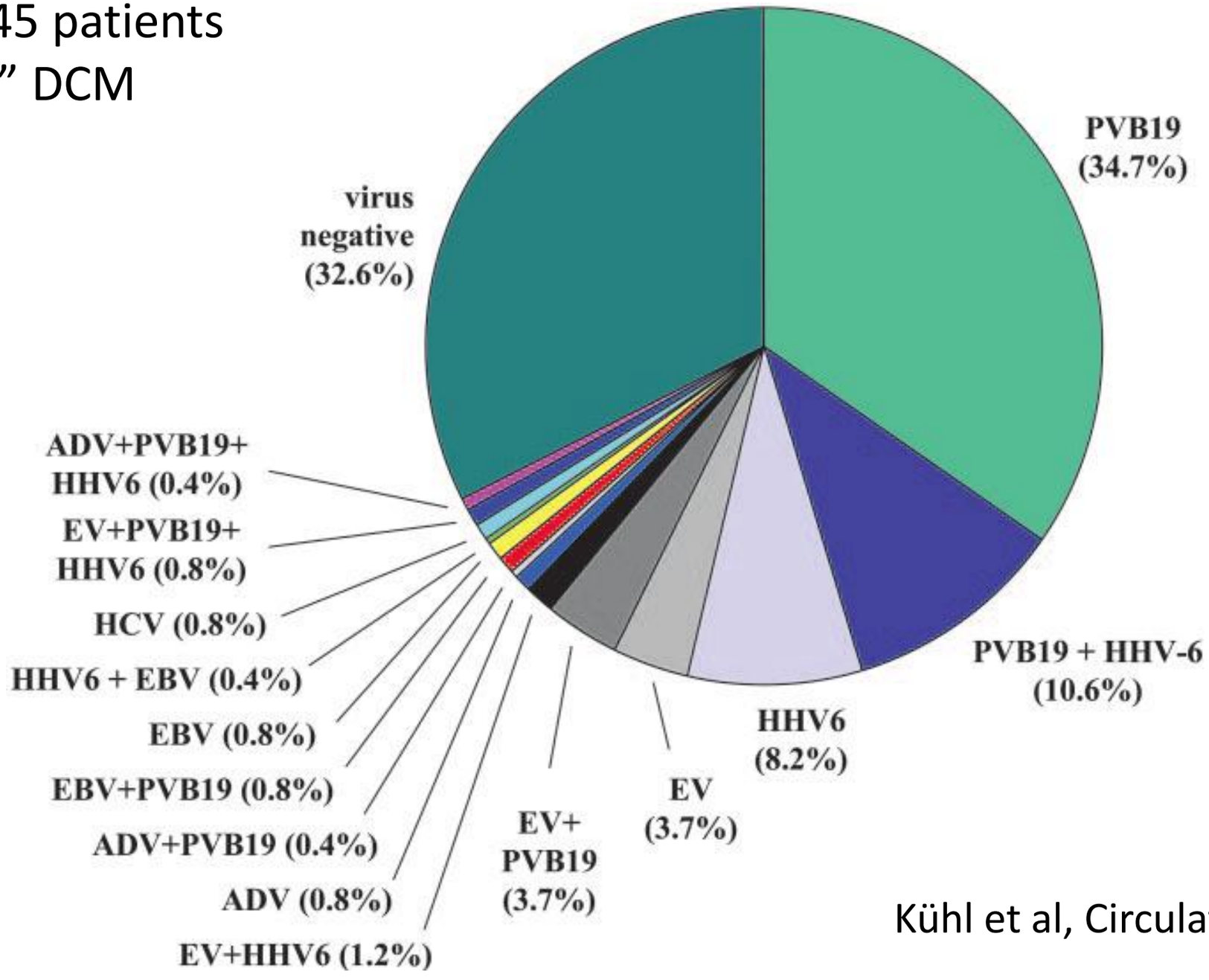
- New molecular techniques (PCR, in situ hybridization):
  virus spectrum shifted from enterovirus and adenovirus to **parvovirus B19** and **human herpesvirus 6**

- **Regional variation** of infectious causes epidemiology

Breinholt et al, J Heart Lung Transplant 2010

Epidemiology: key points

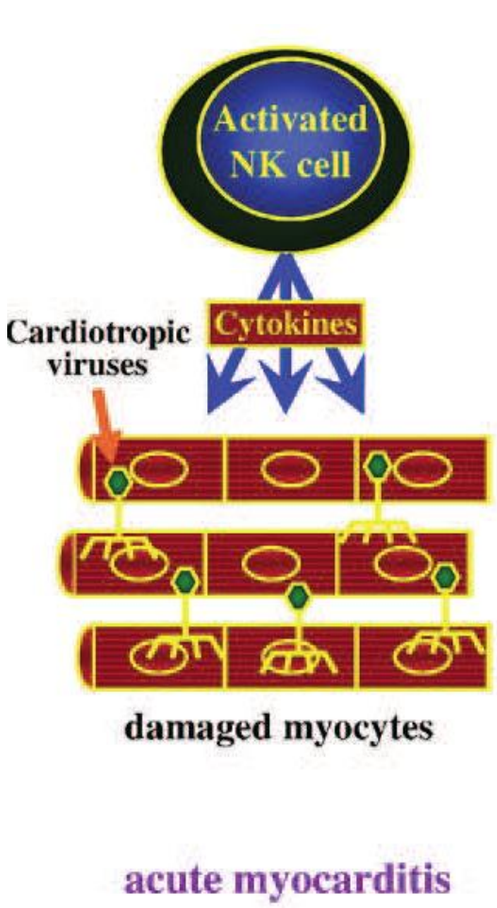
Biopsies from 245 patients with “idiopathic” DCM

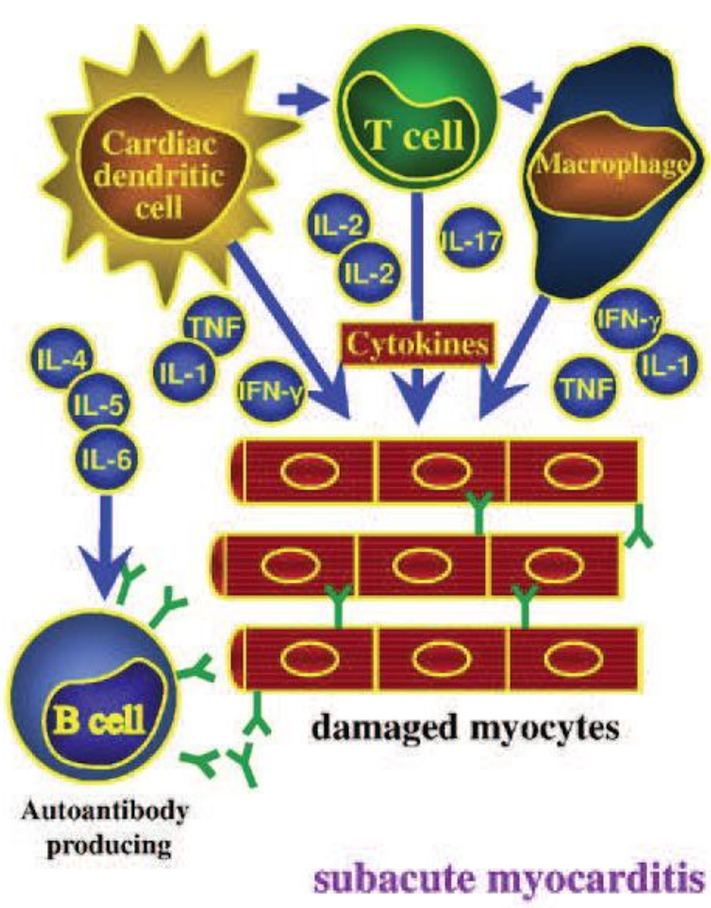


Kühl et al, Circulation 2005

Pathophysiology: key points

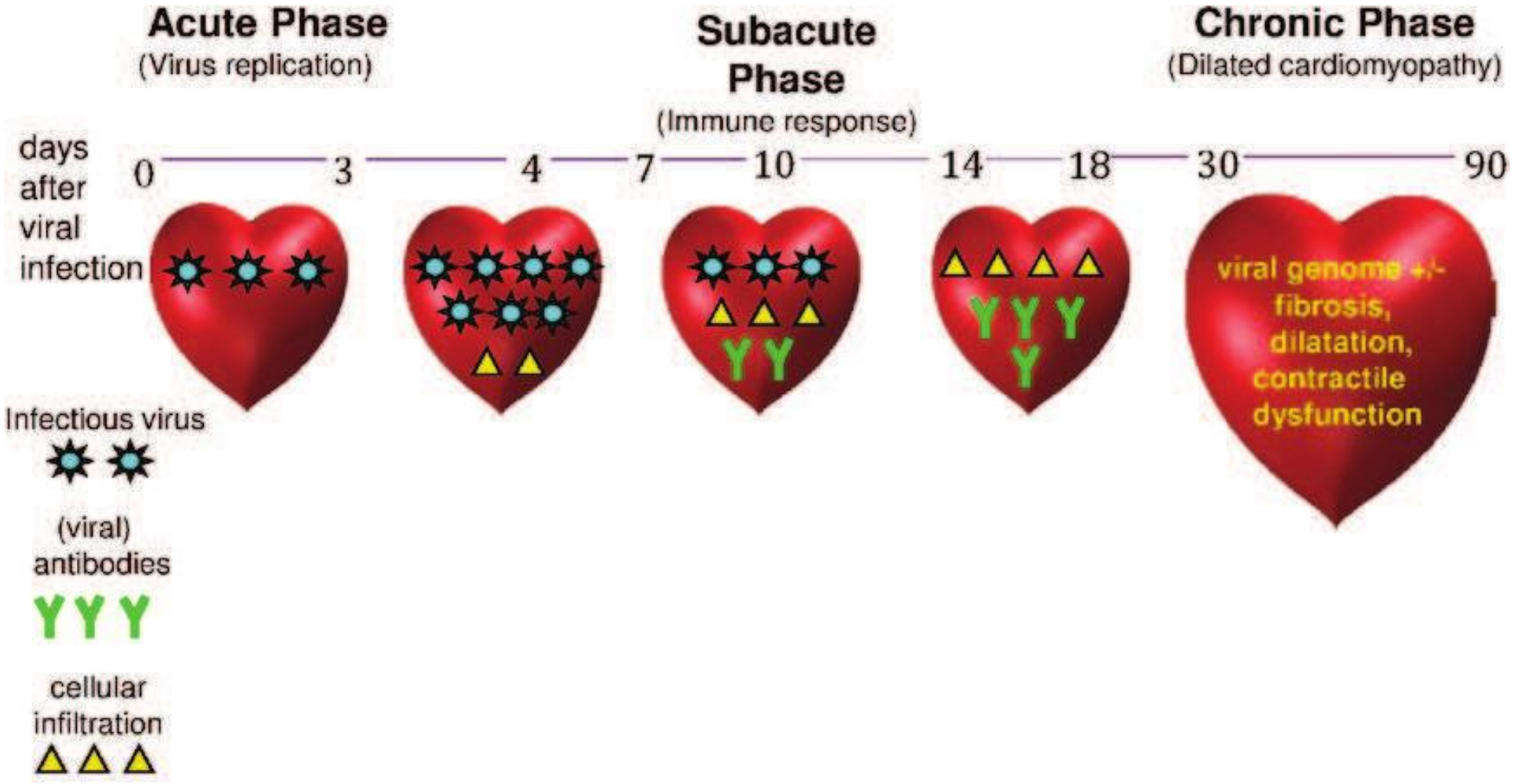
- Viral trigger ***plus*** immune response





Kindermann et al, JACC 2012
Shi et al, JACC 2009
Noutsias et al, Circulation 2001

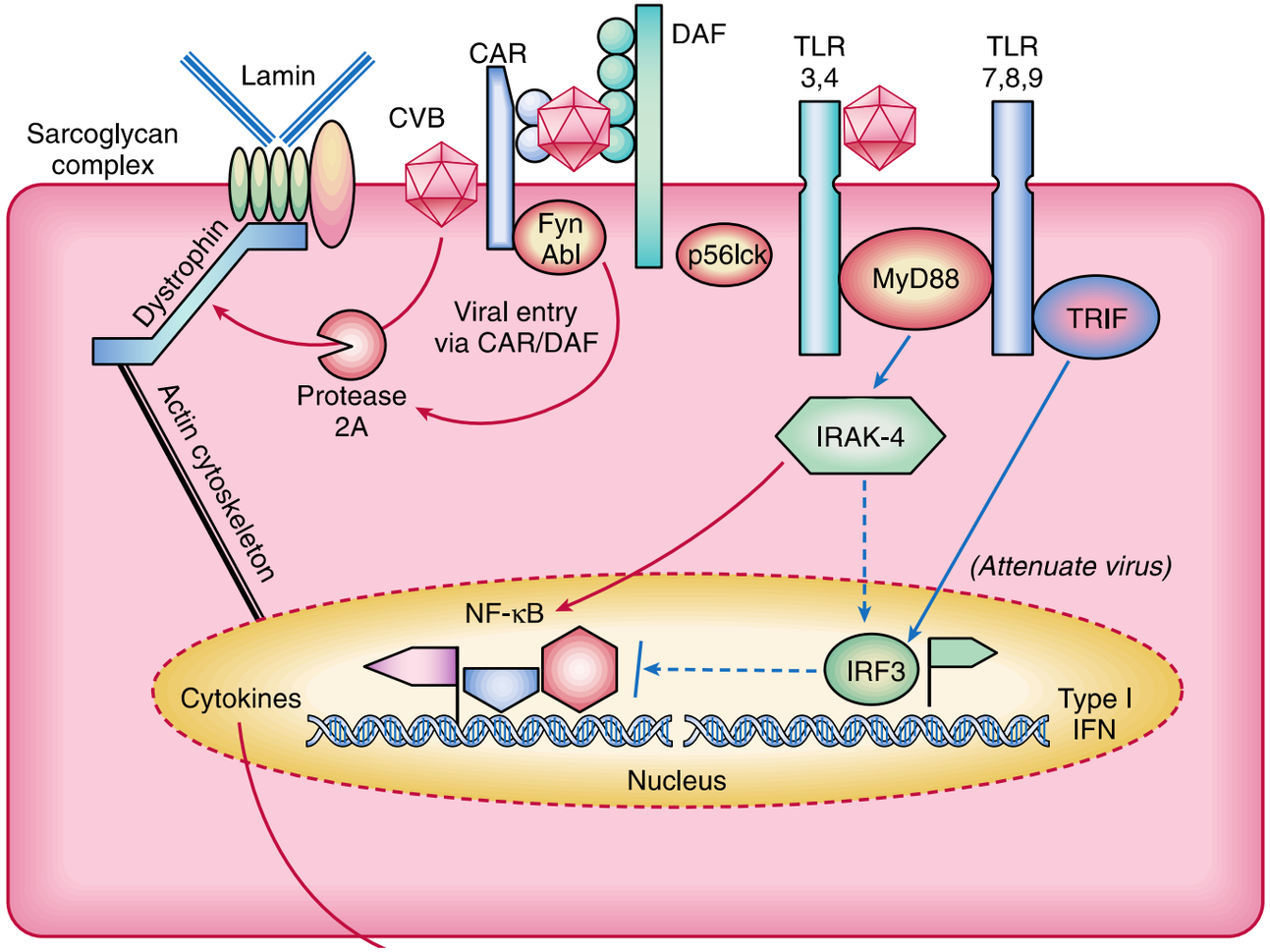
Pathophysiology: key points



Kindermann et al, JACC 2012

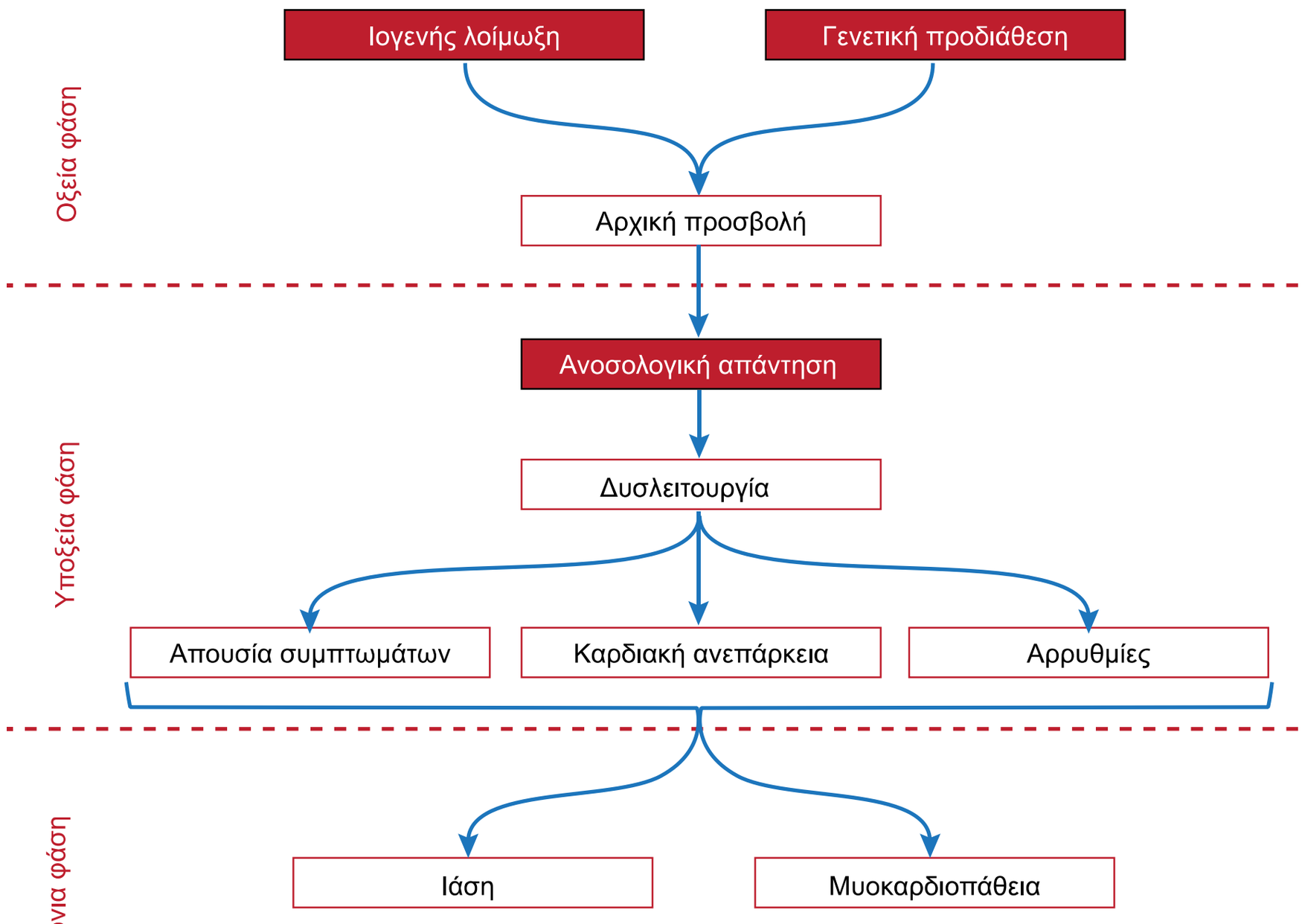
Pathophysiology: key points

- Human cardiomyocyte CAR (coxsackie and adenovirus receptor) expression may be a **predisposing factor** for facilitating viral myocarditis



Shi et al, J Am Coll Cardiol 2009
Noutsias et al, Circulation 2001

Pathophysiology: key points



Farmakis, HJC 2012

Diagnosis: Key points

≥1 clinical presentations
and
≥1 diagnostic criteria
or
≥2 diagnostic criteria

Table 4 Diagnostic criteria for clinically suspected myocarditis

Clinical presentations[a]

- Acute chest pain, pericarditic, or pseudo-ischaemic
- New-onset (days up to 3 months) or worsening of: dyspnoea at rest or exercise, and/or fatigue, with or without left and/or right heart failure signs
- Subacute/chronic (>3 months) or worsening of: dyspnoea at rest or exercise, and/or fatigue, with or without left and/or right heart failure signs
- Palpitation, and/or unexplained arrhythmia symptoms and/or syncope, and/or aborted sudden cardiac death
- Unexplained cardiogenic shock

Diagnostic criteria

I. ECG/Holter/stress test features

- Newly abnormal 12 lead ECG and/or Holter and/or stress testing, any of the following: I to III degree atrioventricular block, or bundle branch block, ST/T wave change (ST elevation or non ST elevation, T wave inversion), sinus arrest, ventricular tachycardia or fibrillation and asystole, atrial fibrillation, reduced R wave height, intraventricular conduction delay (widened QRS complex), abnormal Q waves, low voltage, frequent premature beats, supraventricular tachycardia

II. Myocardiocytolysis markers

- Elevated TnT/TnI

III. Functional and structural abnormalities on cardiac imaging (echo/angio/CMR)

- New, otherwise unexplained LV and/or RV structure and function abnormality (including incidental finding in apparently asymptomatic subjects): regional wall motion or global systolic or diastolic function abnormality, with or without ventricular dilatation, with or without increased wall thickness, with or without pericardial effusion, with or without endocavitary thrombi

IV. Tissue characterization by CMR

- Oedema and/or LGE of classical myocarditic pattern (see text)

Caforio et al, Eur Heart J 2013

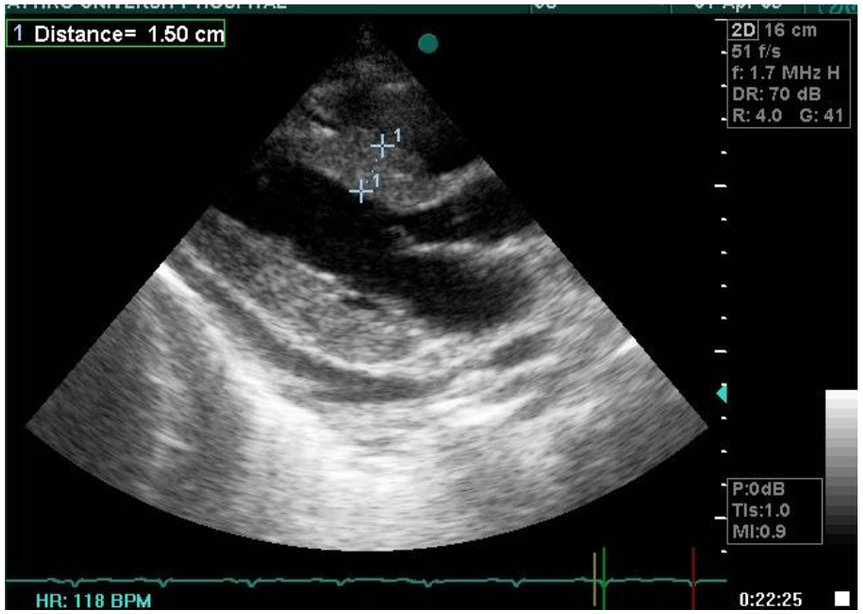
Diagnosis: Key points II

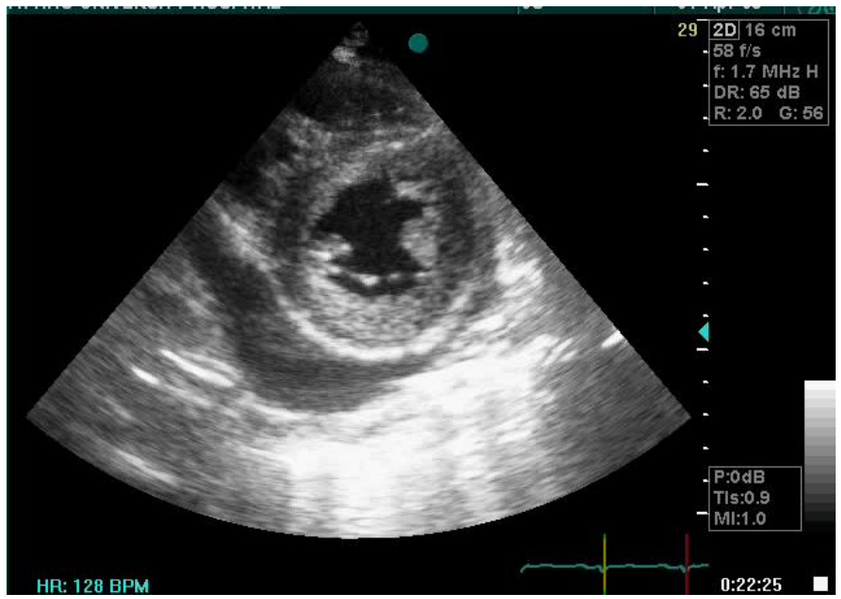
Echocardiography:

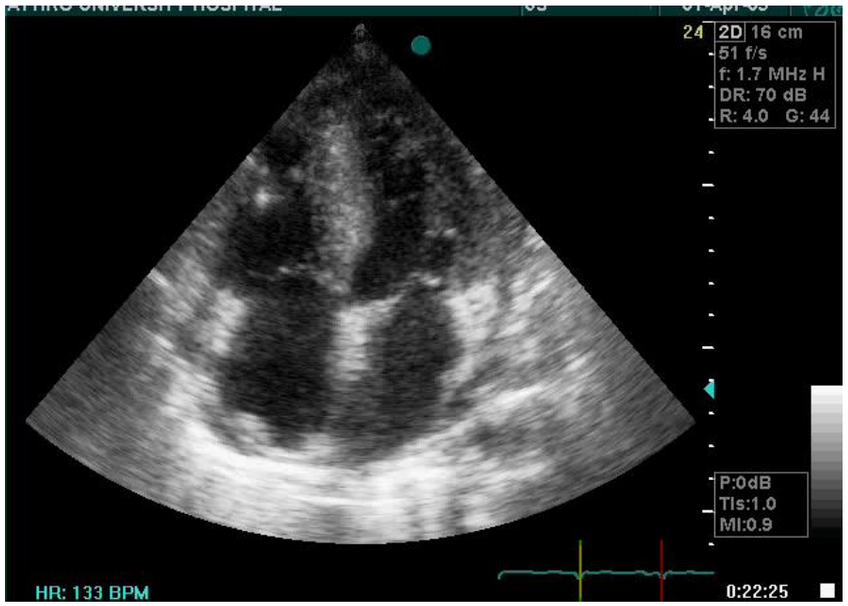
- assess geometry and function
- rule-out other causes of HF
- useful tips
  - **–fulminant: no LV dilatation & increased wall thickness due to edema vs**
  - **–acute: LV dilatation & normal wall thickness**

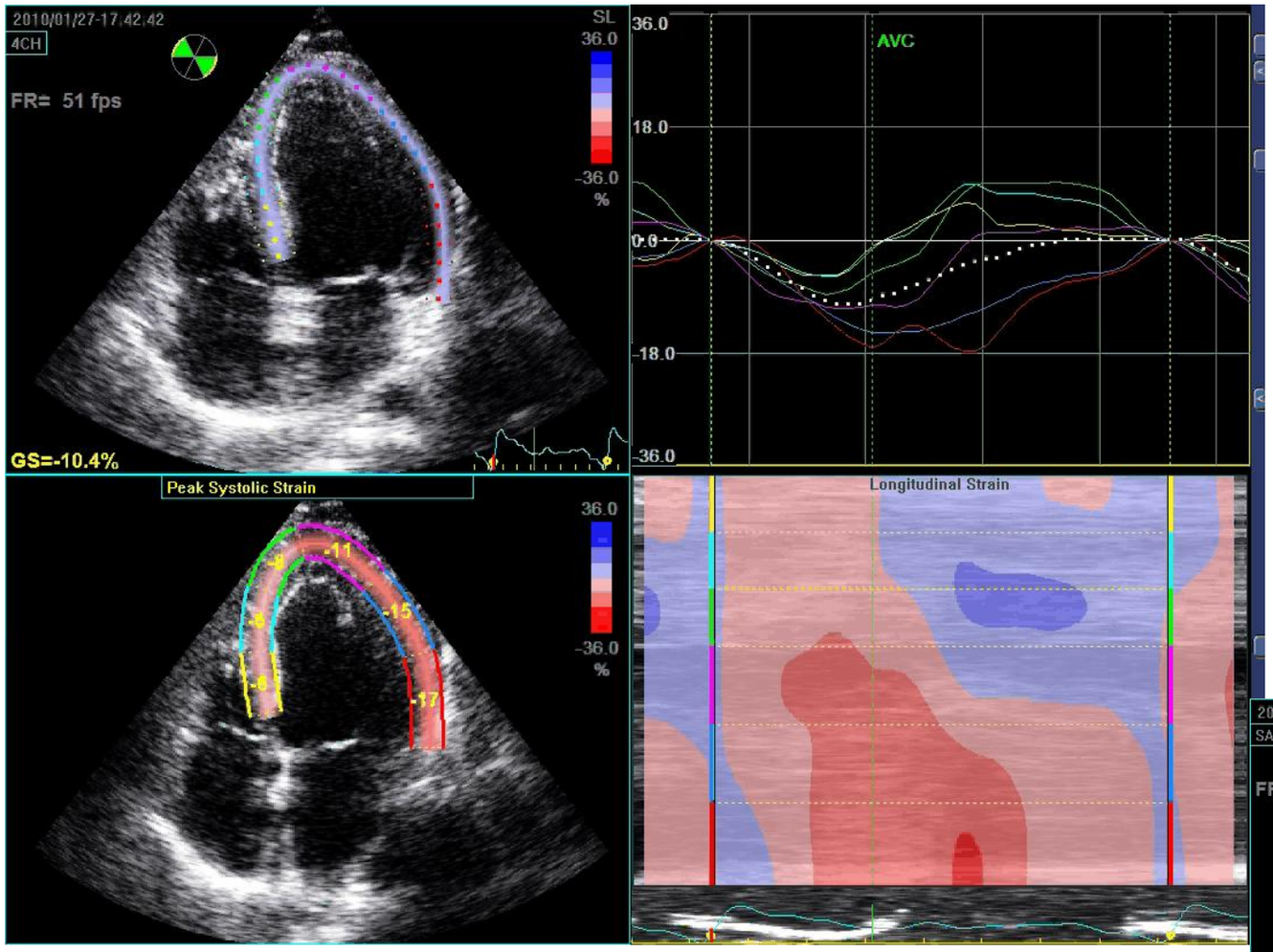
Ukena et al, Eur J Heart Fail 2011

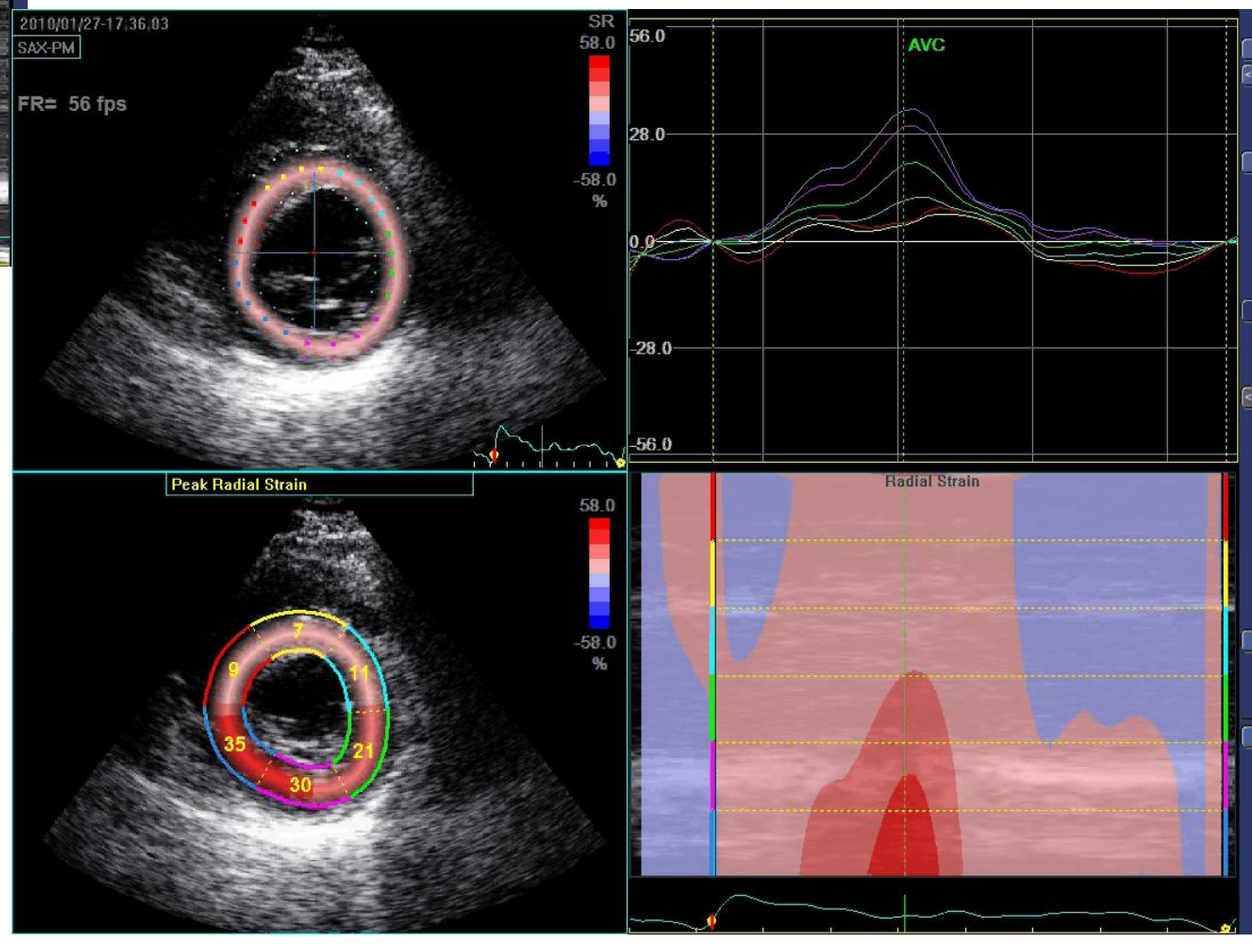
Fulminant myocarditis











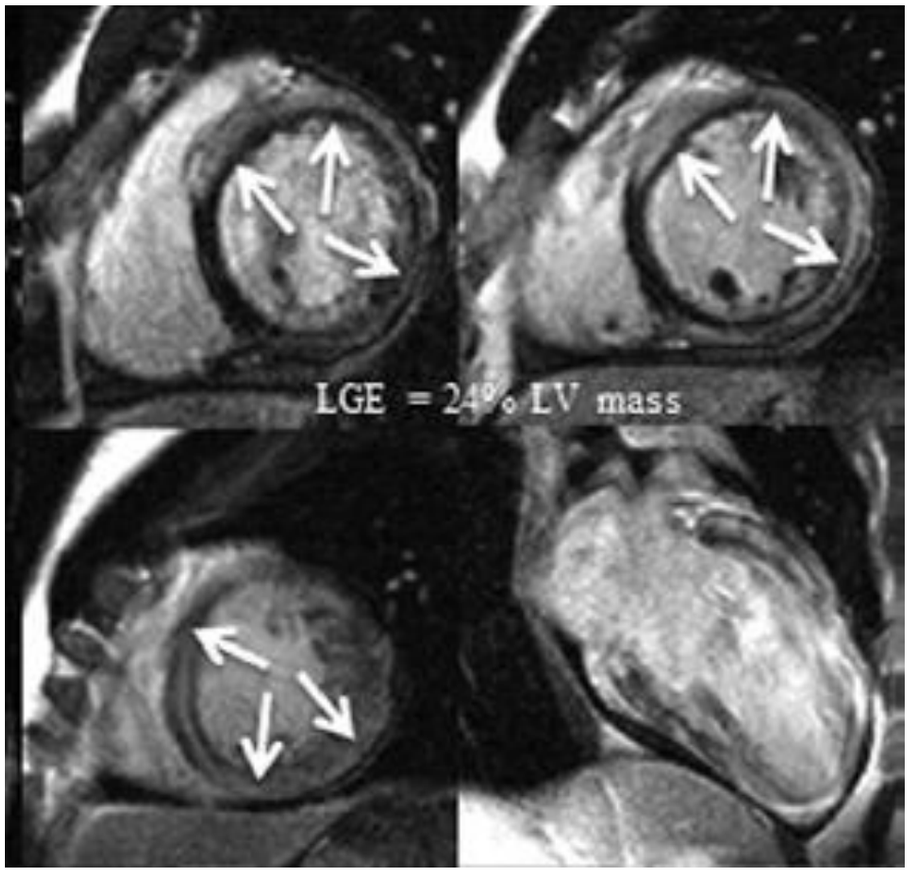
Longitudinal strain had the greatest area under the curve, 0.93 (optimal cutoff value, -15.1 %; sensitivity, 78 %; specificity, 93 %).

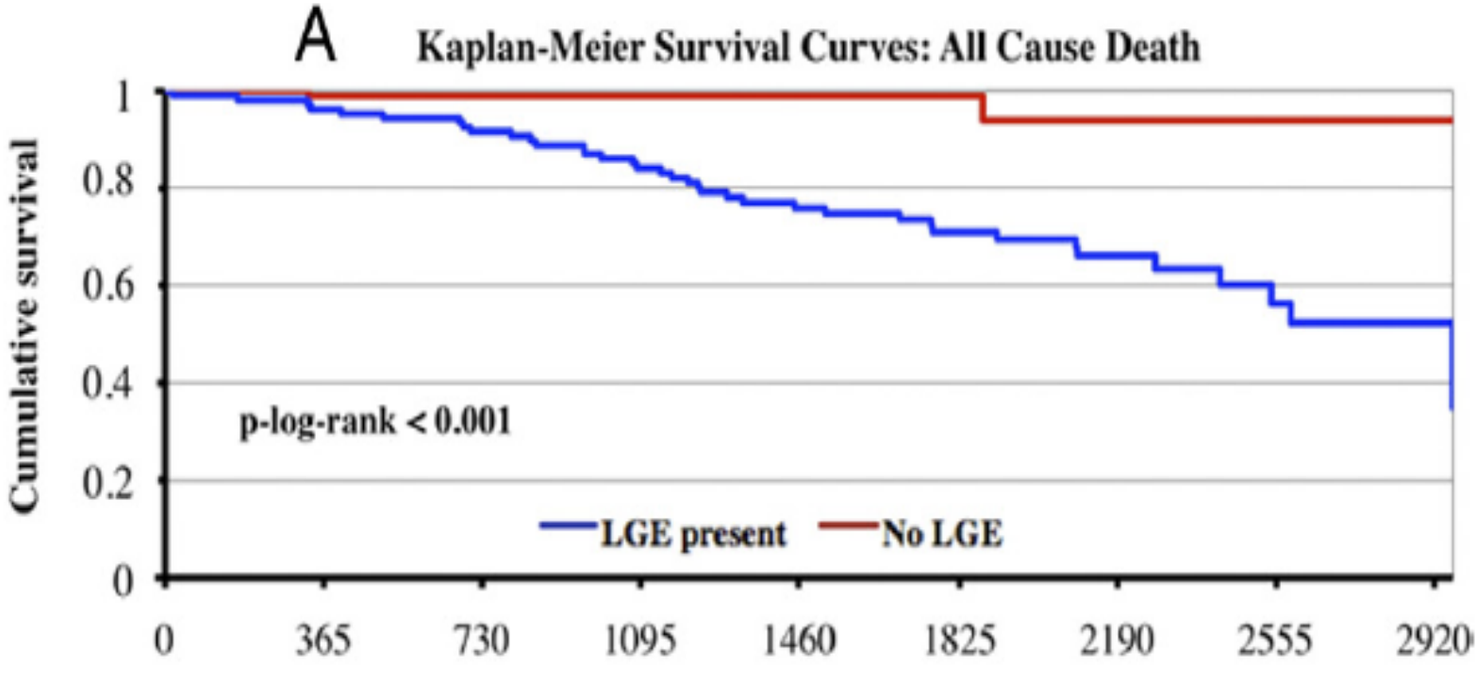
Per 1% reduction of GLS HR (95 % CIs) were 1.26 (1.10-1.47) for events

Int J Cardiovasc Imaging. 2013 Feb;29(2):275-84

CMR

- Highly in agreement with biopsy
- **LGE**, the best predictor of mortality in biopsy-proven viral myocarditis

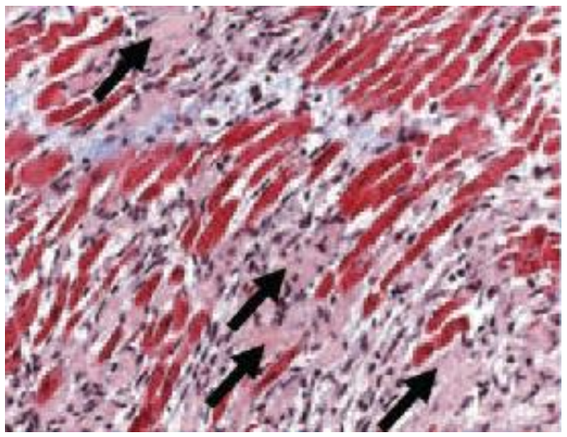


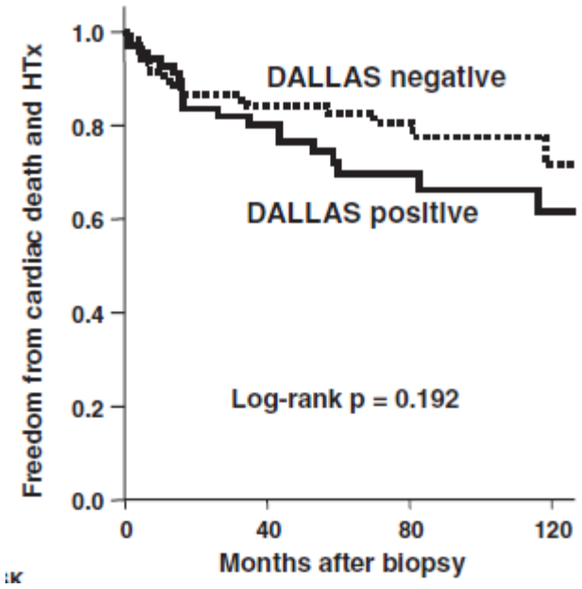


Grun et al, JACC 2012

Endomyocardial biopsy

- **Dallas criteria** alone have low diagnostic/prognostic value (variation in interpretation, inability to detect noncellulary mediated inflammation)

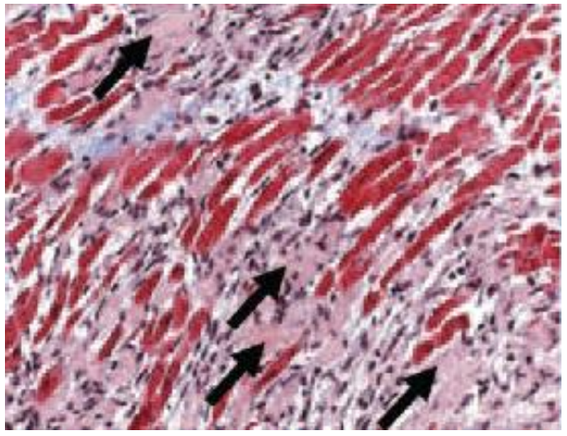


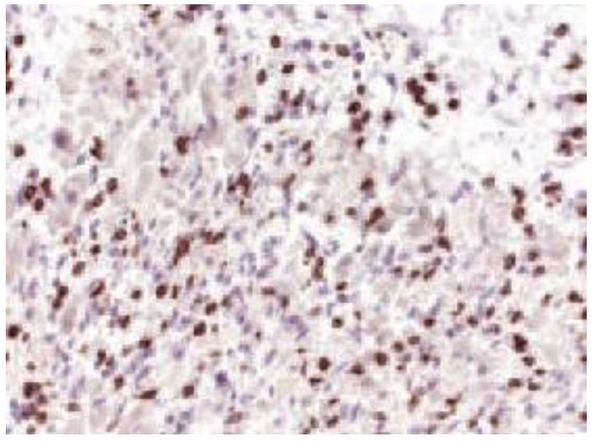


Kindermann et al, Circulation 2008

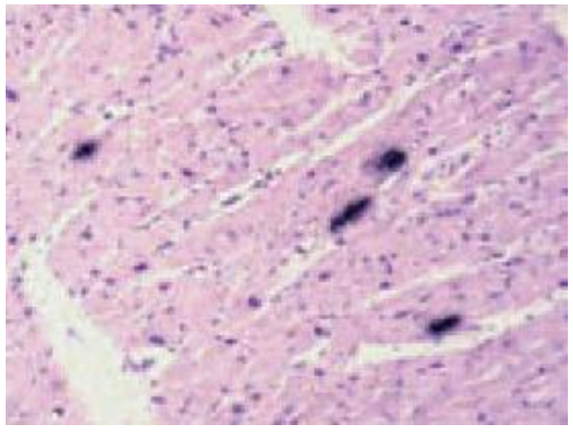
Endomyocardial biopsy

- **Immunohistology** to detect inflammation and **molecular studies** to detect viral genome enhance diagnostic & prognostic value and may guide therapy

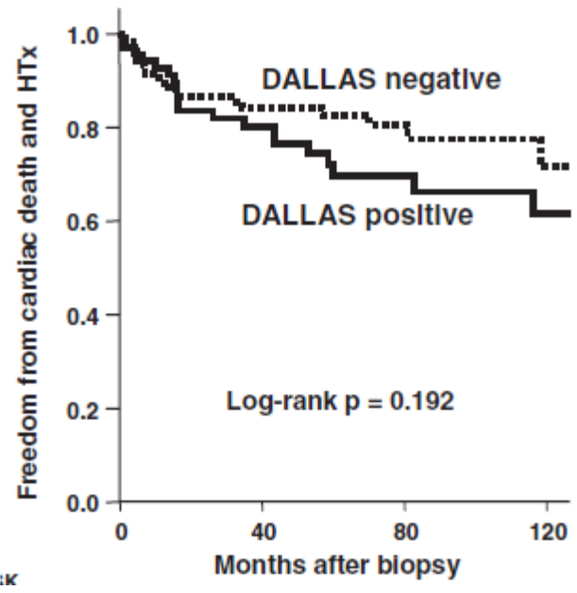


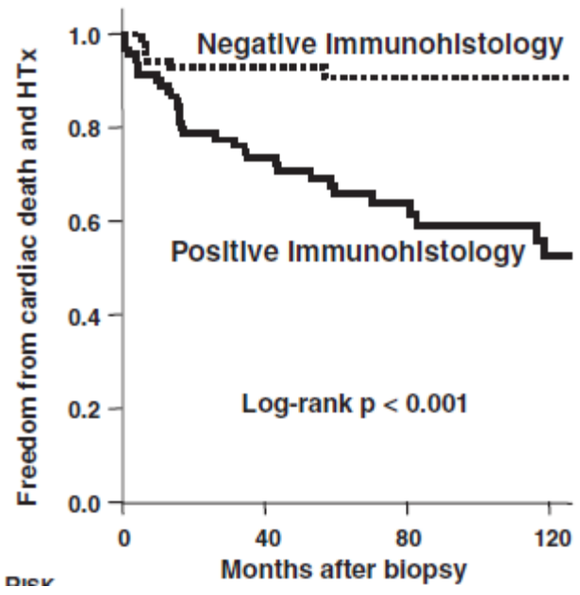


CD3+ T lymphocytes



In situ hybridization (enterovirus genome)





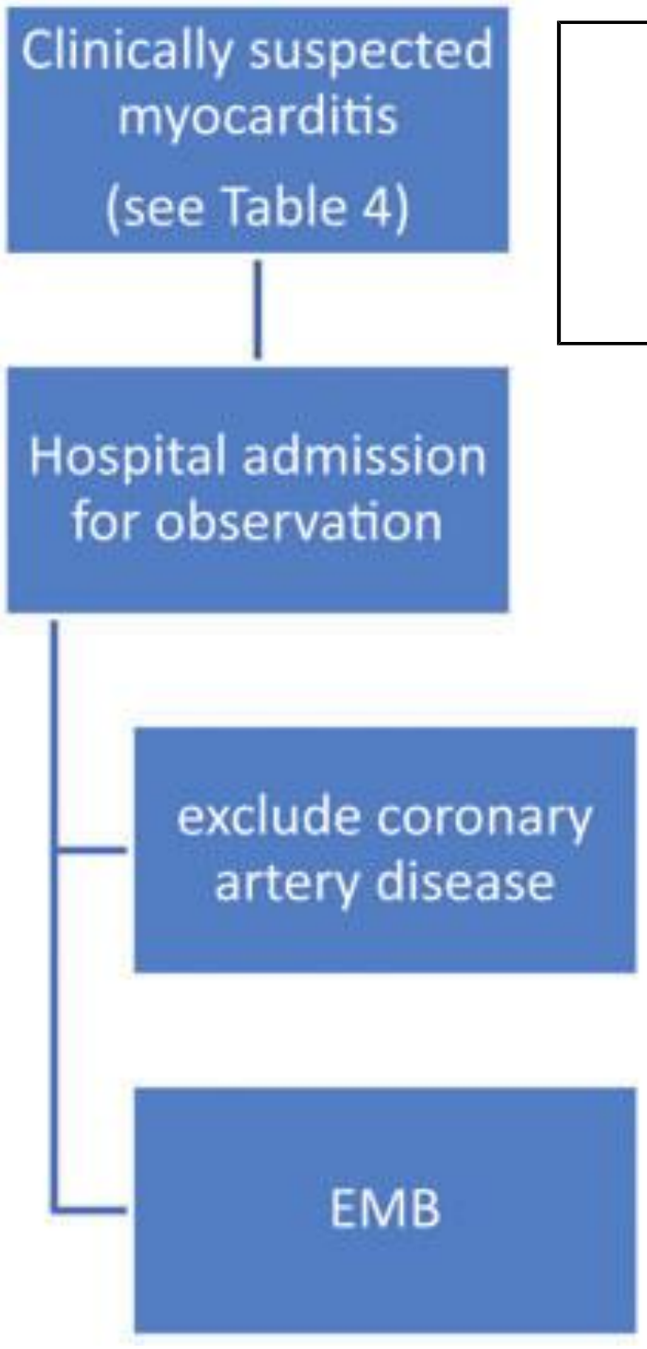
Kindermann et al, Circulation 2008

Endomyocardial biopsy

ESC Guidelines

- New-onset HF <2w and hemodynamic compromise (IB)
- New-onset HF 2w-3m, dilated LV and new arrhythmias or no response to therapy (IB)

Endomyocardial biopsy



Recommendation

10. All patients with clinically suspected myocarditis should be considered for selective coronary angiography and EMB.

Recommendations

11. Tissue obtained from EMB should be analysed using histology, immunohistochemistry, and viral PCR (on heart tissue and a blood sample).

13. Endomyocardial biopsy may be repeated if necessary to monitor response to aetiology-directed therapy, or if a sampling error is suspected in a patient with unexplained progression of heart failure.

Caforio et al, Eur Heart J 2013

Therapy: key points

- No standard therapy for the majority of cases
- General heart failure therapy & physical rest
- Mechanical support & transplantation for severe HF
- Immunosuppression for specific types
- Immune/anti-viral therapies not yet established

Therapy: key points

Αγωγή οξείας/υποξείας φάσης

Κλινική αξιολόγηση
ΗΚΓ
Τροπονίνη, BNP
Ηχωκαρδιογραφία
CMR

Διουρητικά, αγγειοδιασταλτικά

Ινότροπα, Ενδοαορτική αντλία

Συσκευές υποβοήθησης (LVAD, BiVAD, ECMO)

Μεταμόσχευση

Αντιϊκή αγωγή (IFN-B)

Ενδομυοκαρδιακή βιοψία (≤2 εβδομάδες)

Ανοσοκαταστολή (κορτικοστεροειδή, αζαθειοπρίνη, κυκλοσπορίνη κλπ)

Χρόνια αγωγή

Κλινική αξιολόγηση
ΗΚΓ
Τροπονίνη, BNP
Ηχωκαρδιογραφία
CMR

ΑΜΕΑ, β-αναστολείς (± διουρητικά)

Ανταγωνιστές αλδοστερόνης

Ιβαμπραδίνη

CRT, ICD, CRT-D

Συσκευές υποβοήθησης, Μεταμόσχευση

Ενδομυοκαρδιακή βιοψία (≤3 μήνες)

Farmakis, HJC 2012

Heart failure therapy

- Standard HF regimens (RAASi, β-blockers, MRA) according to current guidelines

- Data on myocarditis mainly from animal models

- **Duration of HF therapy?**

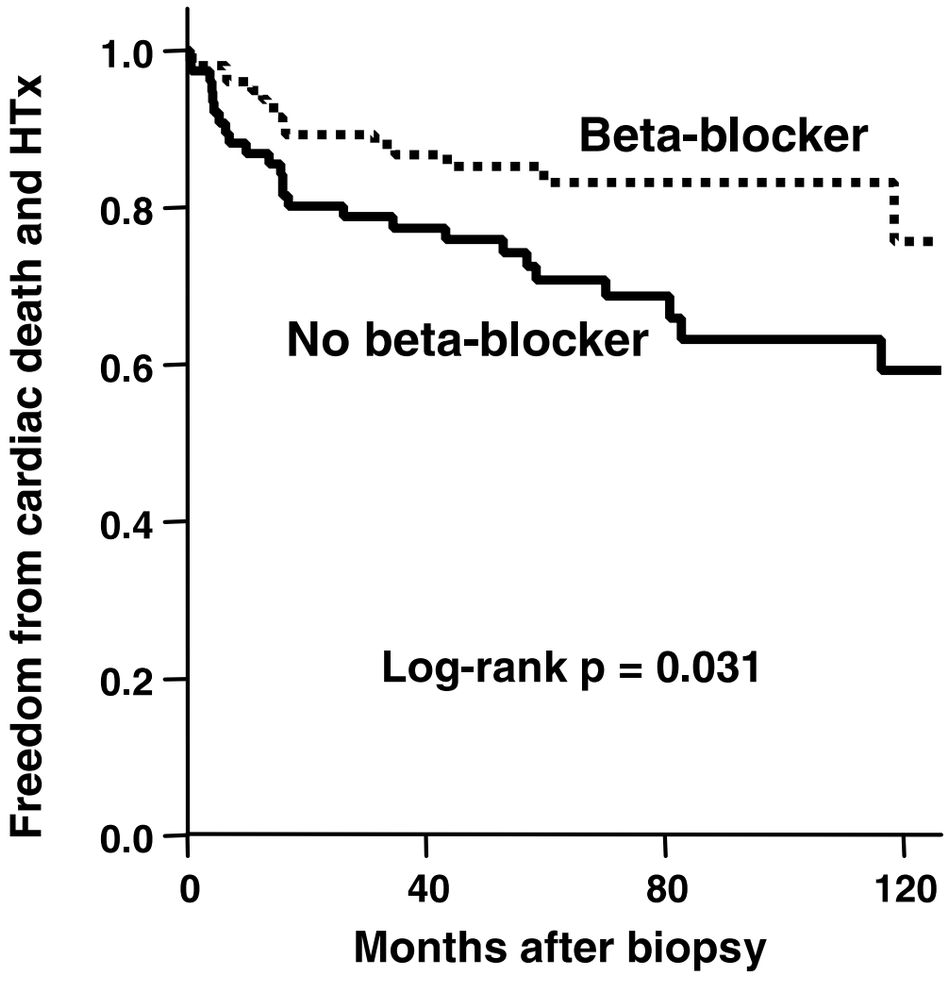
ACEi & ARBs

- **Captopril, losartan** and **olmesartan** reduced inflammation, necrosis, fibrosis and LV remodelling in animal models

Seko, Clin Sci (Lond) 2006
Bahk et al, Int J Cardiol 2008
Sukumaran et al, Exp Biol Med (Maywood) 2010

β-blockers

- Lack of β-blocker treatment is associated with poor outcome (along with NYHA and immunohistology)



Kindermann et al, Circulation 2008

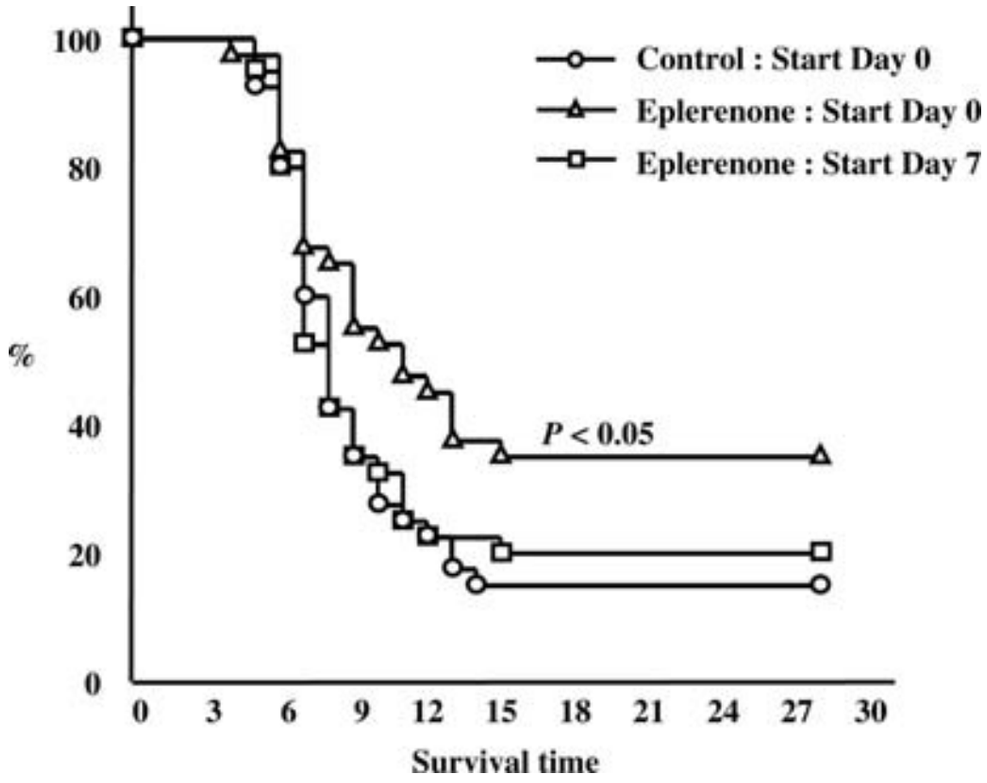
β-blockers

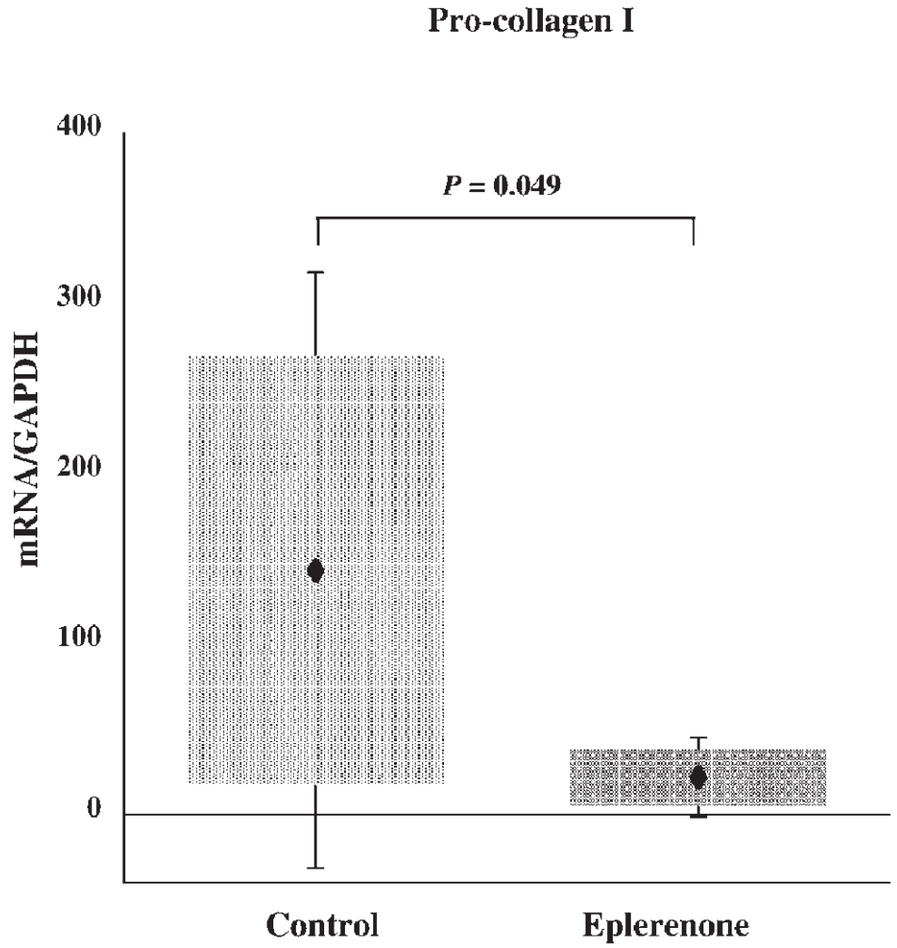
- Should be avoided in acute severe HF
- Type of β-blocker:
  - **Carvedilol** was cardioprotective in rats (anti-inflammatory properties) but metoprolol and propranolol were not
  - **Metoprolol** increased inflammation, necrosis and mortality in mice

Yuan et al, Am J Physiol Heart Circ Physiol 2004

Aldosterone antagonists

- **Eplerenone** improved survival, reduced inflammation and suppressed expression of genes related to fibrosis and remodeling in rats





Xiao et al, Eur J Heart Fail 2009

Physical activity

- may worsen outcome in acute/subacute phase but beneficial in chronic HF
- should be restricted during the acute phase and for **at least 6 months** in athletes and non-athletes
- return to training and competition if LV function and cardiac dimensions return to normal and no clinically relevant arrhythmias exist

36th Bethesda Task Force, Maron et al, JACC 2005
ESC WP Position Paper, Caforio et al, Eur Heart J 2013

Device therapy

- **Temporary pacemaker** in symptomatic AV block II or III (often in Chaga's & Lyme diseases)

- **ICD** after VF or symptomatic VT

- Avoid **premature implantation** of ICD/CRT-D as LV function may improve with medical HF therapy (but early in **giant-cell or sarcoidosis**)

- **How long can we wait for improvement?**
- **Is there a role for EPS?**

Kindermann et al, JACC 2012

Mechanical support

- VADs or ECMO
- Bridge to recovery or transplantation
- **Considered early for patients with fulminant** acute myocarditis when maximal medical therapy fails
  - Despite severe initial presentation, good overall prognosis (>60-80% survival, high rate of LV function recovery)

Mirabel et al, Crit Care Med 2011
Rajagopal et al Crit Care Med 2010

Immunomodulatory therapies

- Immunosuppression
- Immunoglobulin
- Immunoabsorption
- Antiviral

Immunomodulatory therapies

- ≥20 treatment trials with immune or anti-inflammatory therapy
- Limitations:
  - Use of Dallas criteria only - **lack of immunohistochemistry and molecular analysis** (eg, viral myocarditis treated with immunosuppression)
  - **Spontaneous remission is high** and not considered: in ESETCID study, inflammation was eradicated in 60% of immunosuppression arm and in 40% of placebo arm
  - Lack of control groups

Hufnagel et al, Herz 2000

Immunosuppression

- Prednisone, cyclosporine, azathrioprine

- Clear role in **giant cell myocarditis, sarcoidosis** and other **immune** conditions (eg. SLE, Ghurg-Strauss)

- Conflicting evidence in other forms

- **Is the a role for blind immunosuppression in non-responding cases?**
- ***Only after ruling out active infection on EMB by PCR***

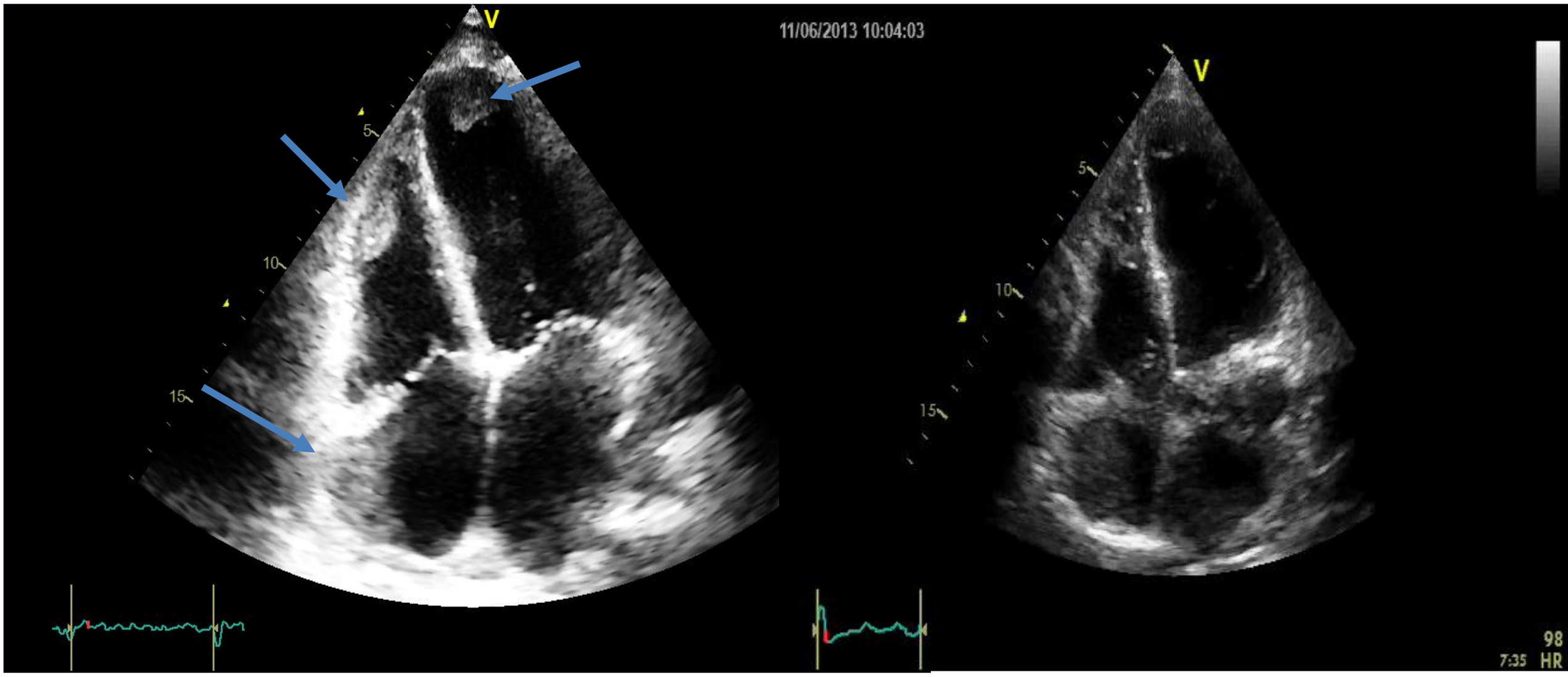
Caforio et al, Eur Heart J 2013

Immunosuppression

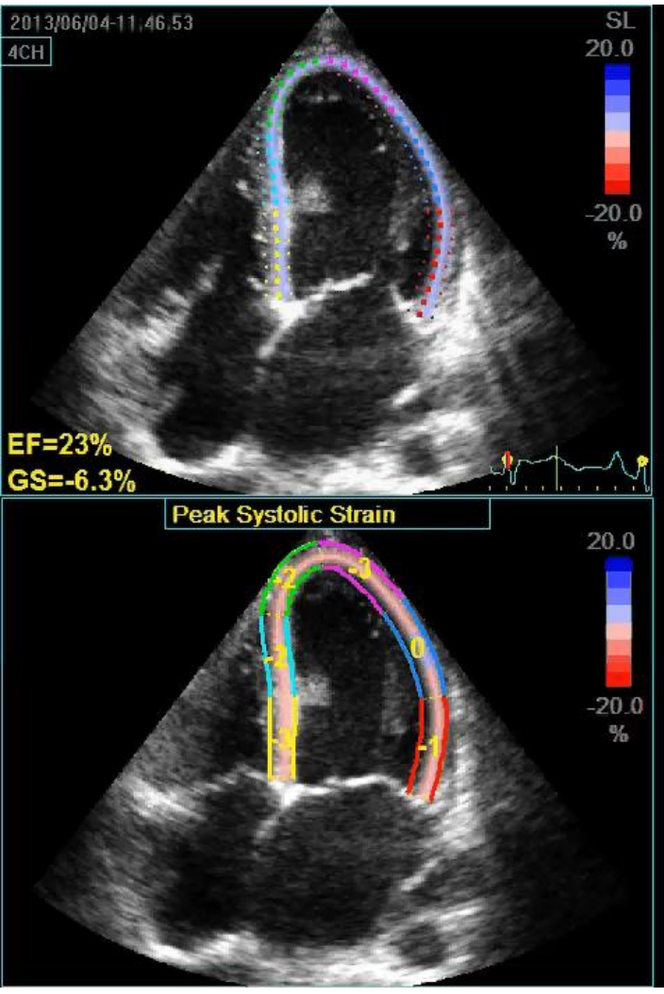
- **Giant cell myocarditis:**
  - corticosteroids, cyclosporine, azathioprine combinations
  - survival, 12 months vs 3 months if untreated
- **Sarcoidosis:**
  - high-dose corticosteroids
  - 5-year survival, 60-90%

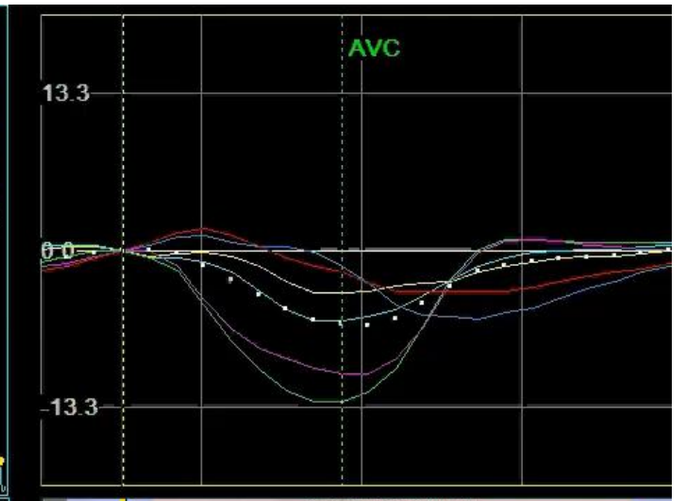
Cooper et al, Am J Cardiol 2008
Kim et al, Am Heart J 2009
Nunes et al, Semin Respir Crit Care Med 2010

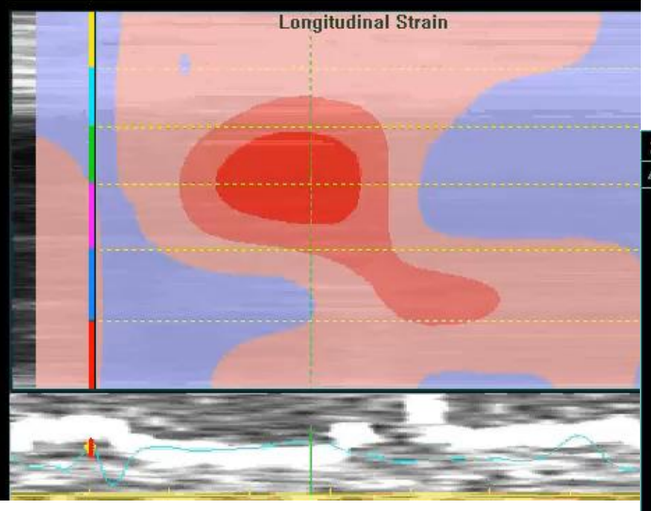
Acute heart failure in Churg Strauss vasculitis



Ikonomidis et al . J Thrombosis and Thrombolysis 2014

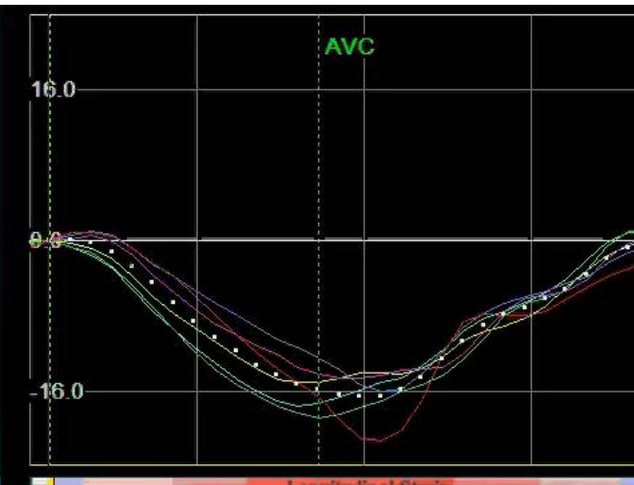


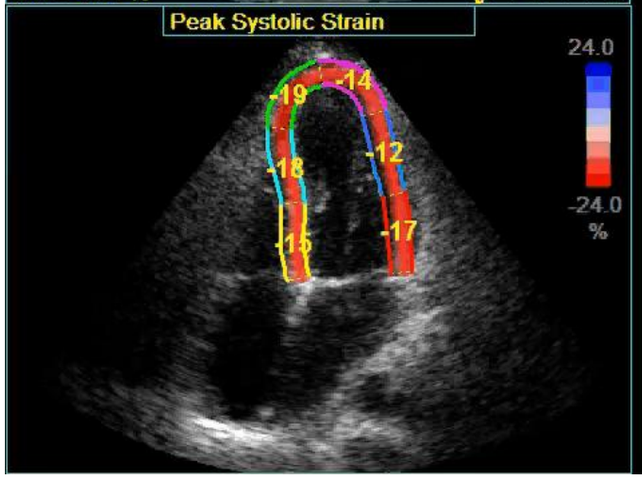


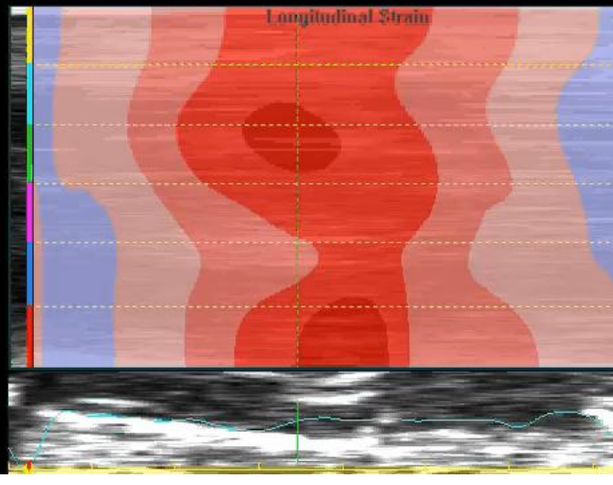


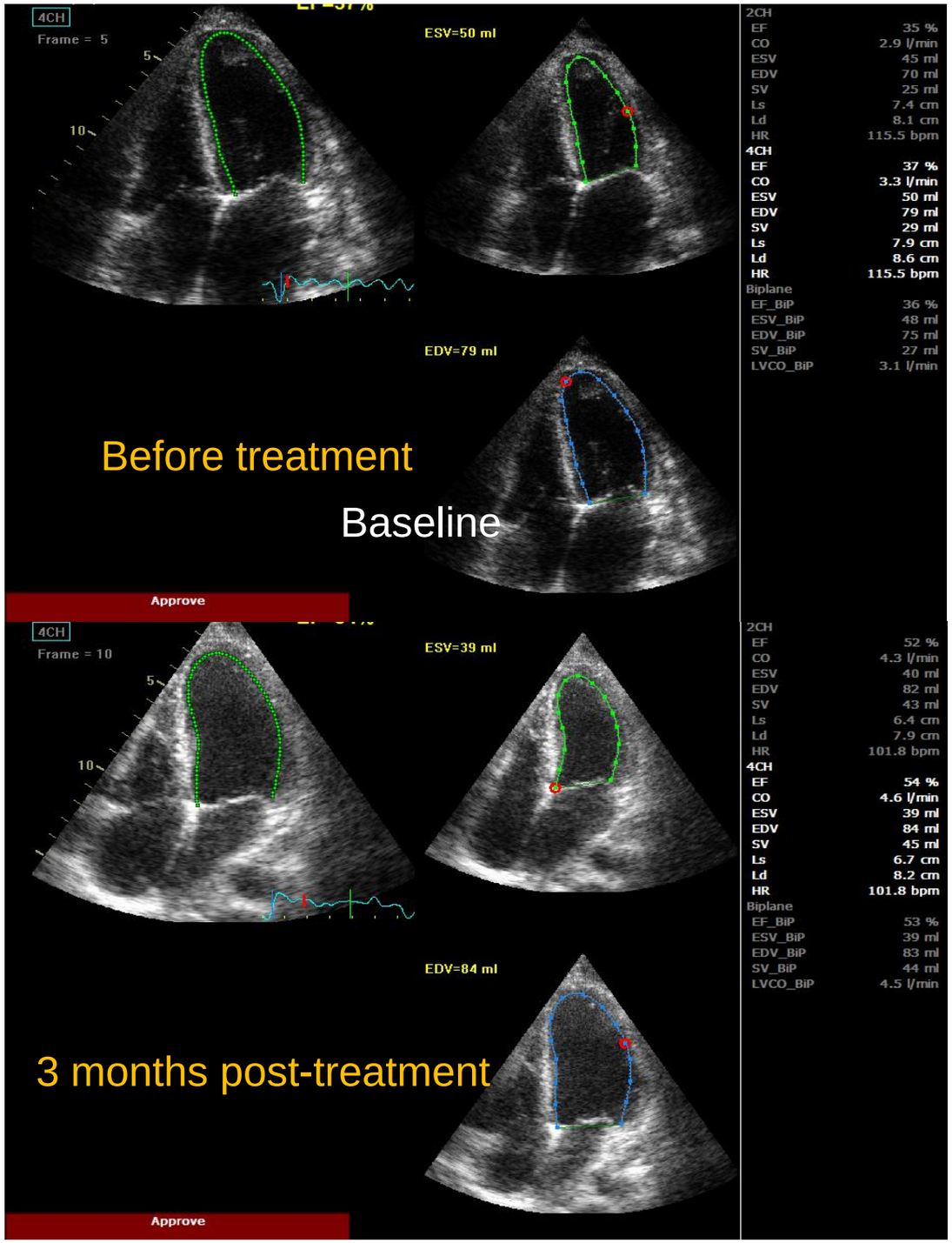
Before treatment
Reduction of strain in all segments excluding CAD

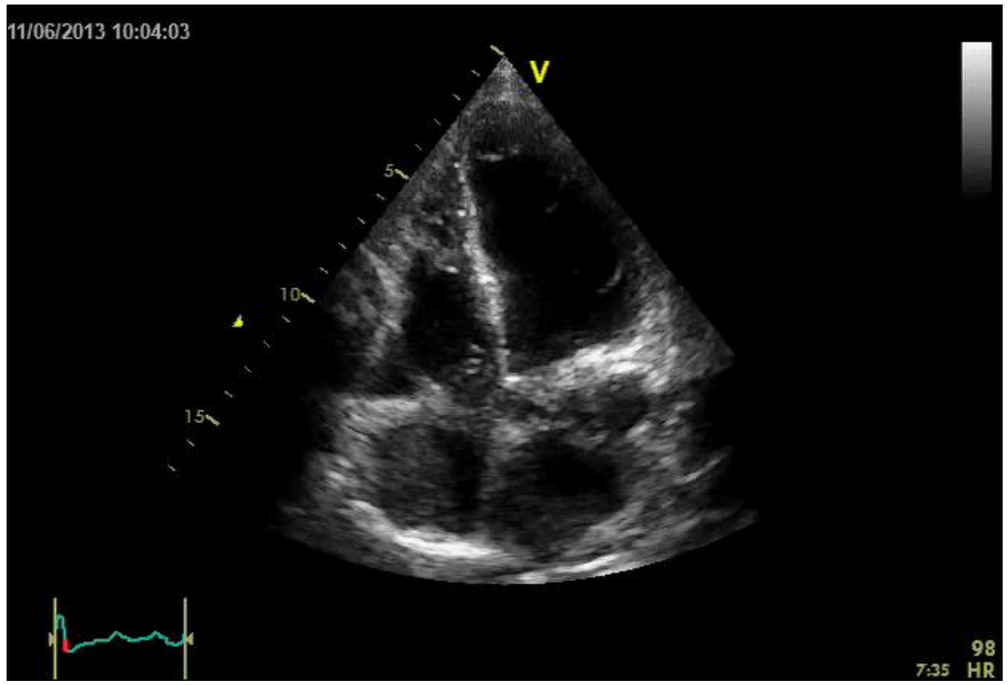
After treatment








4CH
Frame = 5
ESV=50 ml
EDV=79 ml
Before treatment
Baseline
Approve
2CH
EF 35 %
CO 2.9 l/min
ESV 45 ml
EDV 70 ml
SV 25 ml
Ls 7.4 cm
Ld 8.1 cm
HR 115.5 bpm
4CH
EF 37 %
CO 3.3 l/min
ESV 50 ml
EDV 79 ml
SV 29 ml
Ls 7.9 cm
Ld 8.6 cm
HR 115.5 bpm
Biplane
EF_BiP 36 %
ESV_BiP 48 ml
EDV_BiP 75 ml
SV_BiP 27 ml
LVCO_BiP 3.1 l/min
4CH
Frame = 10
ESV=39 ml
EDV=84 ml
3 months post-treatment
Approve
2CH
EF 52 %
CO 4.3 l/min
ESV 40 ml
EDV 82 ml
SV 43 ml
Ls 6.4 cm
Ld 7.9 cm
HR 101.8 bpm
4CH
EF 54 %
CO 4.6 l/min
ESV 39 ml
EDV 84 ml
SV 45 ml
Ls 6.7 cm
Ld 8.2 cm
HR 101.8 bpm
Biplane
EF_BiP 53 %
ESV_BiP 39 ml
EDV_BiP 83 ml
SV_BiP 44 ml
LVCO_BiP 4.5 l/min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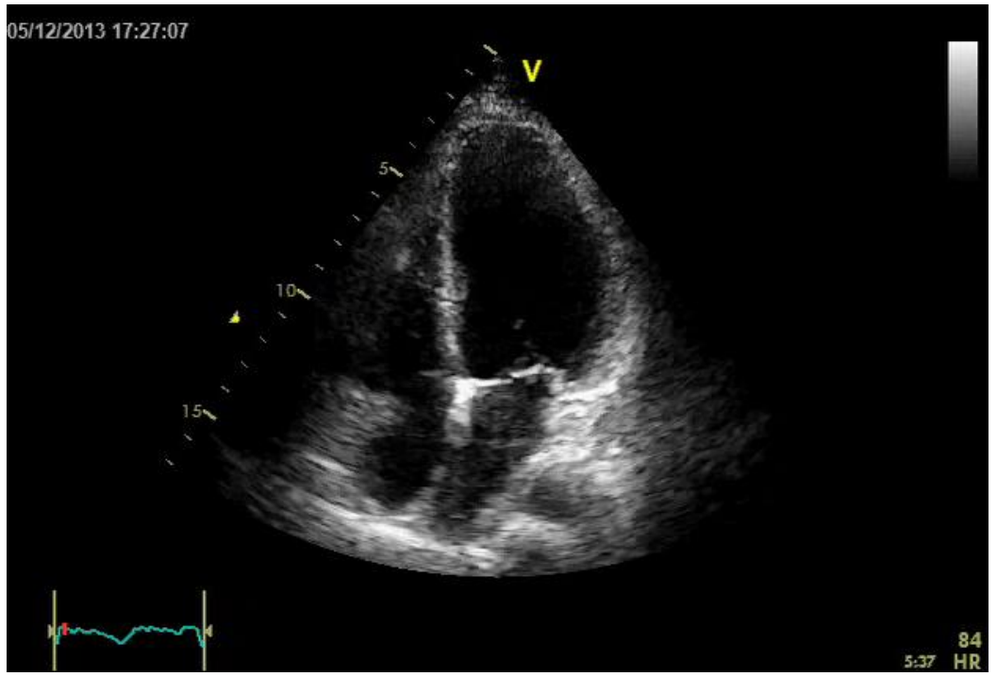
98
7:35 HR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84
5:37 HR

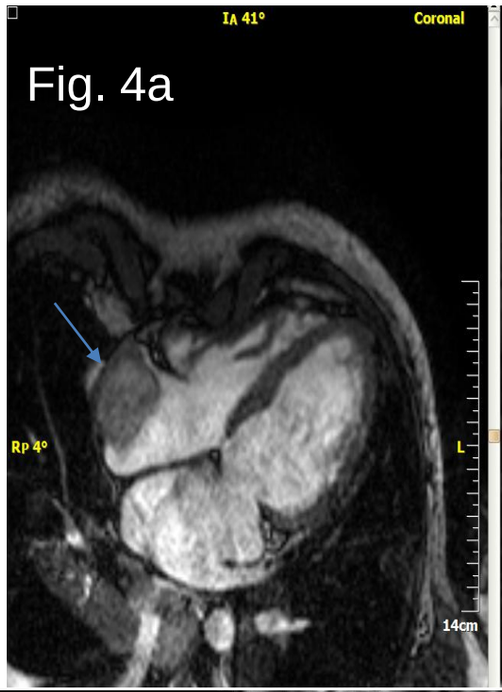
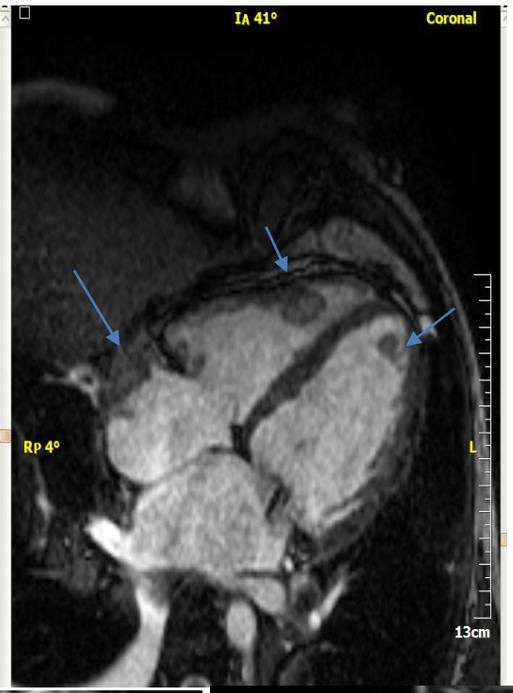

IA 41°
Coronal
RP 4°
L
14cm

Fig. 4a


IA 41°
Coronal
RP 4°
L
13cm

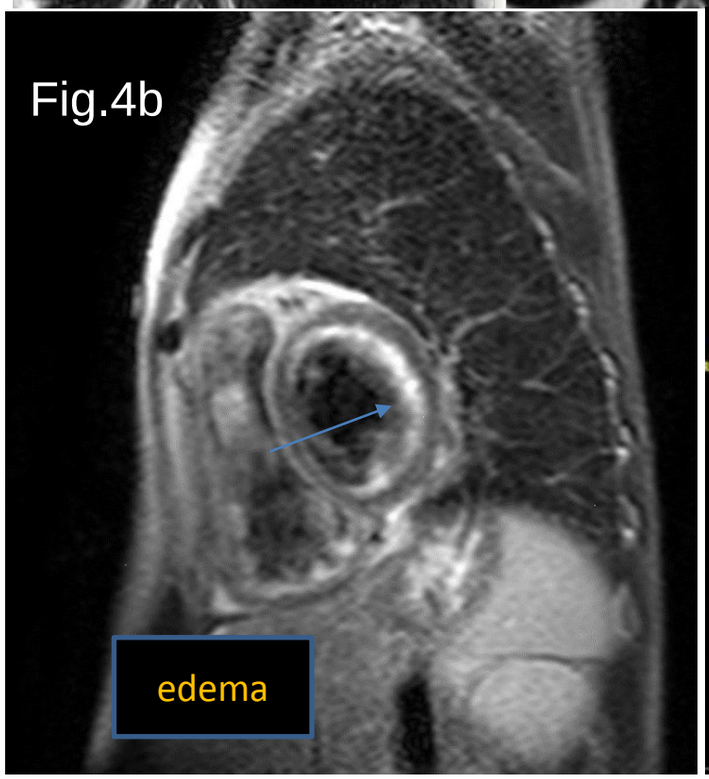


Fig.4b
edema

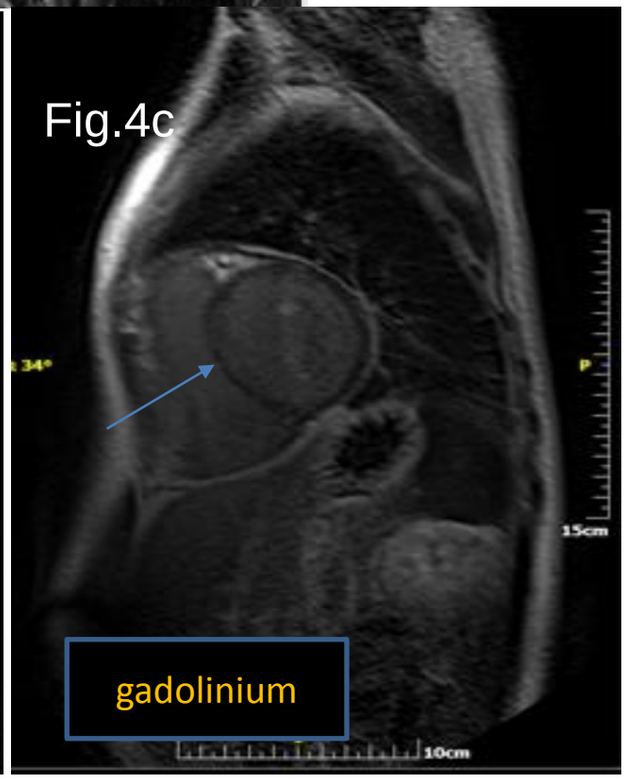


Fig.4c
gadolinium
15cm
10cm

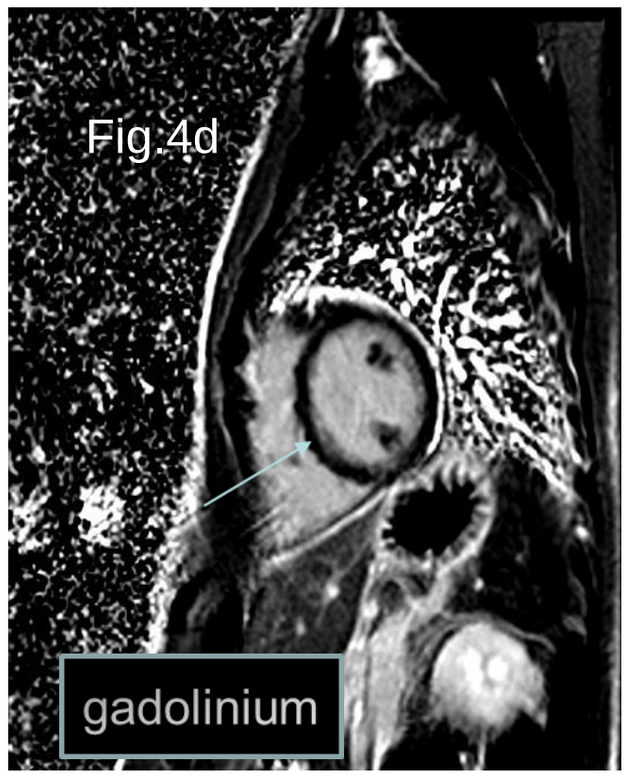
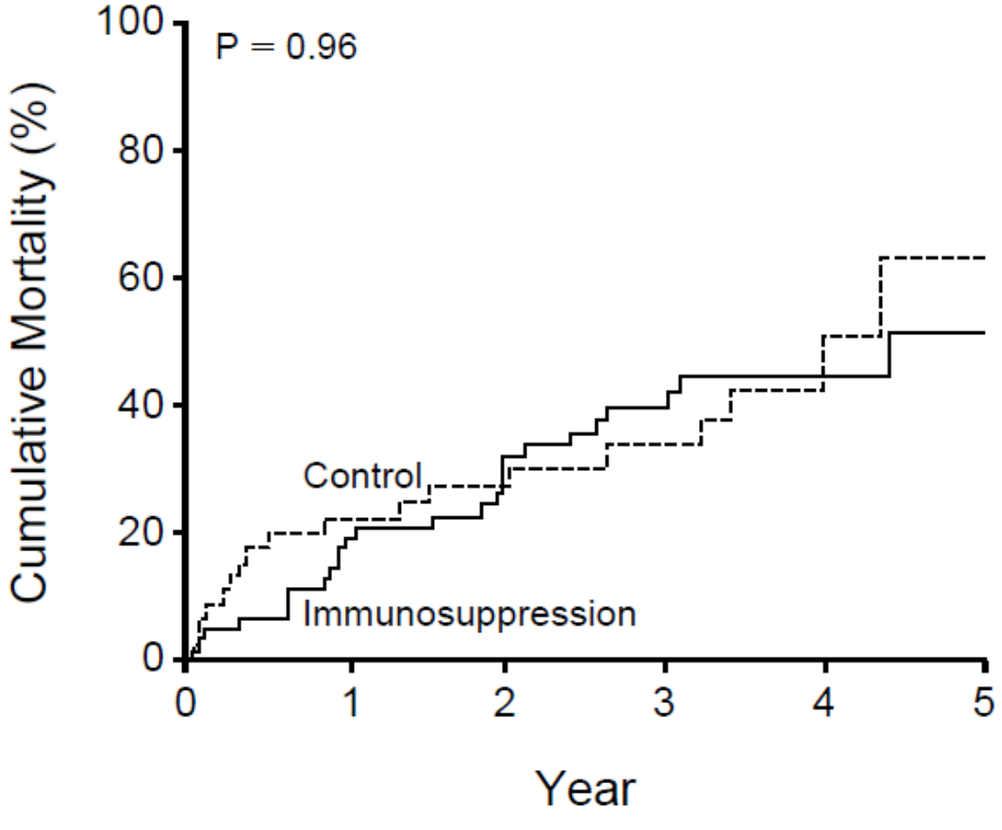
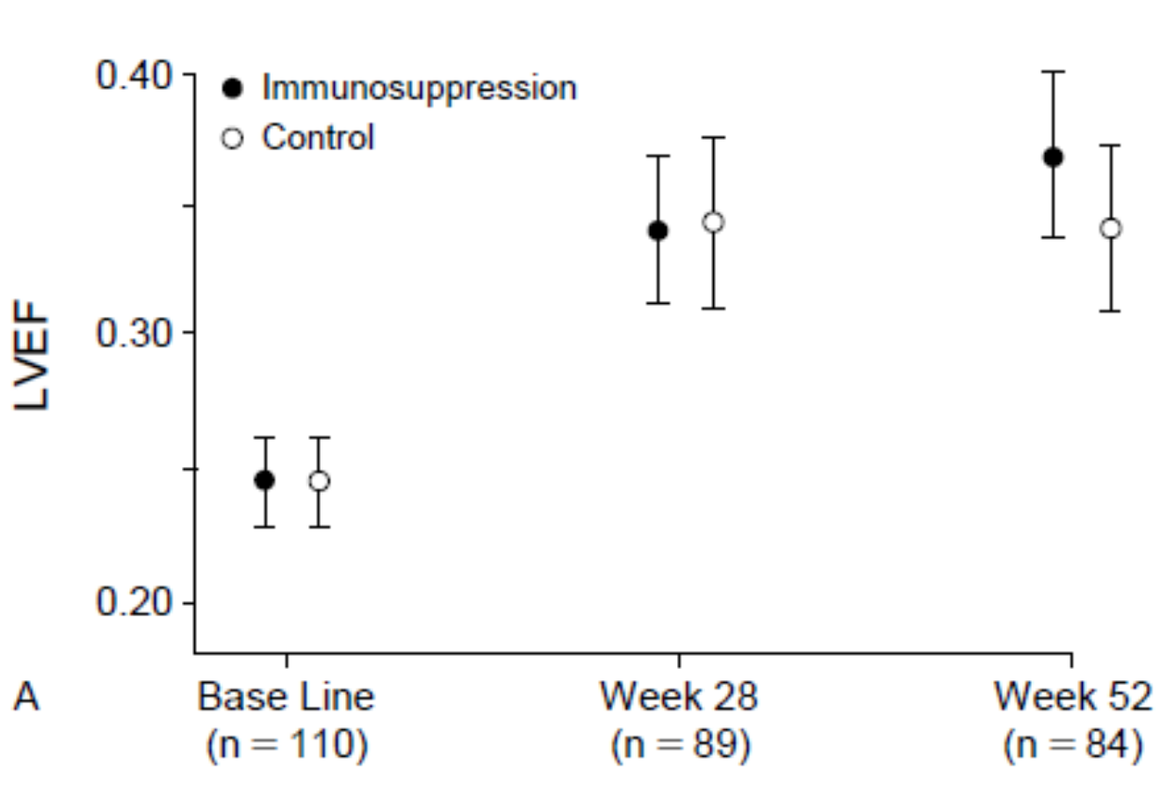


Fig.4d
gadolinium

Immunosuppression Myocarditis Treatment Trial

- N=111
- Prednisone with azathioprine or cyclosporine vs placebo for 6 months
- No difference in survival or LVEF improvement



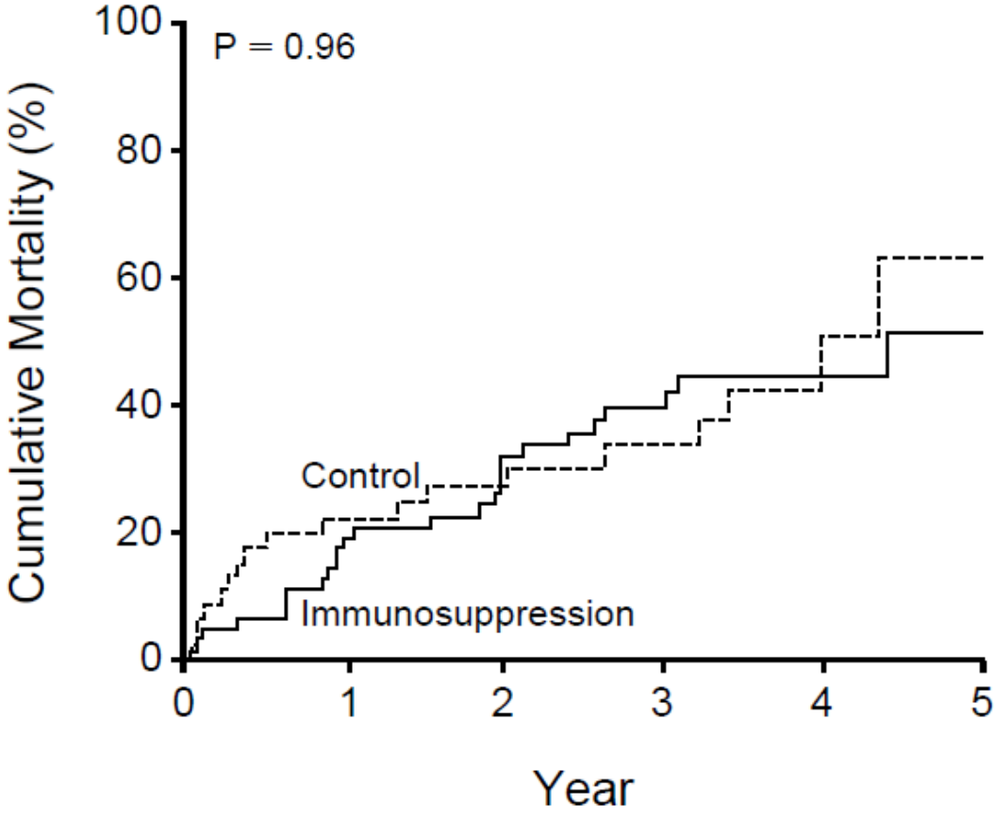


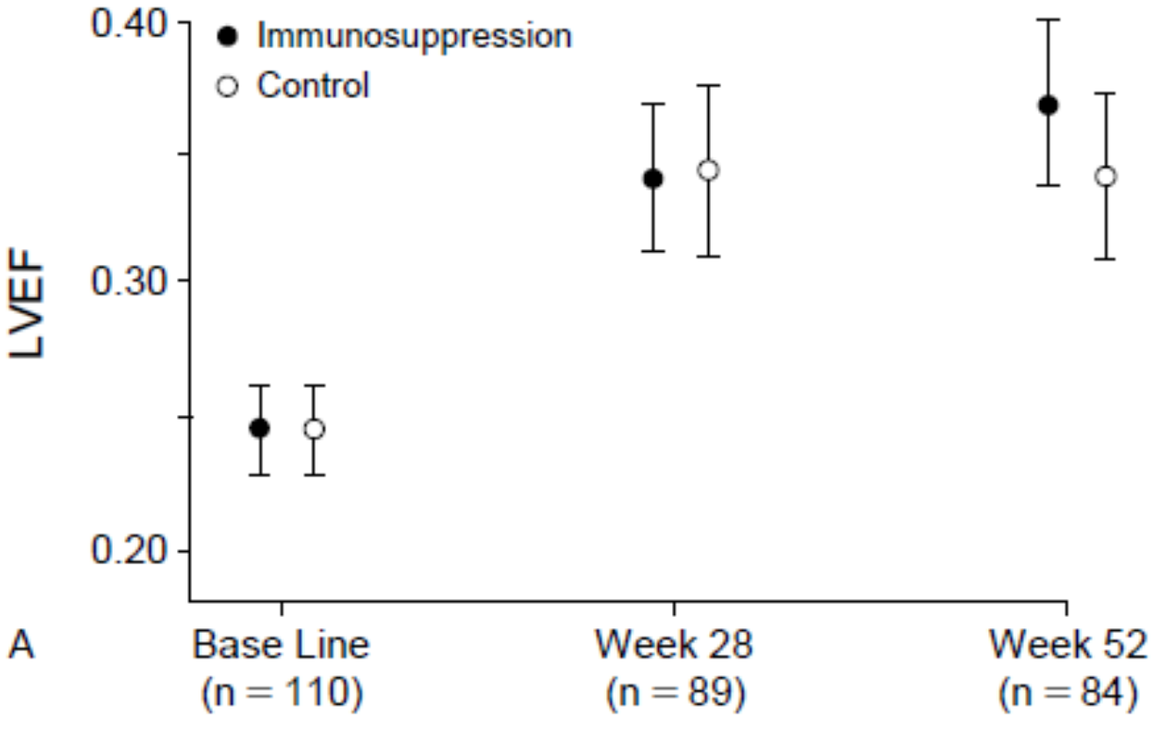
Mason et al, N Engl J Med 1995

Immunosuppression
Myocarditis Treatment Trial

- No immunohistology for the detection of inflammatory cells and no molecular biological analyses for viral exclusion

- Patients with viral infection treated with immuno- suppression

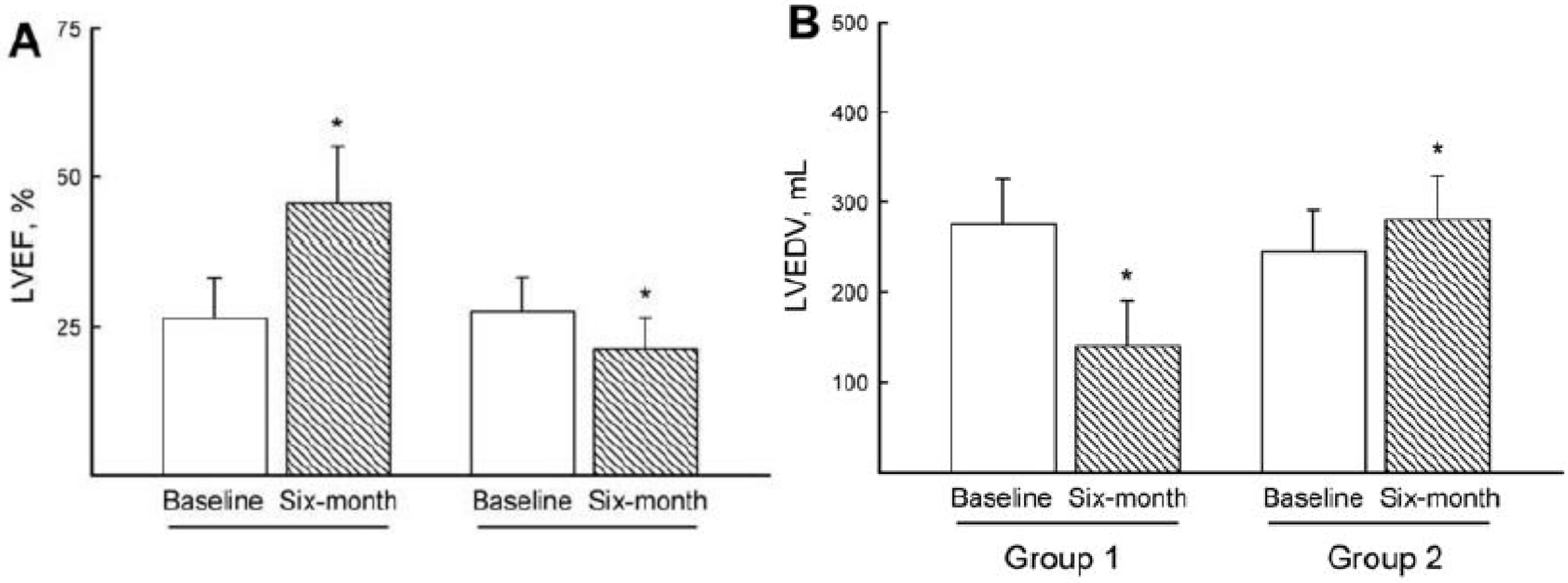




Mason et al, N Engl J Med 1995

Immunosuppression
TIMIC Trial

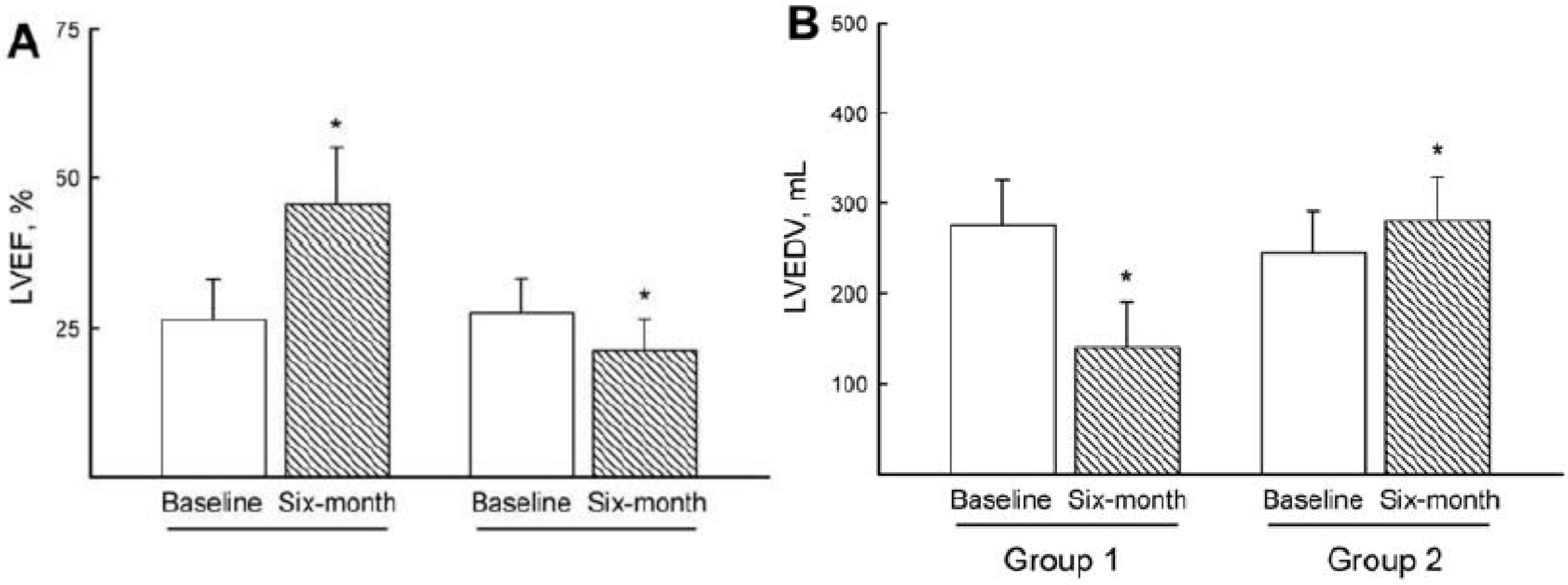
- n=85
- Prednisone & azathiprine vs placebo for 6 months
- Significant improvement in LVEF and LV dimensions



Frustaci et al, Eur Heart J 2009

Immunosuppression
TIMIC Trial

- All biopsies studied with histology and immunohistology and viral infection ruled out by molecular methods



Frustaci et al, Eur Heart J 2009

Immunoglobulin

- High dose intravenous immunoglobulin
- Antiviral and immunomodulating actions
- Conflicting evidence (no benefit in adults, **benefit in children**)
- No benefit in IMAC trial (recent-onset DCM, only 15% biopsy-proven myocarditis of non-specified cause)

McNamara et al, Circulation 2001

Immunoabsorption

- **Elimination of anticardiac antibodies**

- Small studies in DCM, improved LV function and decreased myocardial inflammation

- Ongoing trial in 200 pts in Europe

Mobini et al, I Autoimmun 2003
Felix et al, J Am Coll Cardiol 2000

Antiviral therapy

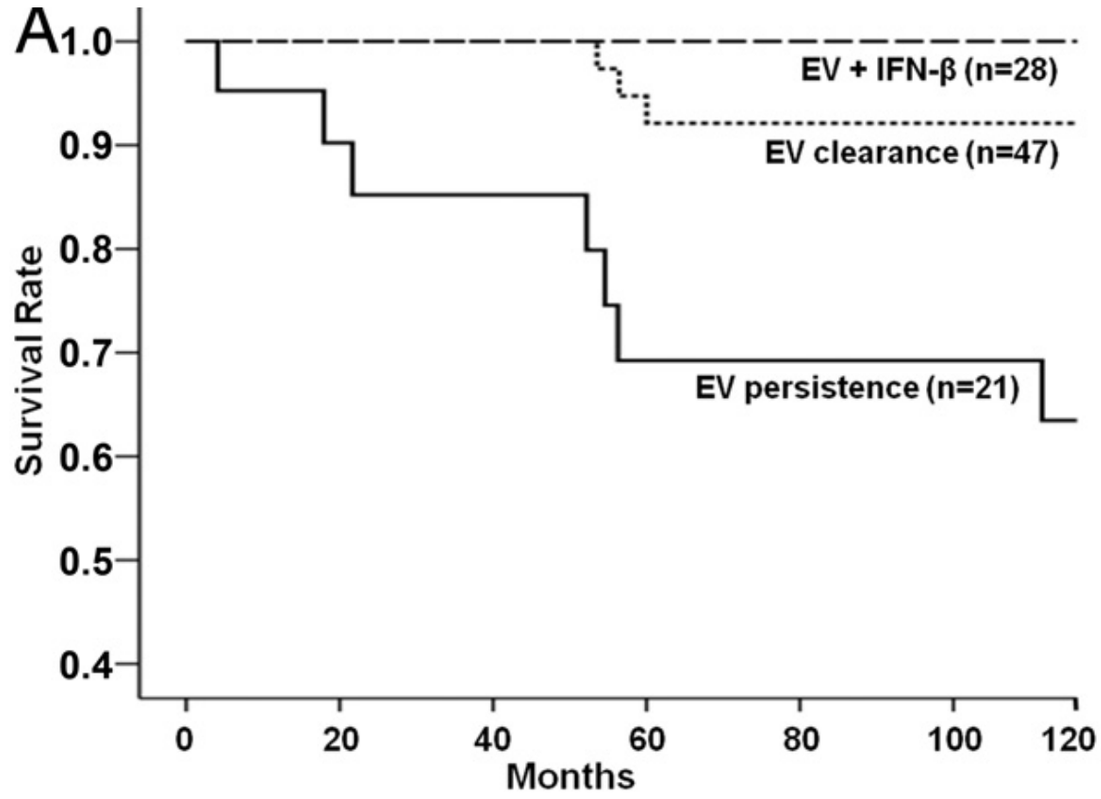
- Viral infection a common cause
- Encouraging results with IFN-β

IFN-β

LV dysfunction and viral persistence (adeno/enterovirus) – 2 studies

IFN-β induced:

- Viral elimination
- Improved NYHA
- Improved LVEF
- Improved survival



Kuhl et al, Circulation 2003

Kuhl et al, J Am Coll Cardiol 2012 (letter)

IFN-β: BICC trial

- 143 patients, inflammatory DCM and confirmed viral infection (adeno/enterovirus, PRVB19)
- Betaferon for 6 months :
  - Viral elimination (*not complete for PVB19*)
  - Improved NYHA and PGA

Schultheiss et al, Circulation 2008 (abstract)

Conclusions I

- Despite advances, standard therapy remains limited to **general HF therapy** in most cases

- Several clinical trials on immune therapies suffer **methodology problems**

- **Biopsy** may guide therapy if classical histology is combined with immunohistochemistry staining for inflammation and molecular studies for viral genome detection

Conclusions II

- **Immunosuppression**
  - indicated for acute giant cell myocarditis, sarcoidosis and acute myocarditis associated with autoimmune diseases (eg SLE,Ghurg Strauss)
  - may be beneficial in virus-negative inflammatory cardiomyopathy

- **Antiviral therapy** may be beneficial in proven viral inflammatory cardiomyopathy

Περικαρδίτιδα

- Εμπύρετος ιογενής συνδρομή
- Πόνος
- Συνεχής
- Επιδείνωση σε ύπτια θέση- μείωση σε πρόσθια κάμψη του κορμού
- Αντανάκλαση σε τραπεζοειδή μυ
- Ακρόαση: Ήχος τριβής- μείωση καρδιακών τόνων

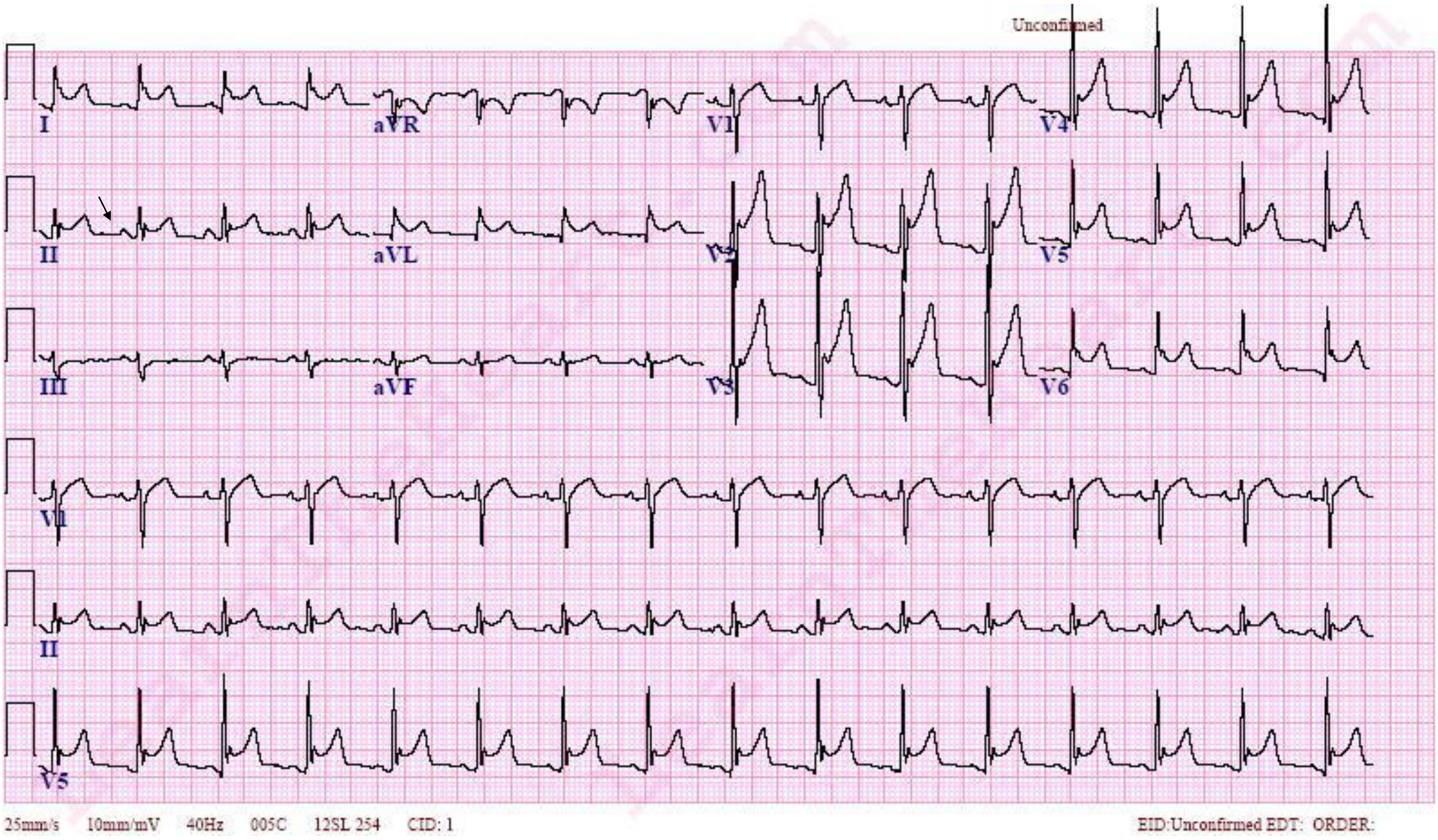
Causes of pericarditis

- ***Unknown cause (so-called "idiopathic")***
- ***Infection —by viral infection***
- ***Radiation***
- ***Trauma.***
- ***Myocardial infarction .***
- ***Drugs and toxins***
- ***Metabolic disorders — kidney failure.***
- ***Cancerous tumors***
- ***Rheumatic diseases***
- ***Gastrointestinal diseases — ulcerative colitis or Crohn's disease)***

Διαγνωστική προσέγγιση

- Δείκτες φλεγμονής (Λευκά, ΤΚΕ, CRP)
- Καρδιακά ενζυμα (Τροπονινη)
- ΗΚΓ (ανάσπαση ST με κυρτό προς τα άνω πτώση του PR, κολπική μαρμαρυγη)
- Α/α θώρακα ↑ΚΘΔ
- Echo (περικαρδιακη συλλογή, ↓ΚΕ)

Περικαρδίτιδα

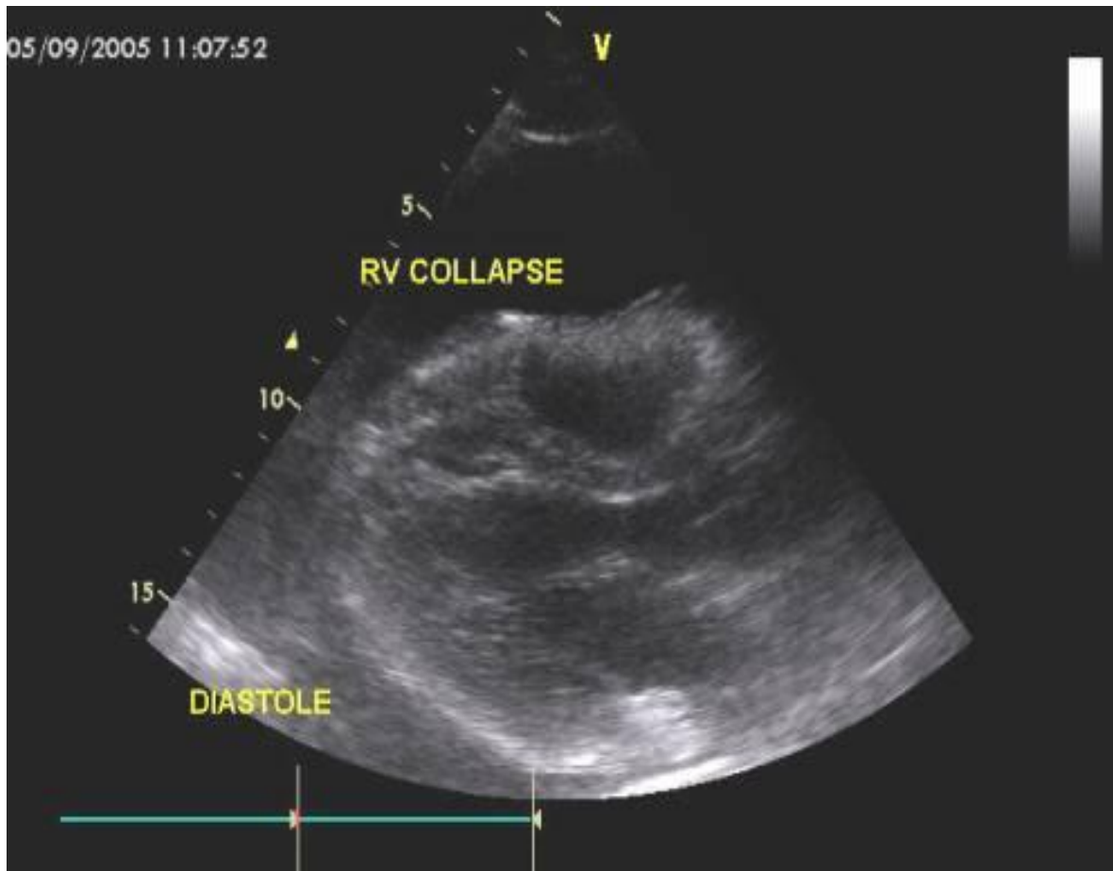


Notice the ST elevations all over the place with no reciprocal changes

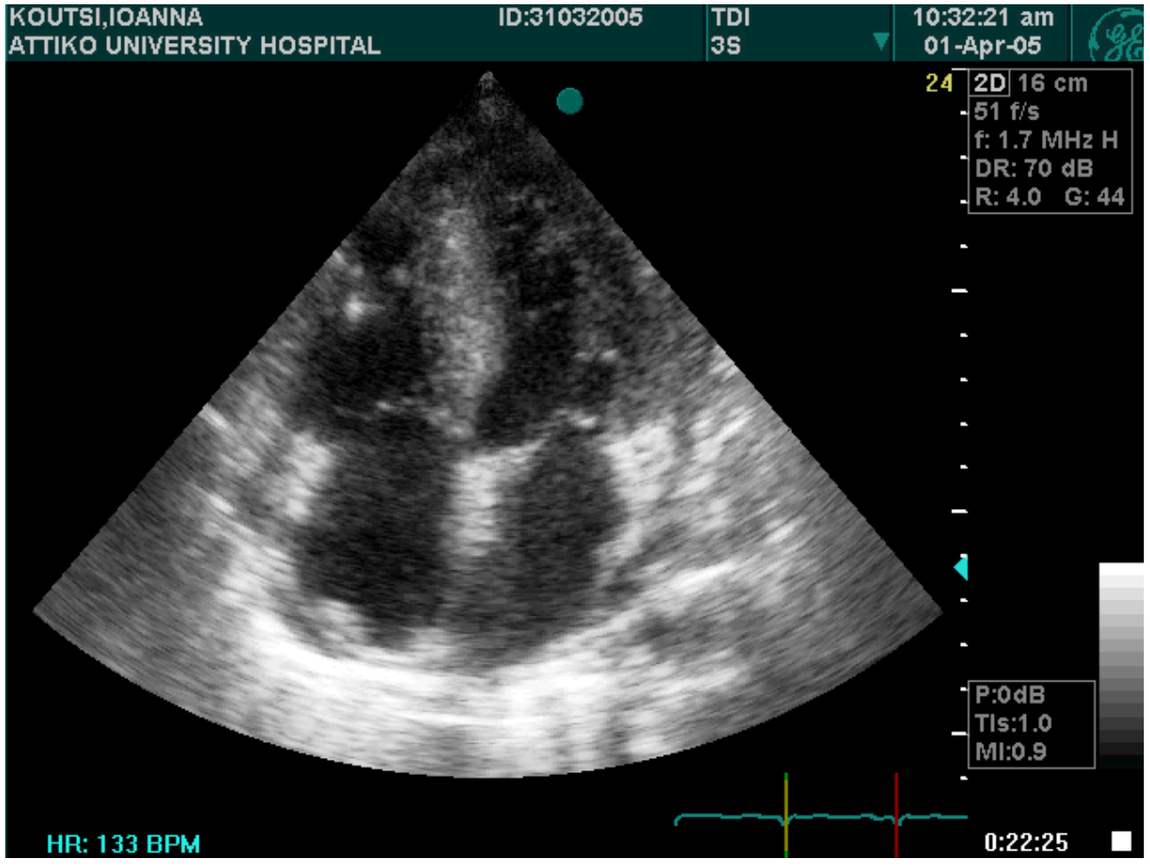
Comparison of ECG Changes Associated with Acute Pericarditis, Myocardial Infarction and Early Repolarization

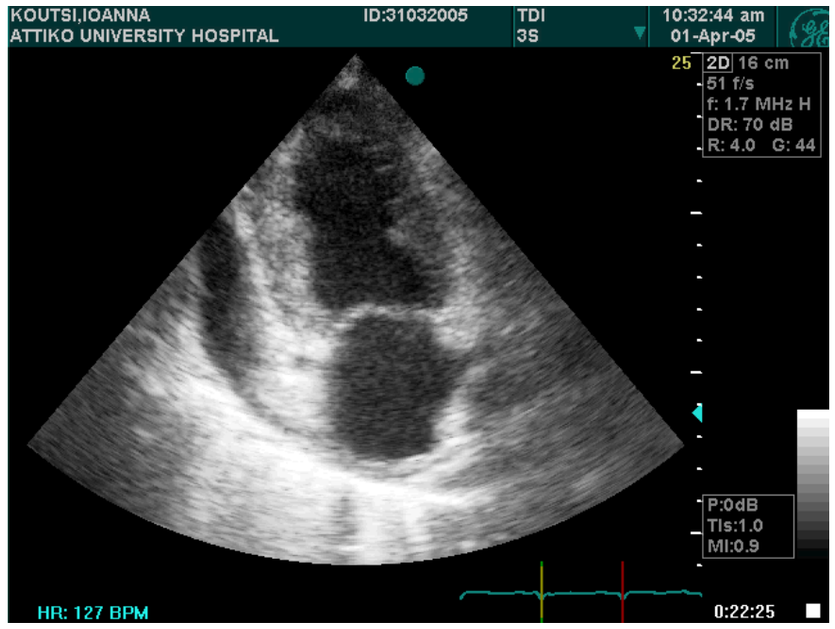
ECG finding	Acute pericarditis	Myocardial infarction	Early repolarization
ST-segment shape	Concave upward	Convex upward	Concave upward
Q waves	Absent	Sometimes Present	Absent
Reciprocal ST-segment changes	Absent	Sometimes Present	Absent
Location of ST-segment elevation	Limb and precordial leads	Area of involved artery	Limb and Precordial leads
ST/T ratio in lead V6*	>0.25	N/A	<0.25
Loss of R-wave voltage	Absent	Present	Absent
PR-segment depression	Sometimes in Lead II	Absent	Absent

If you are unsure if it is a STEMI or not, **KEEP GETTING EKGs! PLASTER THE WALLS WITH EKG PAPER! STEMIs evolve!** They don't look the same. If you have 10 EKGs in the last hour, and they all look the same.... it's not a STEMI!


05/09/2005 11:07:52
V
5
RV COLLAPSE
10
15
DIASTOLE

ΗΧΩΚΑΡΔΙΟΓΡΑΦΗΜΑ ΠΕΡΙΚΑΡΔΙΤΙΣ-ΜΥΟΚΑΡΔΙΤΙΣ





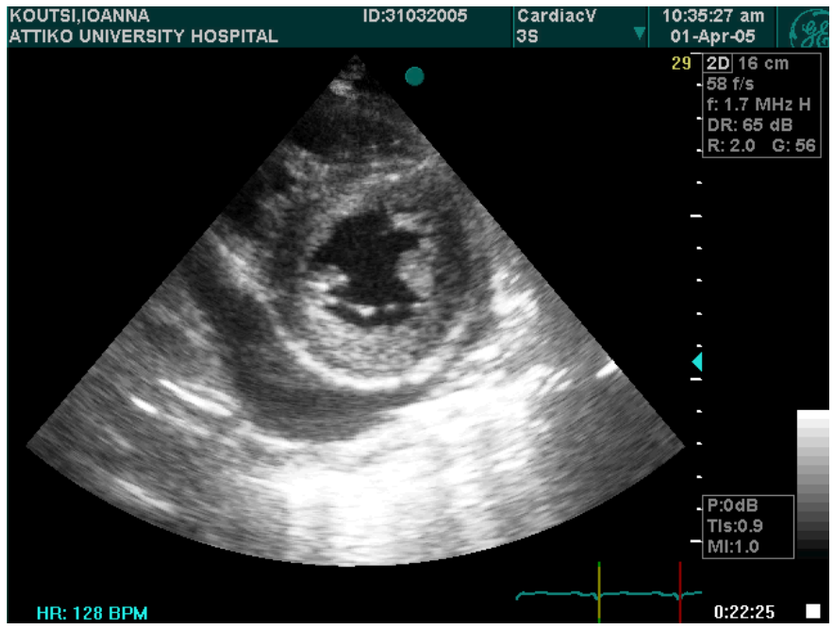


Table 8 Classification of pericardial effusion

Onset	Acute Subacute Chronic (>3 months)
Size	Mild <10 mm Moderate 10–20mm Large >20 mm
Distribution	Circumferential Loculated
Composition	Transudate Exudate

Diagnosis of acute pericarditis
(2 of 4 clinical criteria: pericardial chest pain, pericardial rubs, ECG changes; pericardial effusion)

First line — Aspirin or NSAID + colchicine + exercise restriction

Second line — Low-dose corticosteroids
(in case of contraindications to aspirin/NSAID/colchicine and after exclusion of infectious cause)

Recurrent pericarditis
(after symptom-free interval 4–6 weeks)

First line — Aspirin or NSAID + colchicine + exercise restriction

Second line — Low-dose corticosteroids
(in case of contraindications to aspirin/NSAID/colchicine and after exclusion of infectious cause)

Third line — i.v. immunoglobulin or anakinra or azathioprine[a]

Fourth line — Pericardiectomy

Low-dose corticosteroids are considered when there are contraindications to other drugs or when there is an incomplete response to aspirin/NSAIDs plus colchicine; in this case physicians should consider adding these drugs instead of replacing other anti-inflammatory therapies.

[a]Azathioprine is steroid-sparing and has a slow onset of action compared with IVIG and anakinra. Cost considerations may apply considering the cheaper solution first (e.g. azathioprine) and resorting to more expensive options (e.g. IVIG and anakinra) for refractory cases.

Ερωτησεις

- Ποιος β αναστολέας είναι ασφαλέστερος στη μυοκαρδίτιδα?
- 1 carvedilol
- 2 metoprolol
- 3 atenolol
- 4 κανένας

- Η βιοψία μυοκαρδίου παρουσιάζει PCR θετική για HHV6 σε ασθενή με οξεία μυοκαρδίτιδα. Η ενδεικνυόμενη αγωγή είναι
- 1. ιντερφερόνη β
- 2. αζαθειοπρίνη με κορτικοστεροειδή
- 3. κυκλοσπορίνη με κορτικοστεροειδή
- 4. κλαριθρομυκίνη

- Η βιοψία μυοκαρδίου παρουσιάζει ανοσο ιστοχημεια συμβατή με γιγαντοκυτταρικη μυοκαρδίτιδα. Ποιο είναι σωστό
- 1. Τοποθέτηση απινιδωτη άμεσα μόλις εκδηλωθεί κοιλιακή ταχυκαρδία
- 2 χορήγηση αζαθειοπρίνης και στεροειδών
- 3 ενδαορτική αντλία σε αιμοδυναμική κατέρριψη
- 4.Όλα τα παραπάνω

Position paper ESC

European Heart Journal (2013) 34, 2636–2648
doi:10.1093/eurheartj/eht210

ESC REPORT

Current state of knowledge on aetiology, diagnosis, management, and therapy of myocarditis: a position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases

Alida L. P. Caforio[1†*], Sabine Pankuweit[2†], Eloisa Arbustini[3], Cristina Basso[4], Juan Gimeno-Blanes[5], Stephan B. Felix[6], Michael Fu[7], Tiina Heliö[8], Stephane Heymans[9], Roland Jahns[10], Karin Klingel[11], Ales Linhart[12], Bernhard Maisch[2], William McKenna[13], Jens Mogensen[14], Yigal M. Pinto[15], Arsen Ristic[16], Heinz-Peter Schultheiss[17], Hubert Seggewiss[18], Luigi Tavazzi[19], Gaetano Thiene[4], Ali Yilmaz[20], Philippe Charron[21], and Perry M. Elliott[13]

Πανελλήνια καταγραφή μυοκαρδίτιδας ΕΜΕΚΑ –ΟΕ Καρδιακής Ανεπάρκειας ΕΚΕ

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Study ID **1-2**

Data Collection Instrument	Events						
	Visit 0 (1)	Visit 1 (1 month) (2)	Visit 2 (3 months) (3)	Visit 3 (6 months) (4)	Visit 4 (12 months) (5)	Visit 5 (24 months) (6)	Visit 6 (36 months) (7)
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Past_medical_history	●						
Symptoms	●	●	●	●	●	●	●
Physical_examination	●	●	●	●	●	●	●
Hematologic_examination	●	●	●	●	●	●	●
Electrocardiography	●	●	●	●	●	●	●
Echocardiography	●	●	●	●	●	●	●
CMR	●			●	●		
Coronary_Arteriography	●						
Coronary_CT	●						
ECG Holter monitoring	●	●	●	●			
Endomyocardial_biopsy	●						
Discharge_medication	●	●	●	●	●	●	●

Πανελλήνια καταγραφή μυοκαρδίτιδας ΕΜΕΚΑ –ΟΕ Καρδιακής Ανεπάρκειας ΕΚΕ

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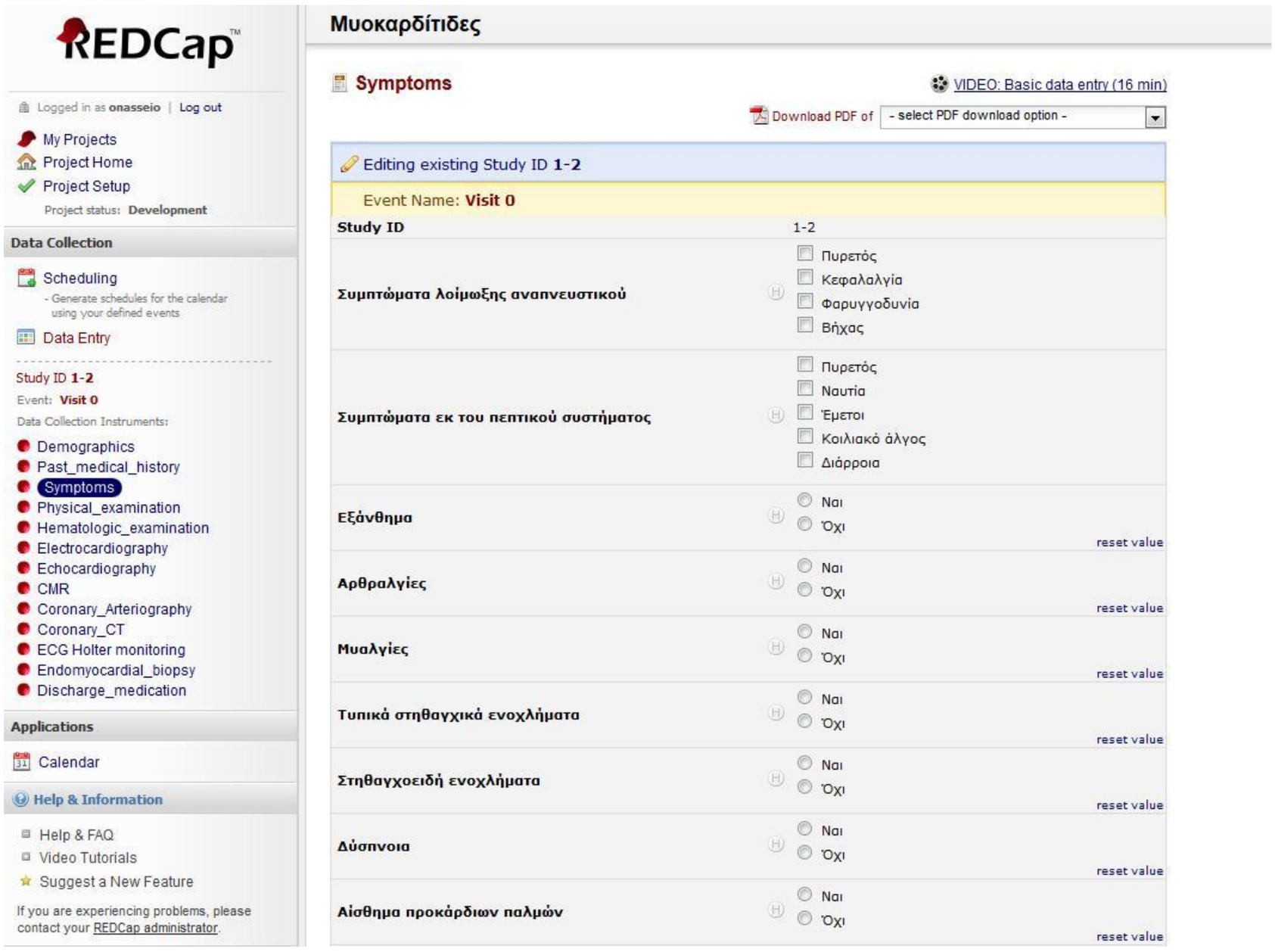

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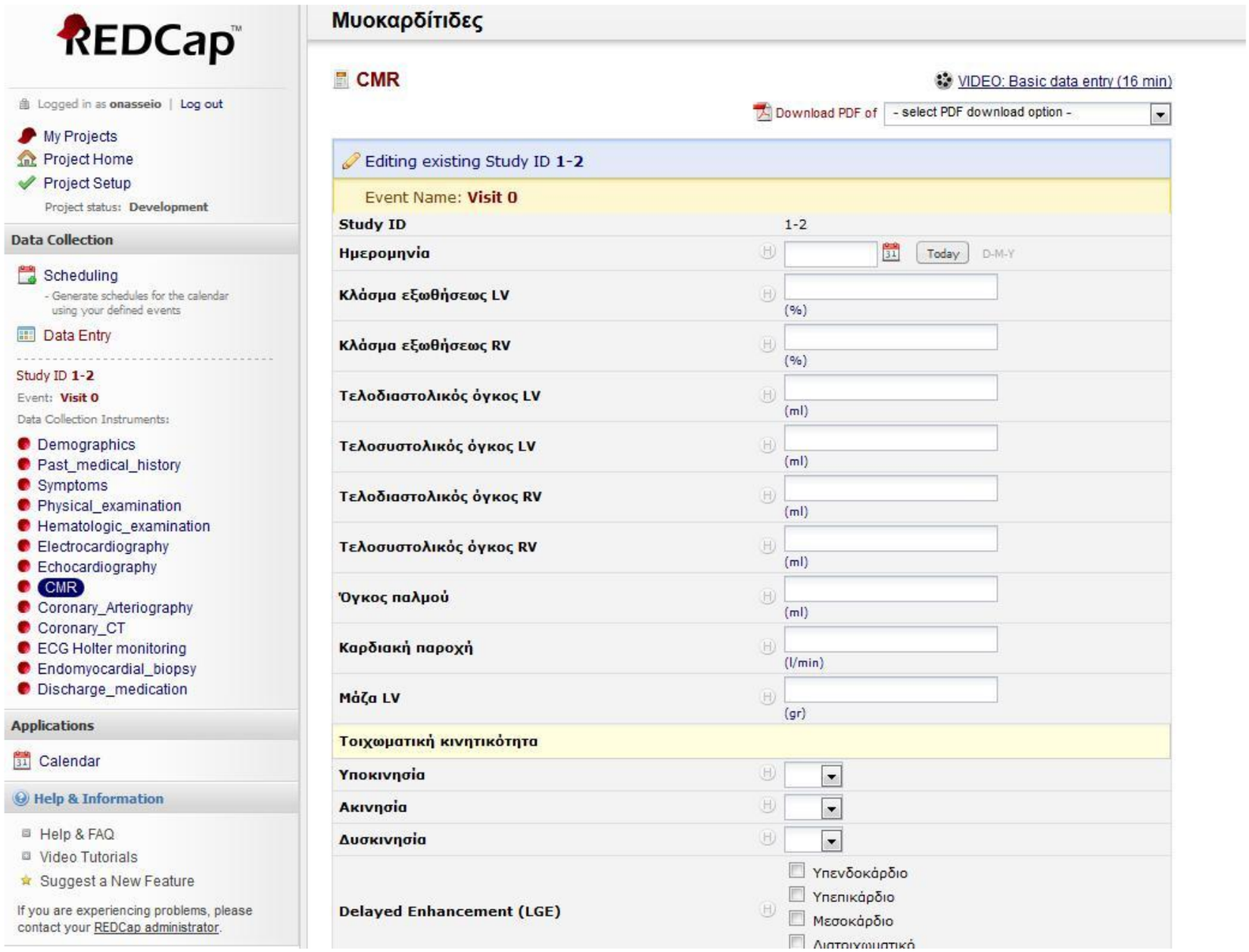
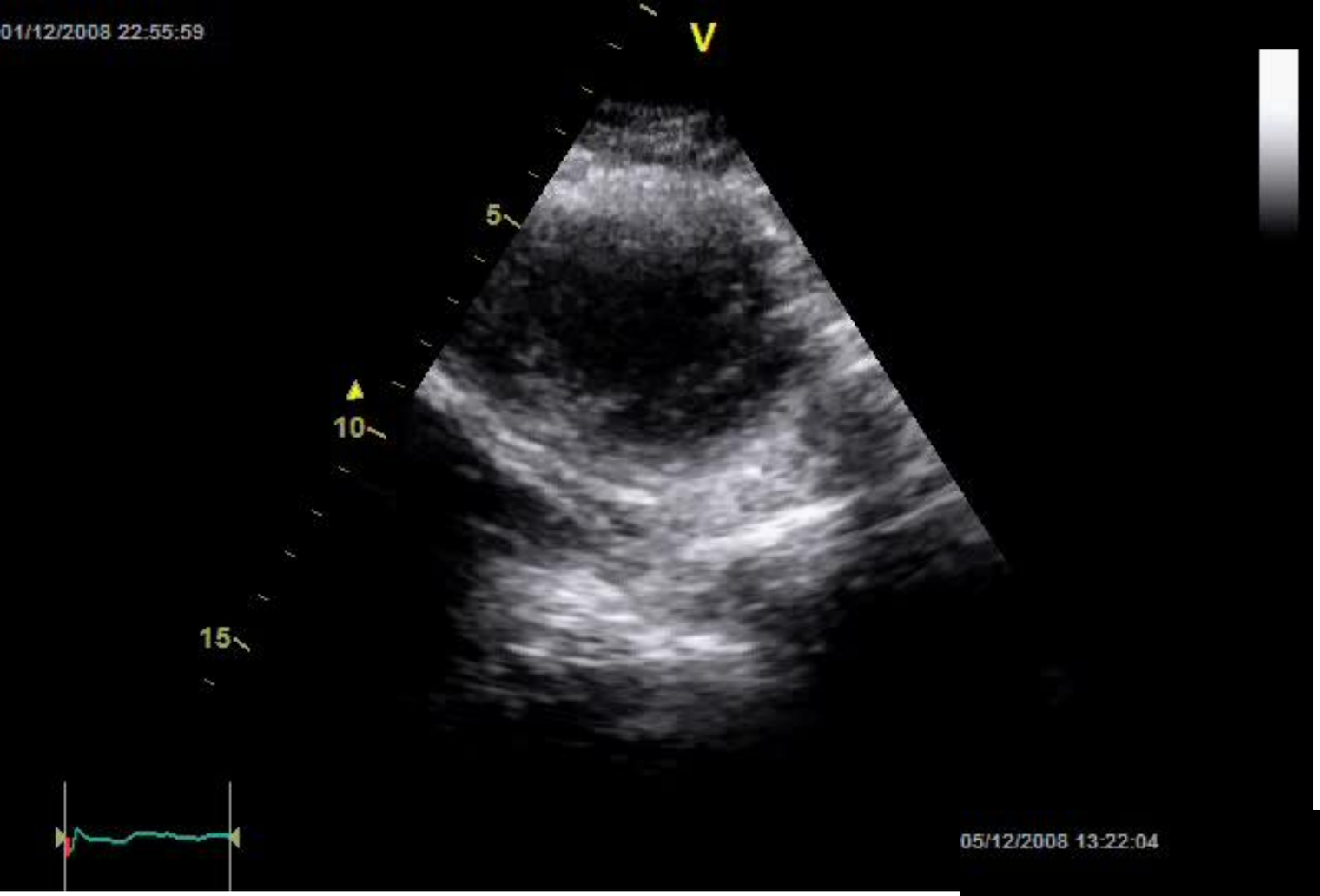


Table 2 **Serum cardiac autoantibodies in autoimmune myocarditis/dilated cardiomyopathy: frequency in myocarditis/dilated cardiomyopathy, other cardiac disease (OCD) and normals**

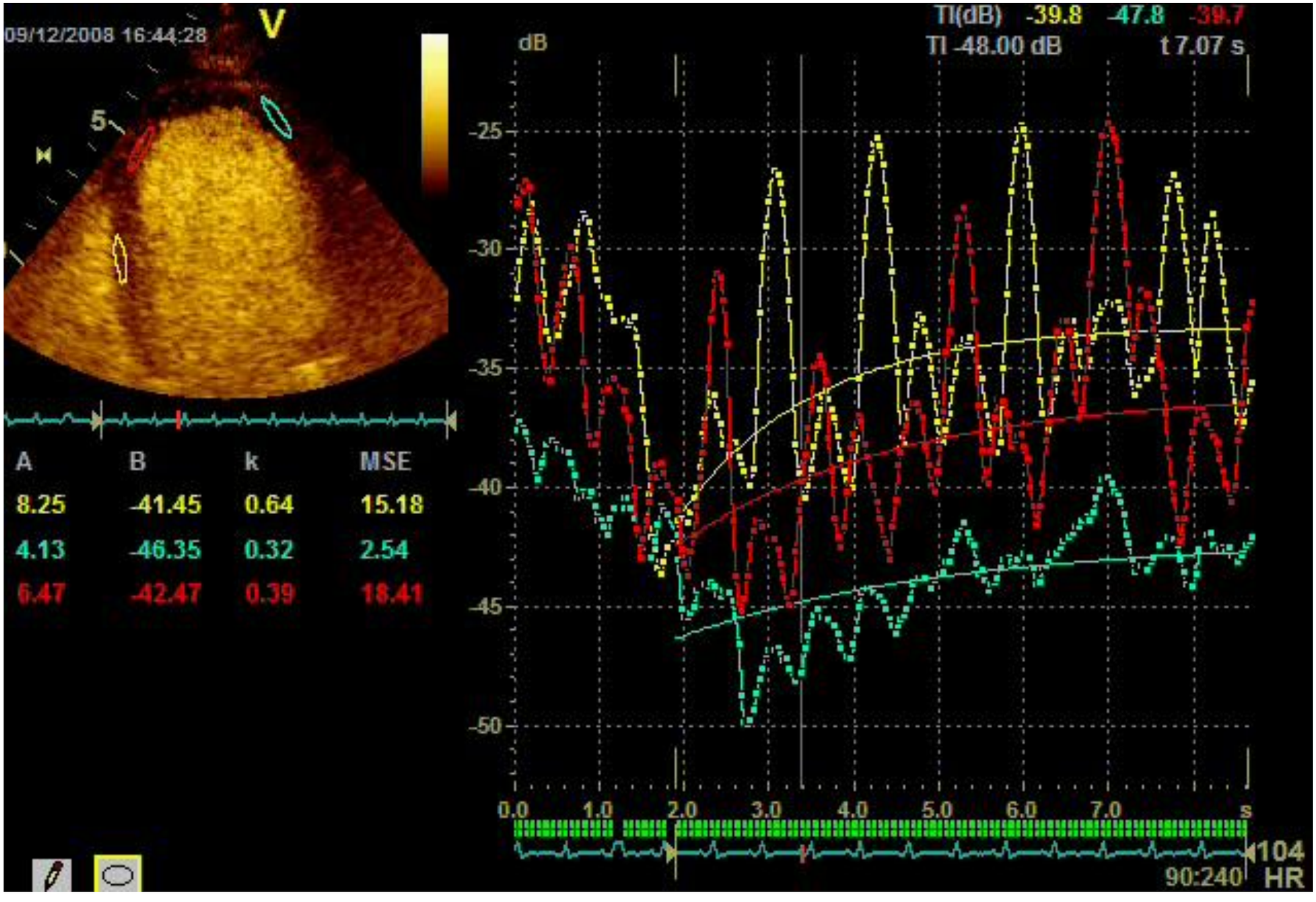
Cardiac autoantibody (Ab)	% aabs positive		%antibody positive		Functional effect/clinical relevance	References
	Myoc	DCM	OCD	Normal		
Muscle-specific ASA, (AFA,IFA,AMLA)	28–59*	9–41*	NT	0–25	Myocytolysis	72,77,57,64
Cardiac-specific						
AHA	41–56*,^,a	26–30*,^,a	1–4	3	Cardiac- and disease-specific early predictors; predict DCM development in relatives	9a,50a,35a, 36a,118a,52a
AIDA	17*,^,a	16*,^,a	2–4	0		
Anti-Beta1-AR	33	40–51^	13–55	0–13	Negative predictors, pro-apoptotic and other *in vitro* effects[b]	48,55,61–63,66a,72,74–76, 78,84,109,88,90,92,93,98
	NT	35*,^,a	16	7		
	73–96*,^,a	29–95*,^,a	8	0		
	NT	27–28	10	0		
Anti-Beta2-AR	NT	30–38^	33	15	Association with idiopathic arrhythmia	53^,62,69a,89
	NT	13–14				
	NT	30–75a	37	18		
Anti-muscarinic acetylcholine receptor-2	11	30–77[c]	23[d]–61	8–13	Negative inotropic, muscarinic effects	47d,48c,54,58,59,70,74–75,88,94,98
	NT	83[e]			Association with atrial arrhythmia	
Cardiodepressant (Fγ-gamma-receptor 2a)	NT	64			Negative inotropic effects in rat and human myocytes *in vitro*	56,66^,85–87,91^
Anti-Ky channel-interacting protein 2, KChIP2.6—ELISA)	NT	14^	8	4	Increased cell death in myocytes *in vitro*	
Anti-Alpha-MHC (cardiac-specific)	17–37*,^,a	20–46*,^,a	4–16	0–2.5	Negative predictors, pro-apoptotic	109,51a,60a, 118a,140a
Anti-Beta-MHC (muscle-cross reactive)						68,95,96
Anti-MLC 1v	NT	17^–35	25	0–15		51,67^
Anti-tropomyosin	NT	55^	21	NT		67
Anti-non-myofibrillar	NT	46*,^,a	17	0		51a
Anti-MHC	NT	67^	42	NT		67
Anti-actin	NT	71^	21	NT		67
Anti-Troponin I ,T	NT	1.7^–20^	0^–18	0–4	Negative predictors	66,68,80
Anti-laminin	73	78	25–35	6		97
Anti-HSP60,70	NT	10–85^	1–42	3		67,79
Anti-s.Na/K-ATPase	26*		NT	2	Ventricular tachycardia predictors	49
Anti- ANT	91*,^,a	57*,^,a	0	0	Negative inotropic	81–83a
Anti-M7	13*	31*	10	0		65
Anti-BCKD-E2	100*^	60*^	4	0		46

01/12/2008 22:53:24
V
5
10
15

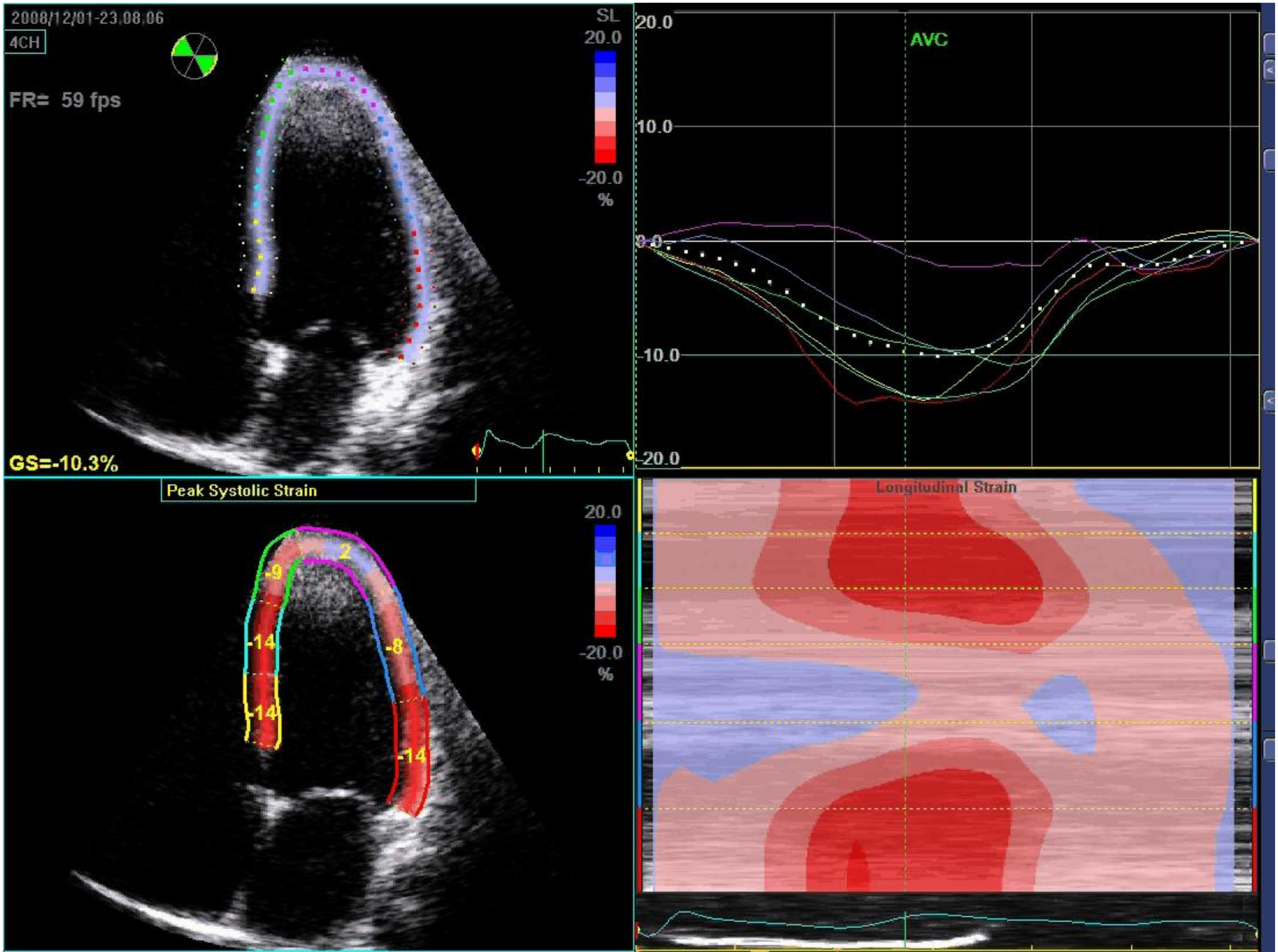
01/12/2008 22:55:36
V
5
10
15
104
2:39 HR


01/12/2008 22:55:59
V
5
10
15

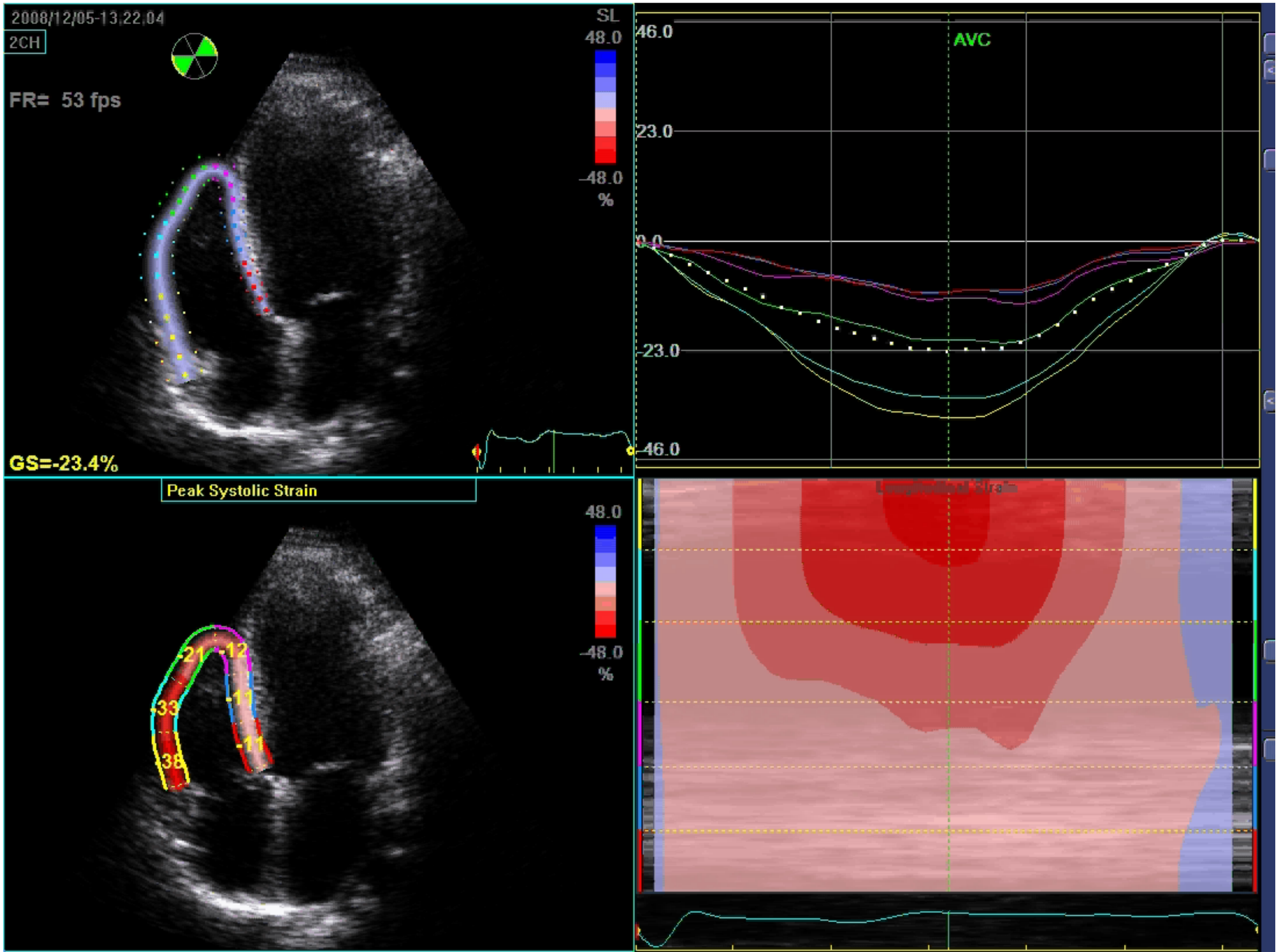
05/12/2008 13:22:04
V
5
10
15
95
3:38 HR

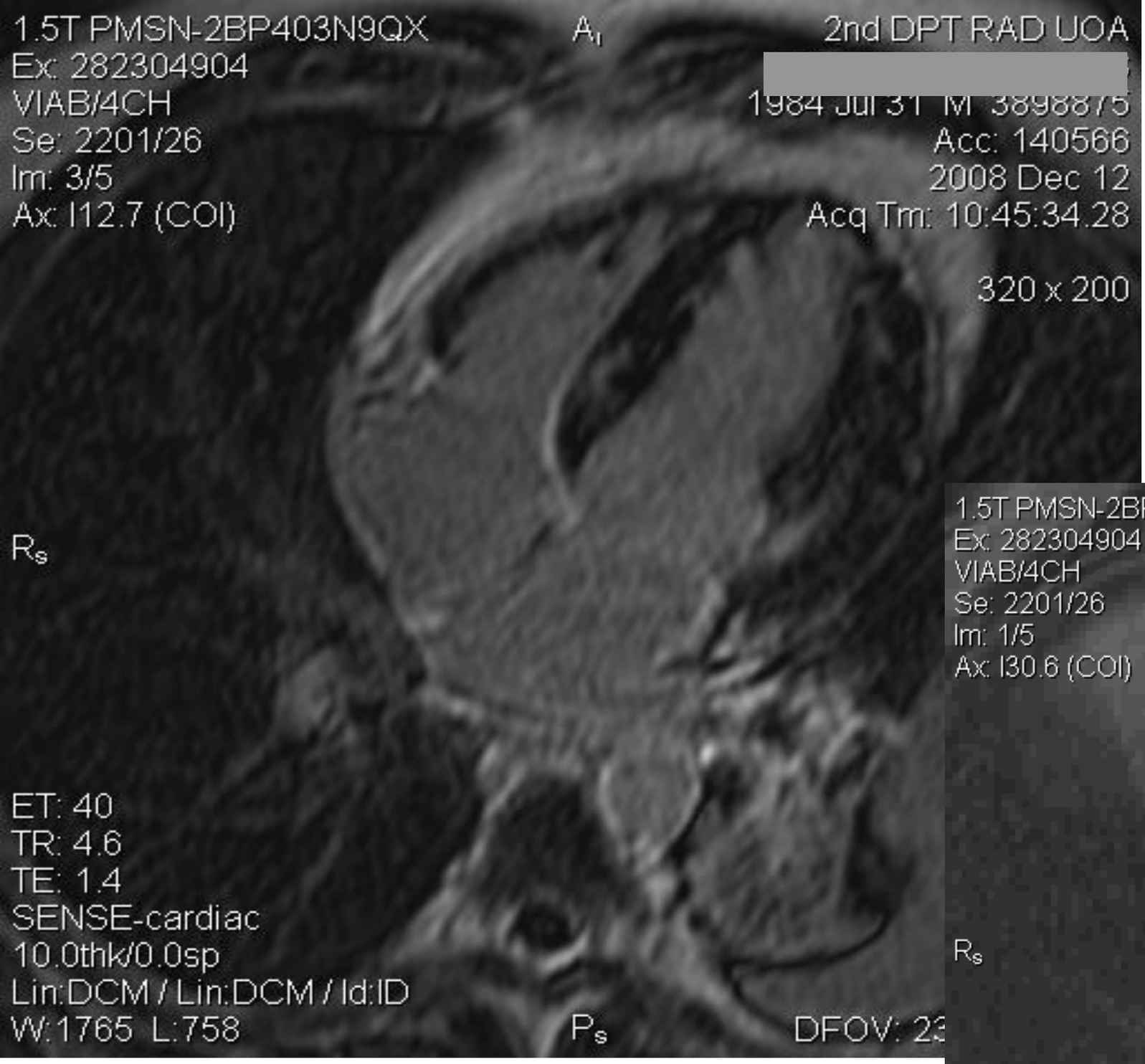

09/12/2008 16:44:28
V
5
A
B
k
MSE
8.25
-41.45
0.64
15.18
4.13
-46.35
0.32
2.54
6.47
-42.47
0.39
18.41
dB
TI(dB) -39.8 -47.8 -39.7
TI -48.00 dB
t 7.07 s
-25
-30
-35
-40
-45
-50
0.0
1.0
2.0
3.0
4.0
5.0
6.0
7.0
s
104
90:240
HR

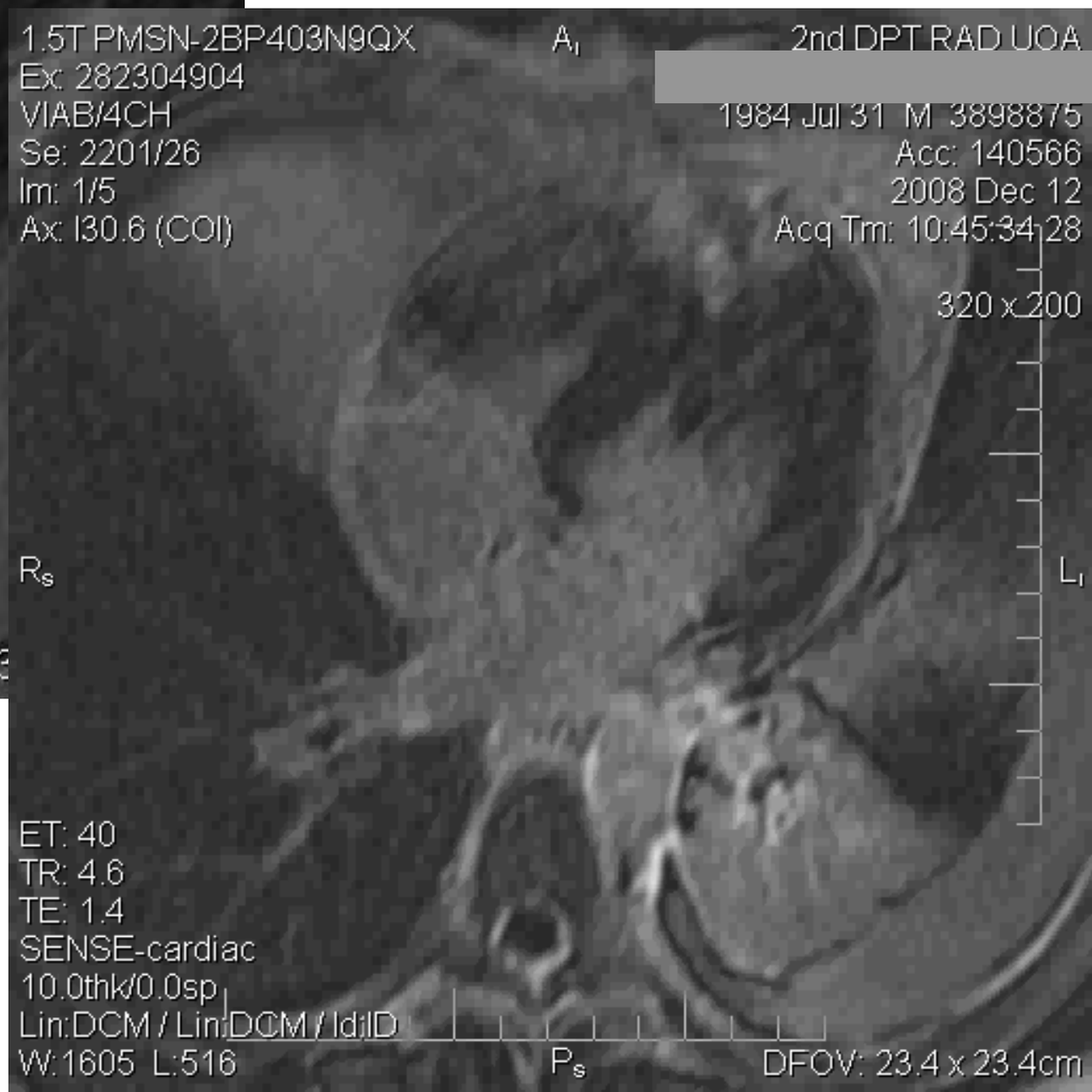
4 chamber view Longitudinal Strain -normal values 15%



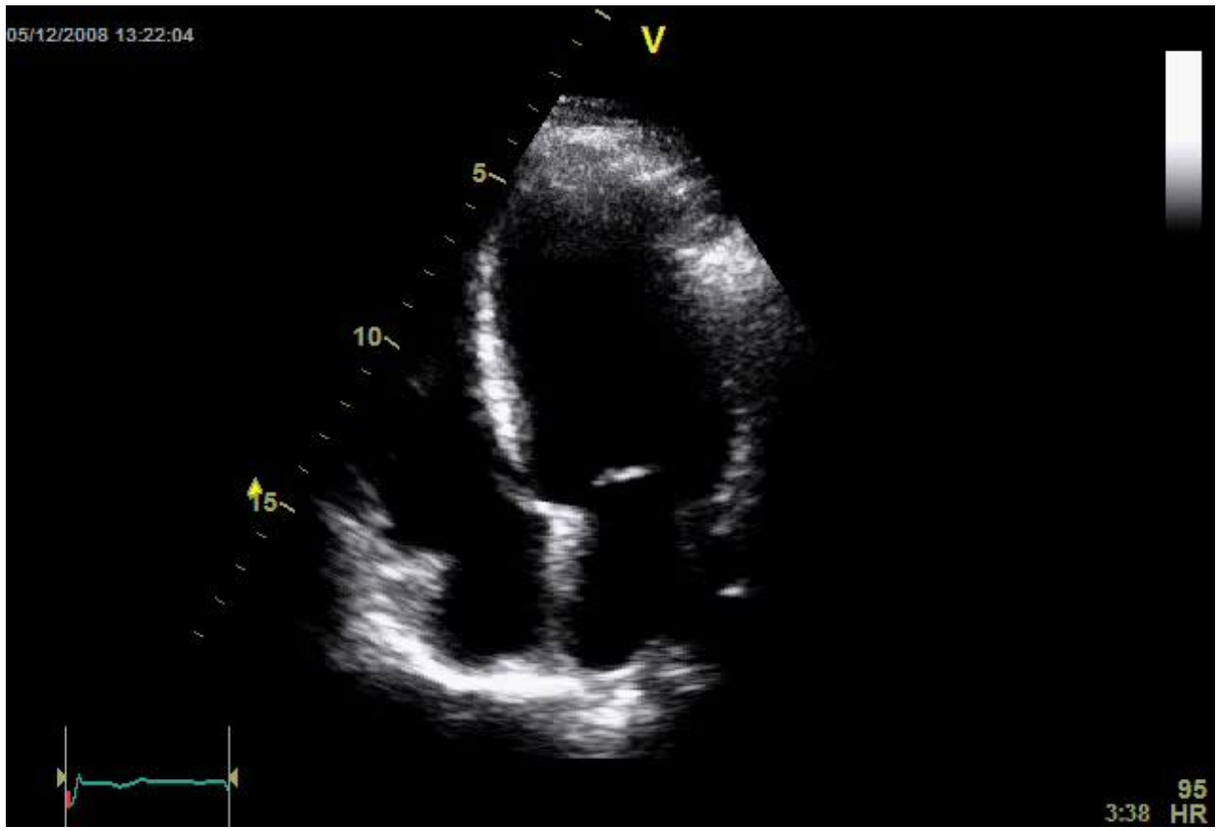
Δεξιά κοιλία

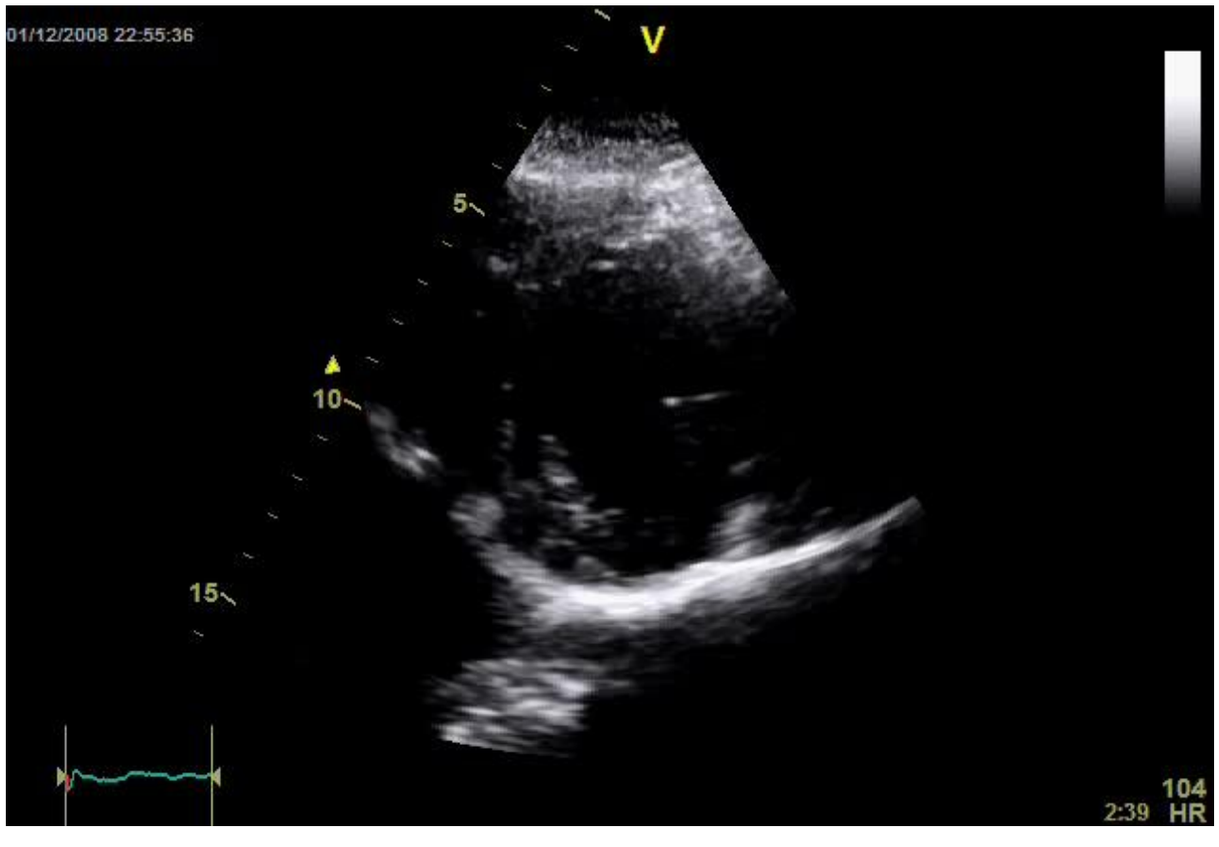



1.5T PMSN-2BP403N9QX
A,
2nd DPT RAD UOA
Ex: 282304904
VIAB/4CH
1984 Jul 31 M 3898875
Se: 2201/26
Acc: 140566
Im: 3/5
2008 Dec 12
Ax: I12.7 (COI)
Acq Tm: 10:45:34.28
320 x 200
Rs
ET: 40
TR: 4.6
TE: 1.4
SENSE-cardiac
10.0thk/0.0sp
Lin:DCM / Lin:DCM / Id:ID
W:1765 L:758
Ps
DFOV: 23

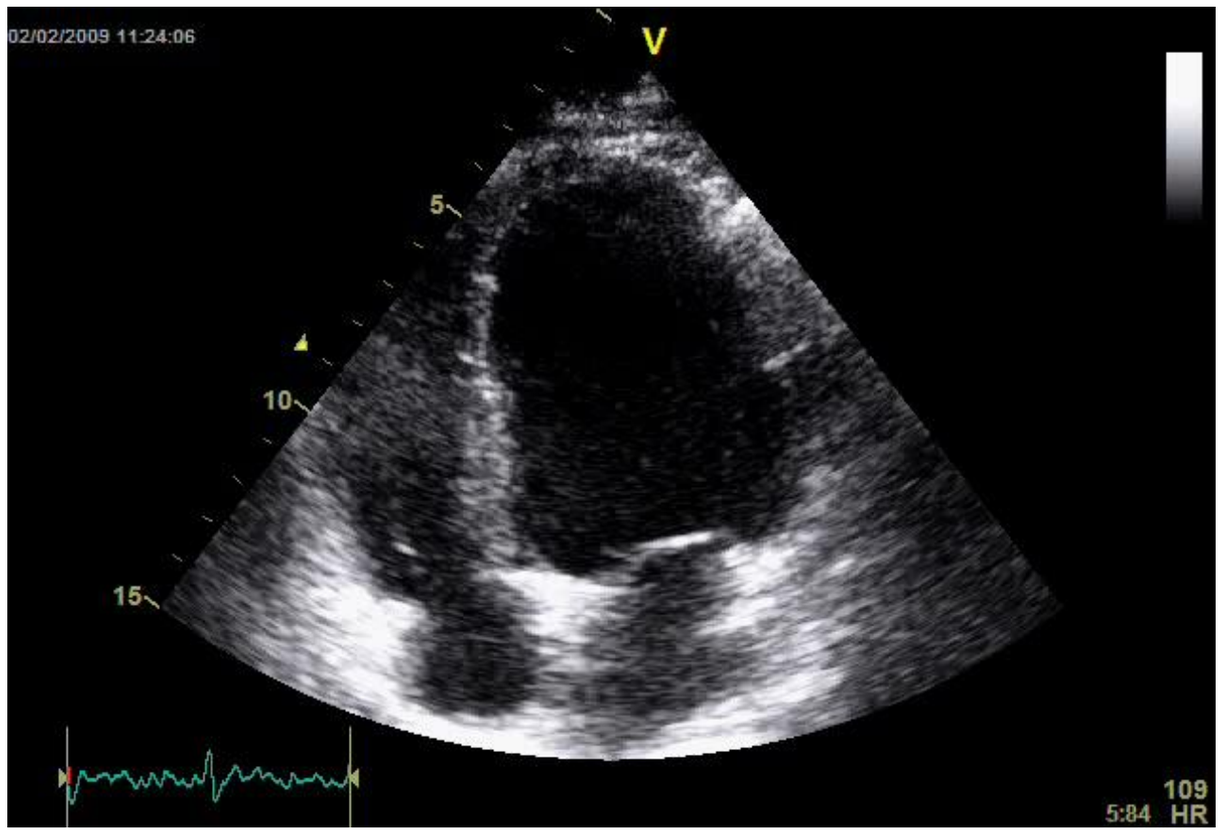

1.5T PMSN-2BP403N9QX
A,
2nd DPT RAD UOA
Ex: 282304904
VIAB/4CH
1984 Jul 31 M 3898875
Se: 2201/26
Acc: 140566
Im: 1/5
2008 Dec 12
Ax: I30.6 (COI)
Acq Tm: 10:45:34.28
320 x 200
Rs
L,
ET: 40
TR: 4.6
TE: 1.4
SENSE-cardiac
10.0thk/0.0sp
Lin:DCM / Lin:DCM / Id:ID
W:1605 L:516
Ps
DFOV: 23.4 x 23.4cm

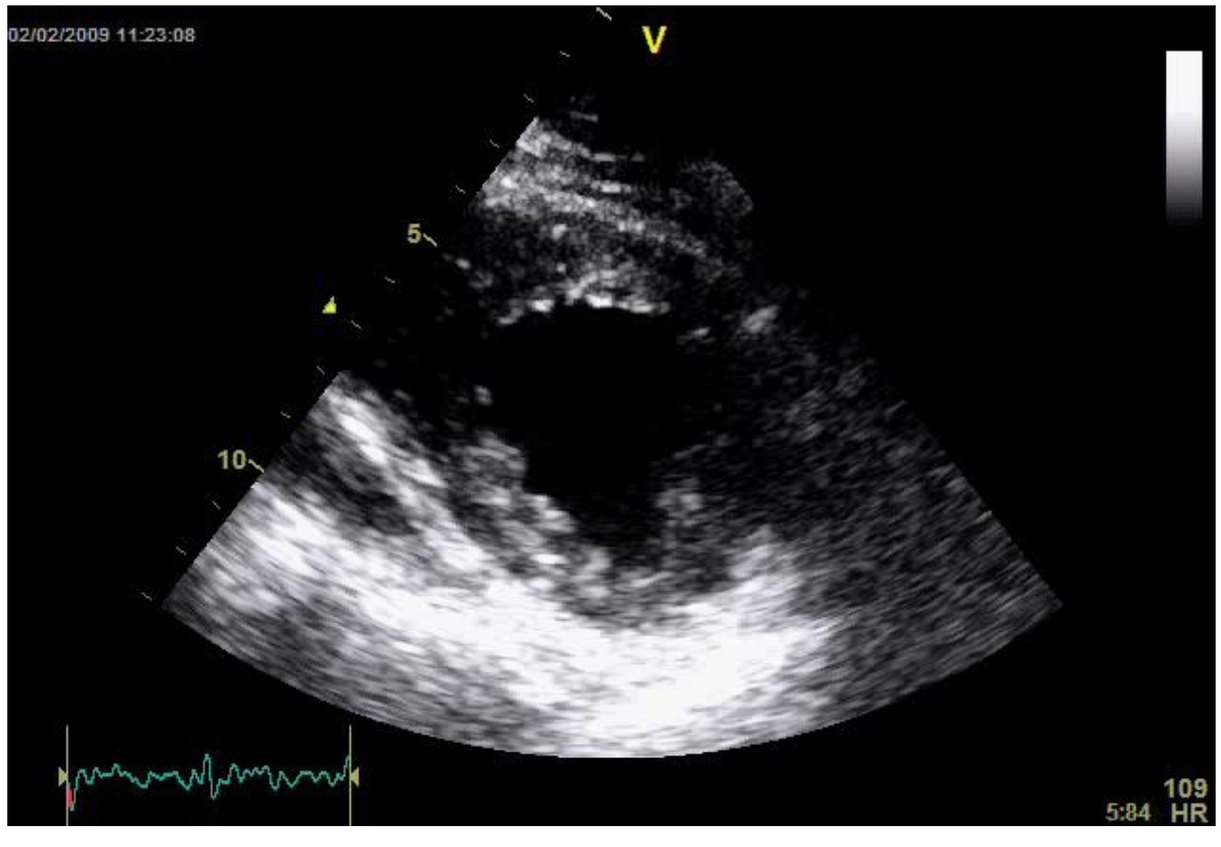
Εισαγωγή



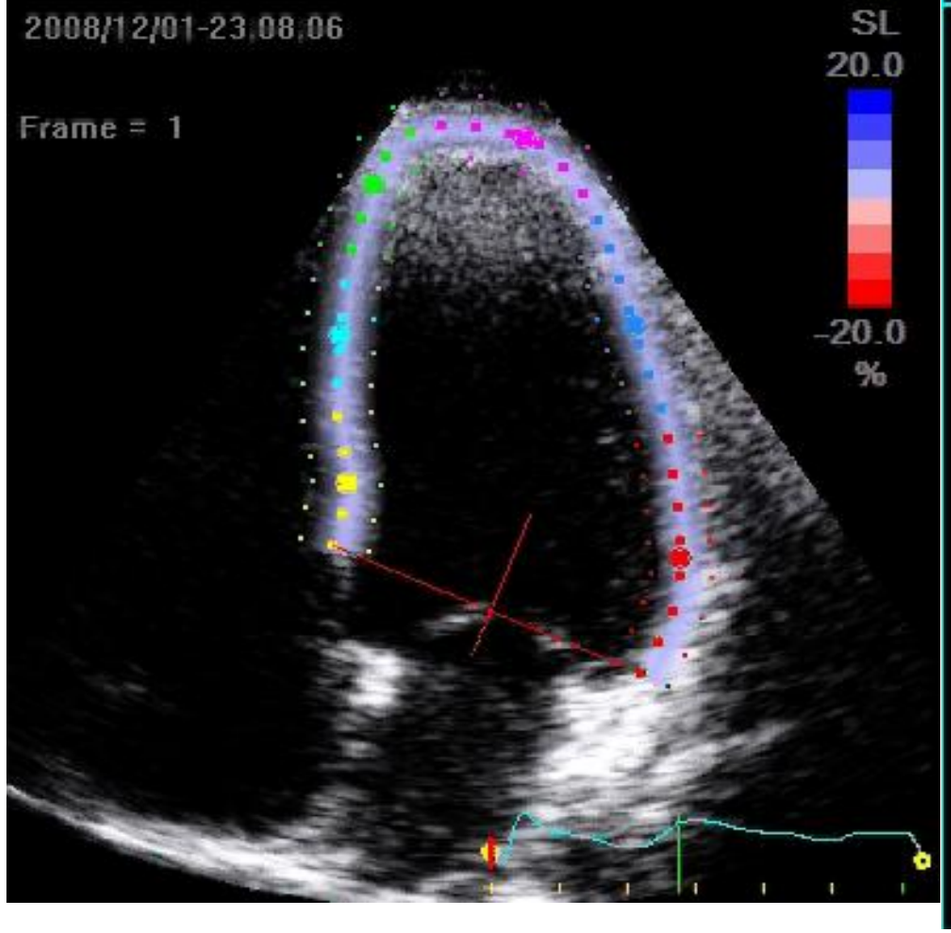


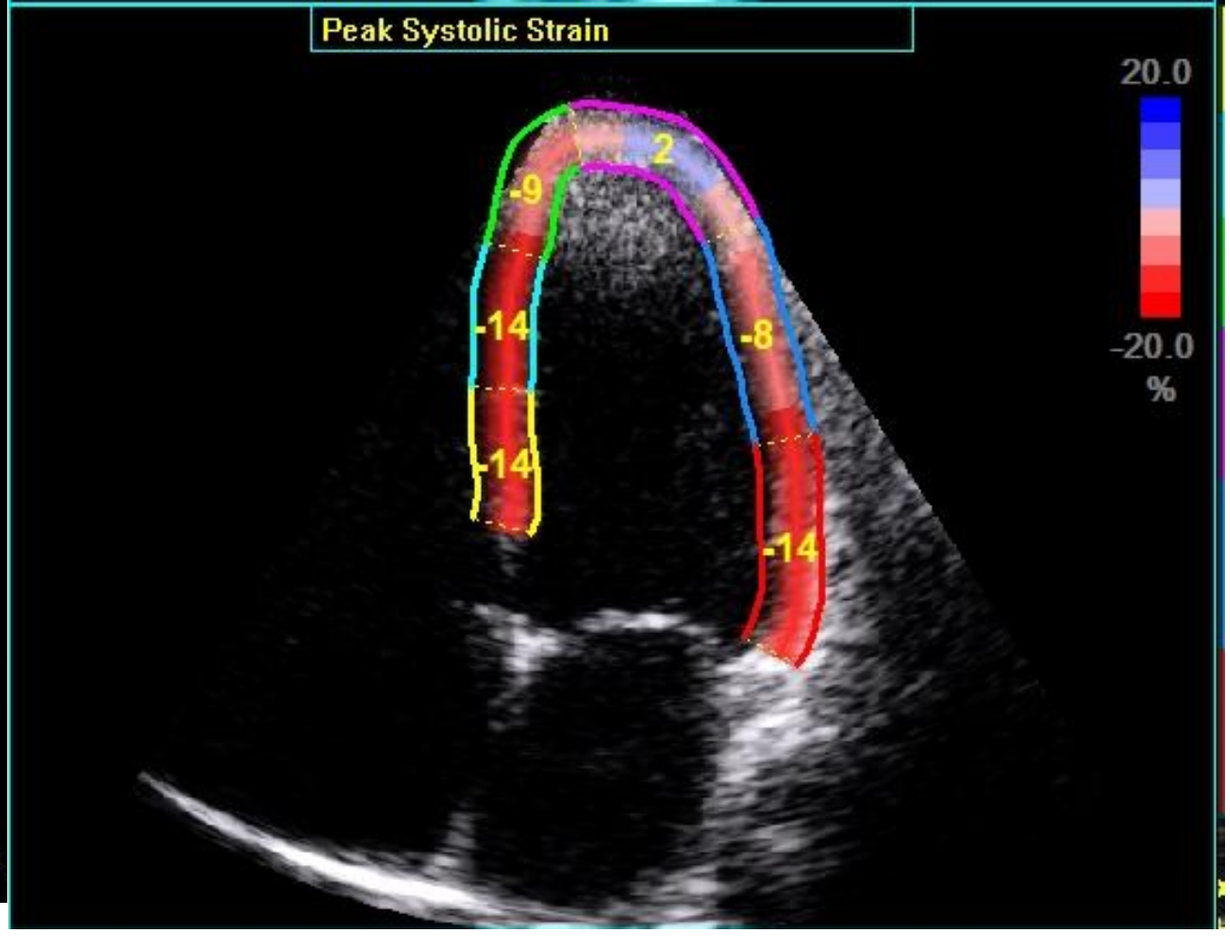
2 μήνες μετά



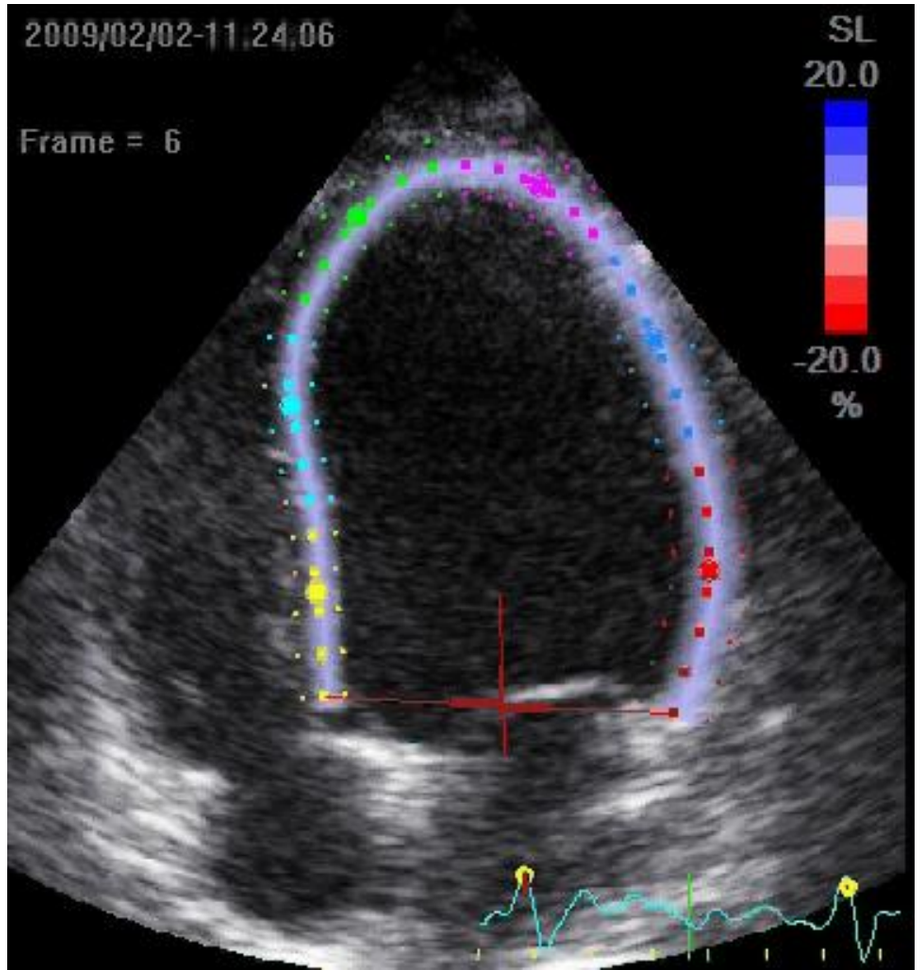


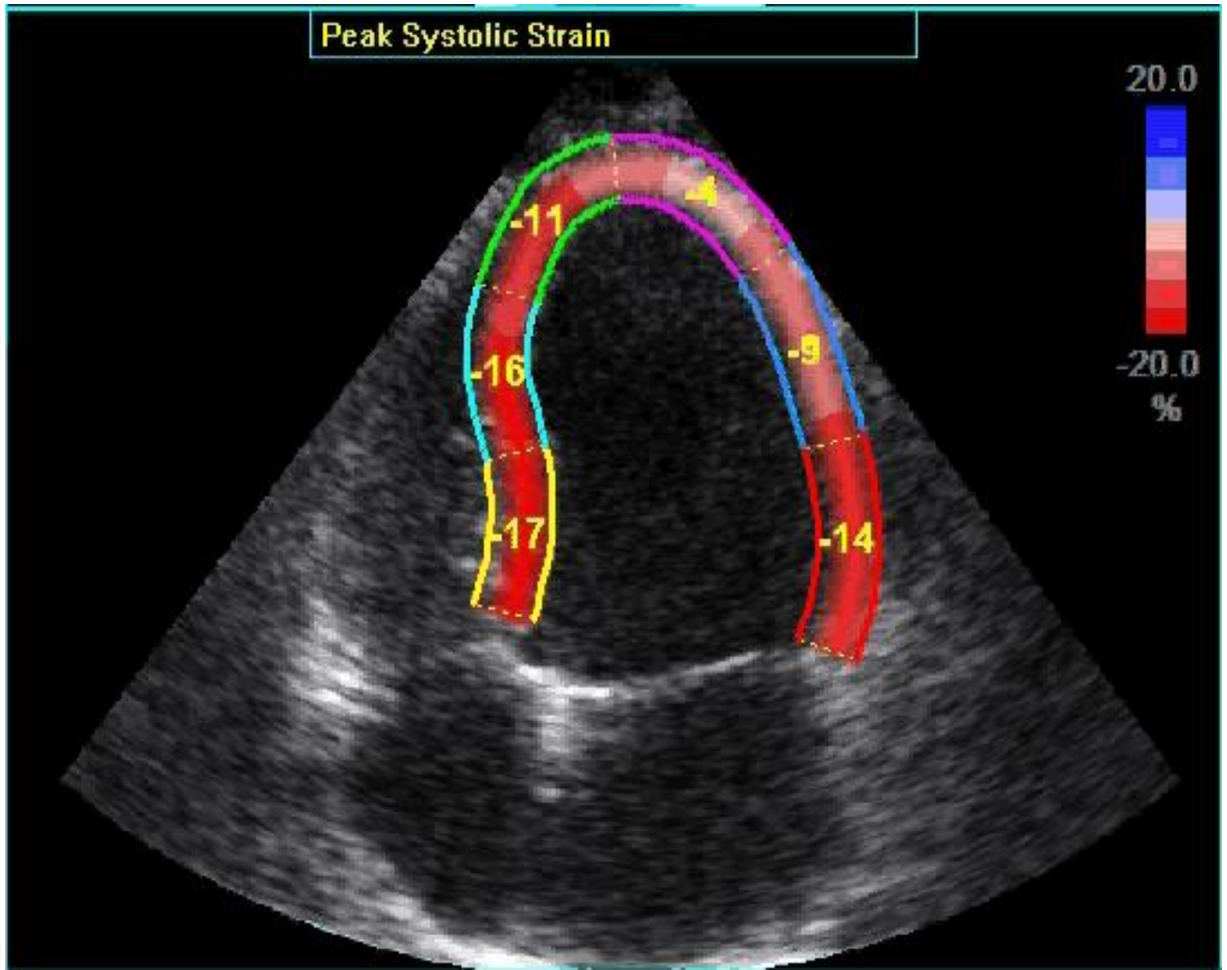
Εισαγωγή

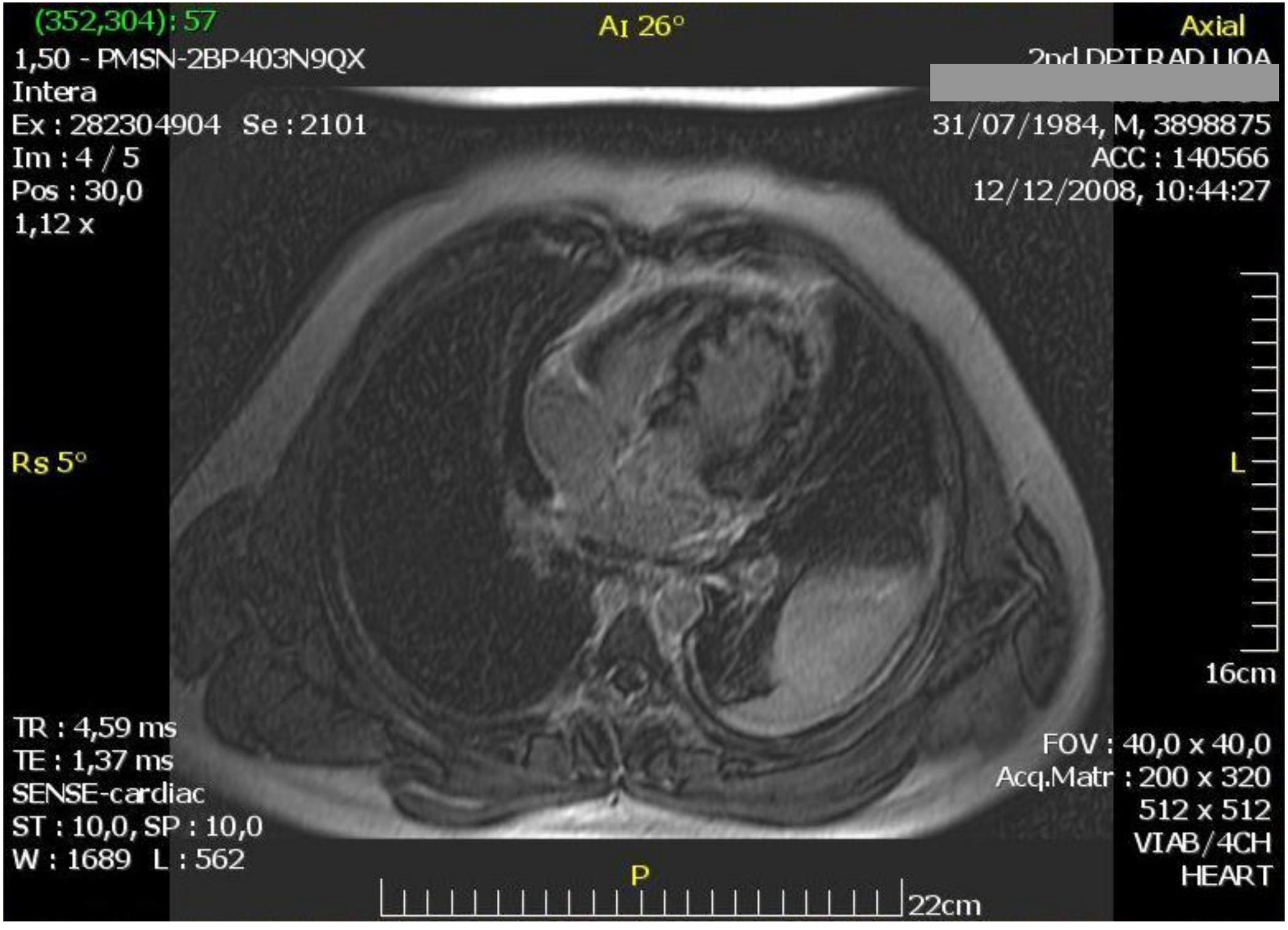




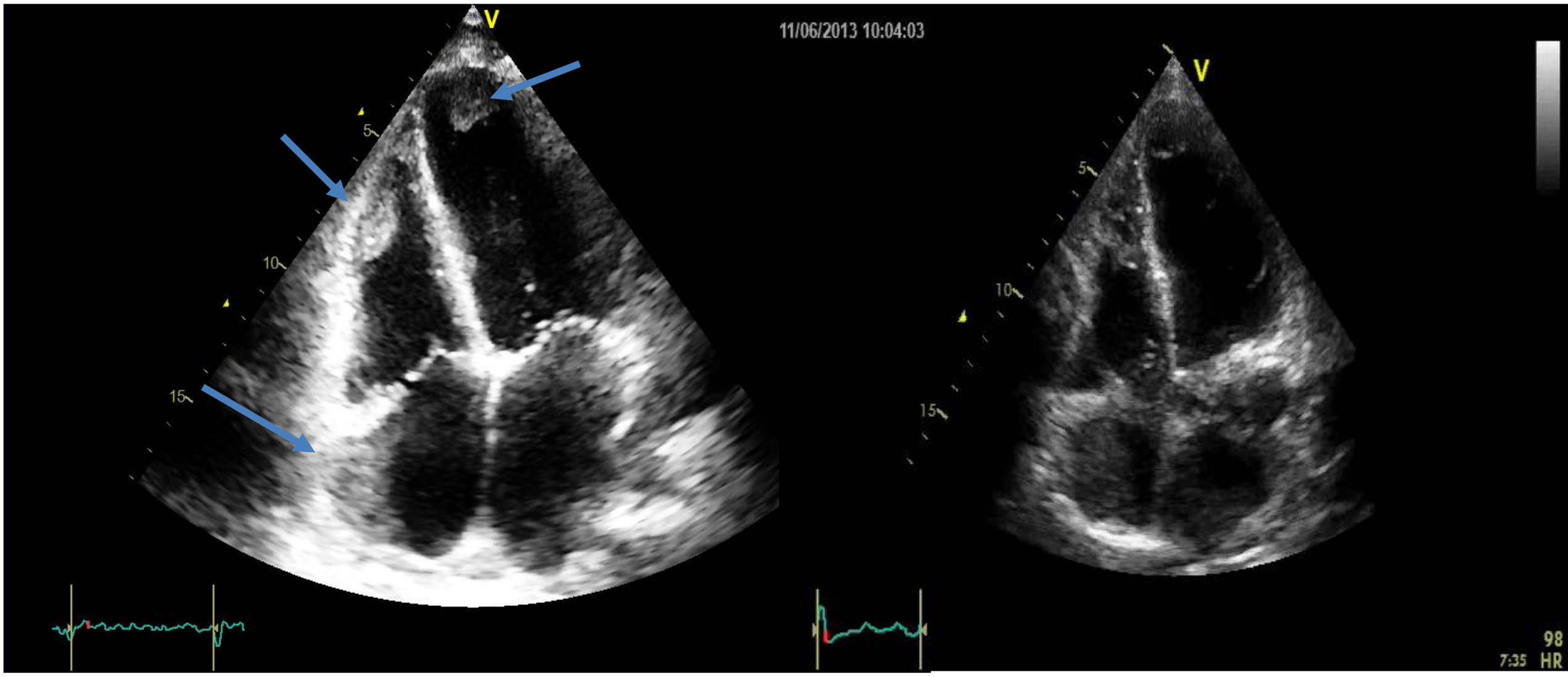
2 μήνες μετά



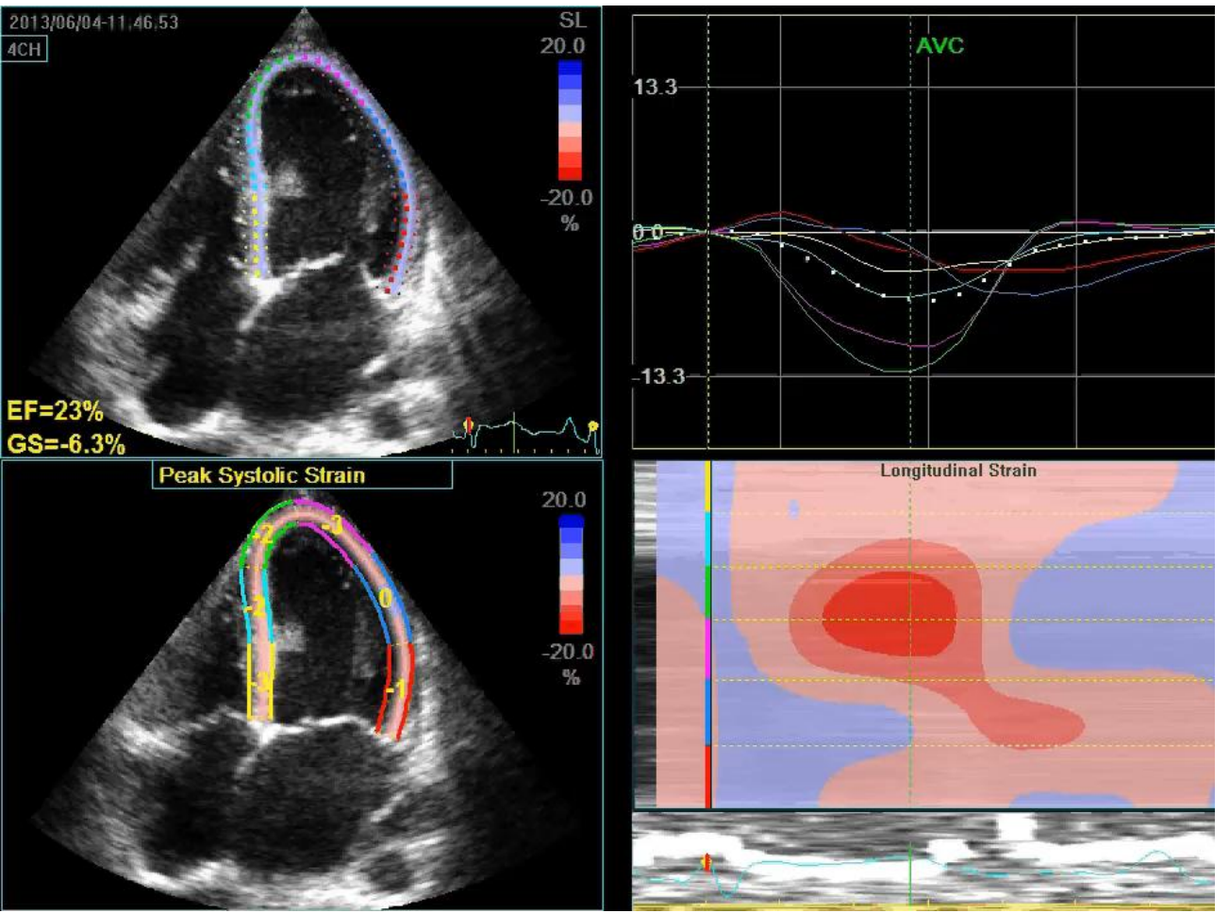



(352,304): 57
AI 26°
Axial
1,50 - PMSN-2BP403N9QX
Intera
Ex : 282304904 Se : 2101
31/07/1984, M, 3898875
Im : 4 / 5
ACC : 140566
Pos : 30,0
12/12/2008, 10:44:27
1,12 x
Rs 5°
L
16cm
TR : 4,59 ms
TE : 1,37 ms
SENSE-cardiac
ST : 10,0, SP : 10,0
W : 1689 L : 562
FOV : 40,0 x 40,0
Acq.Matr : 200 x 320
512 x 512
VIAB/4CH
HEART
P
22cm

Acute heart failure in Churg Strauss vasculitis



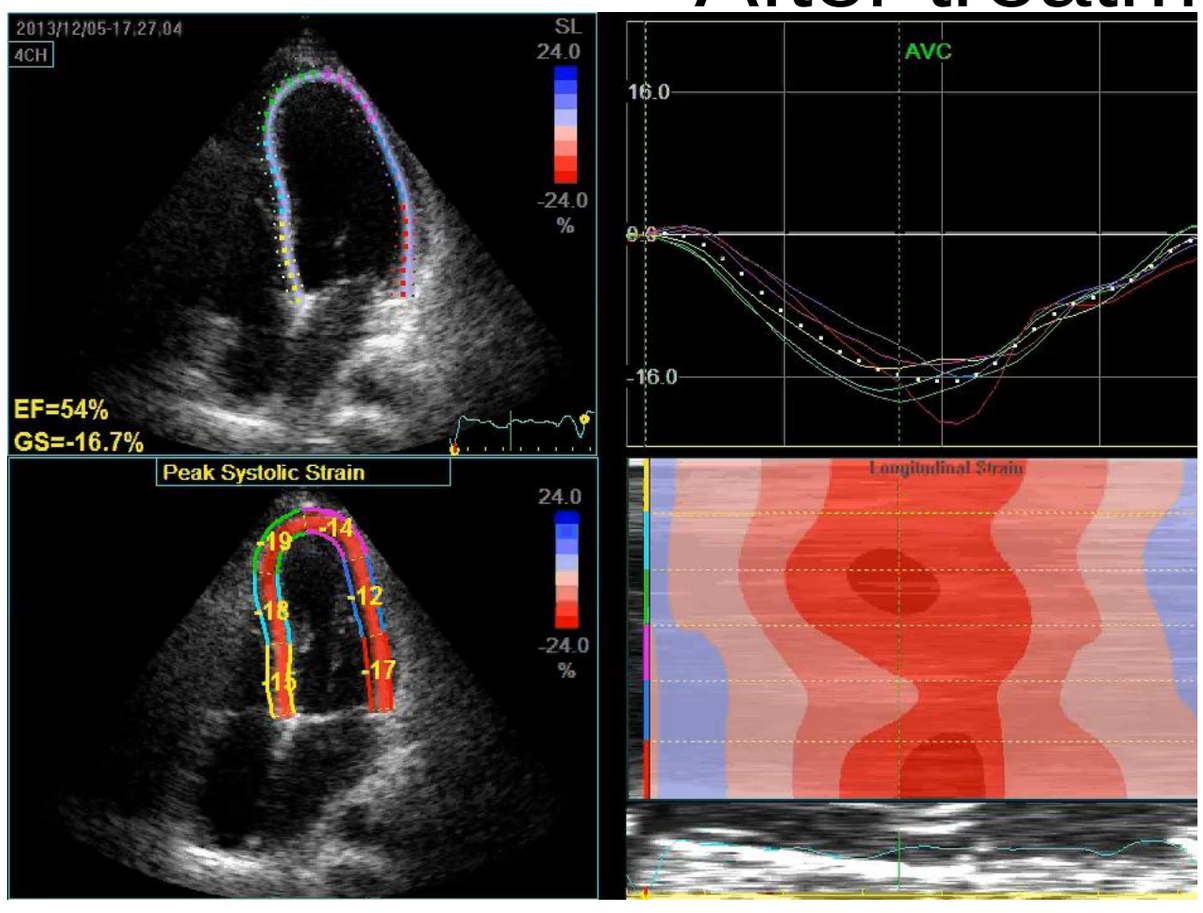
Ikonomidis et al . J Thrombosis and Thrombolysis A

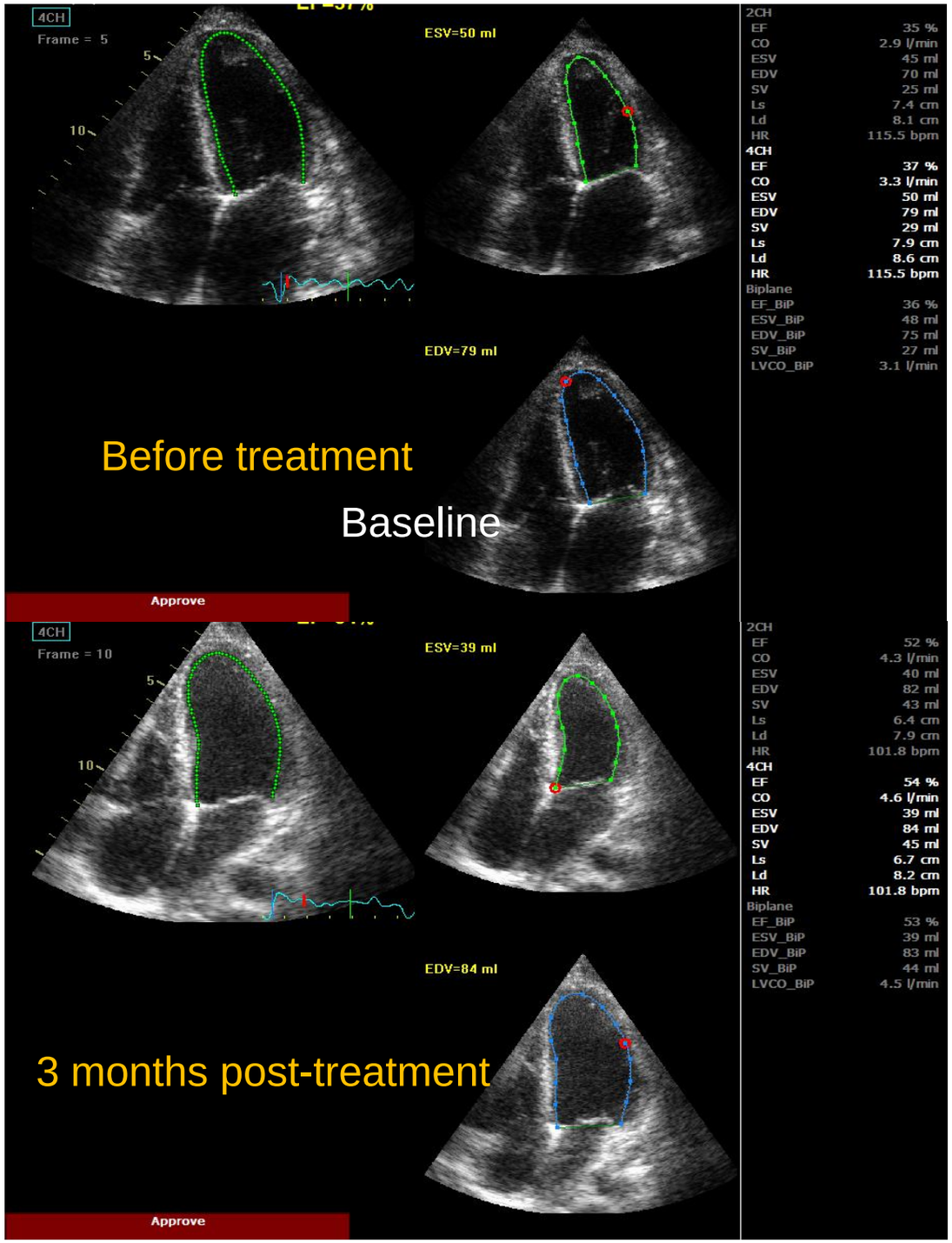


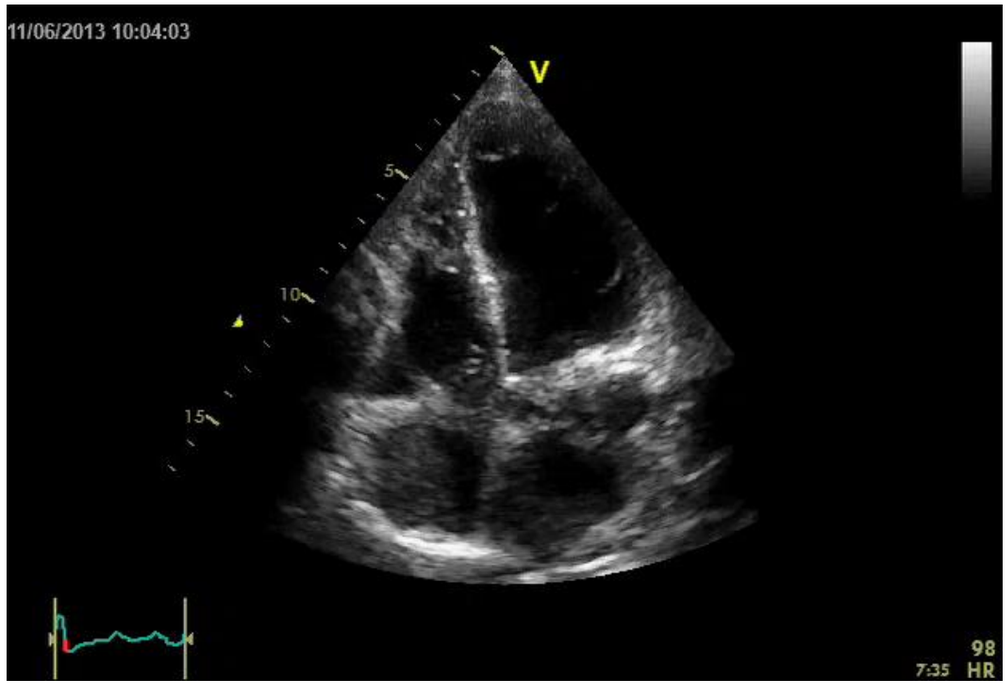
Before treatment

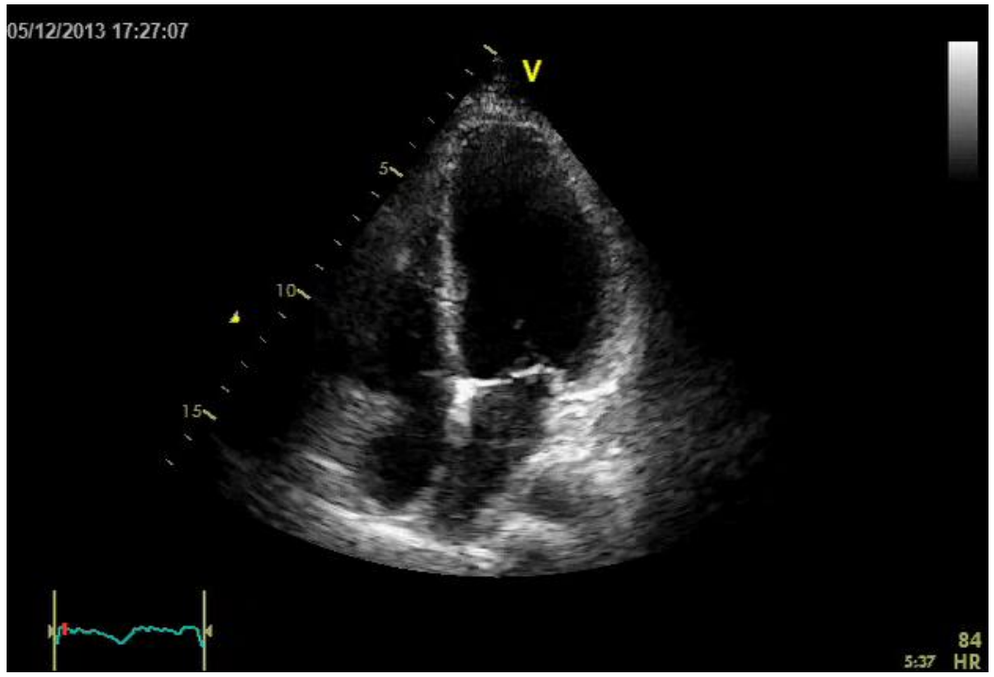
Reduction of strain in all segme

After treatme




4CH
Frame = 5
ESV=50 ml
EDV=79 ml
Before treatment
Baseline
Approve
2CH
EF 35 %
CO 2.9 l/min
ESV 45 ml
EDV 70 ml
SV 25 ml
Ls 7.4 cm
Ld 8.1 cm
HR 115.5 bpm
4CH
EF 37 %
CO 3.3 l/min
ESV 50 ml
EDV 79 ml
SV 29 ml
Ls 7.9 cm
Ld 8.6 cm
HR 115.5 bpm
Biplane
EF_BiP 36 %
ESV_BiP 48 ml
EDV_BiP 75 ml
SV_BiP 27 ml
LVCO_BiP 3.1 l/min
4CH
Frame = 10
ESV=39 ml
EDV=84 ml
3 months post-treatment
Approve
2CH
EF 52 %
CO 4.3 l/min
ESV 40 ml
EDV 82 ml
SV 43 ml
Ls 6.4 cm
Ld 7.9 cm
HR 101.8 bpm
4CH
EF 54 %
CO 4.6 l/min
ESV 39 ml
EDV 84 ml
SV 45 ml
Ls 6.7 cm
Ld 8.2 cm
HR 101.8 bpm
Biplane
EF_BiP 53 %
ESV_BiP 39 ml
EDV_BiP 83 ml
SV_BiP 44 ml
LVCO_BiP 4.5 l/min


11/06/2013 10:04:03
98
7:35 HR


05/12/2013 17:27:07
84
5:37 HR

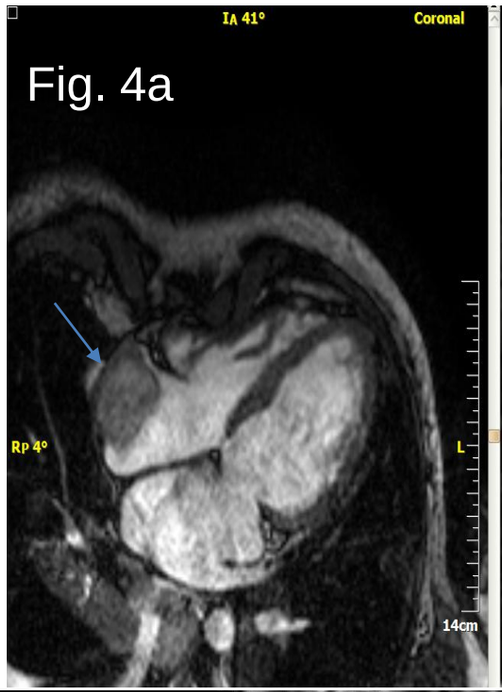
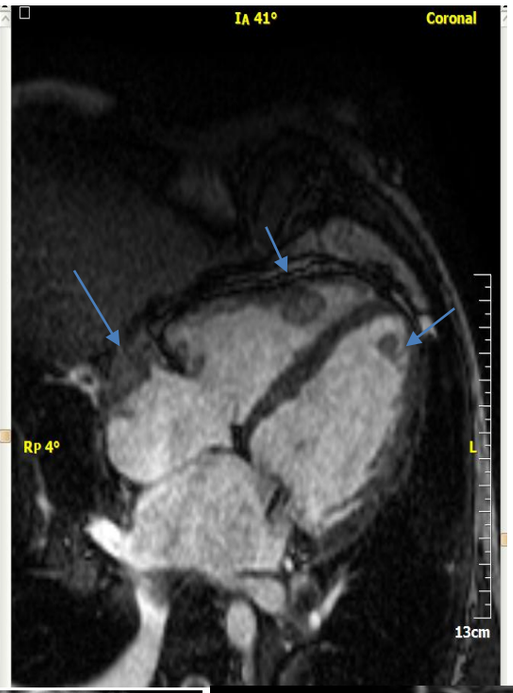

IA 41°
Coronal
RP 4°
L
14cm

Fig. 4a


IA 41°
Coronal
RP 4°
L
13cm

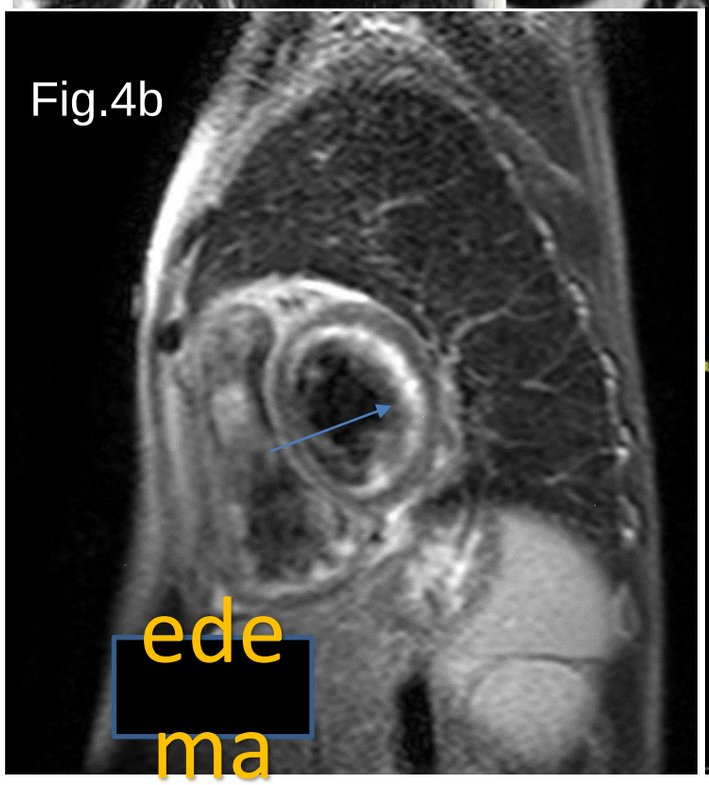


Fig.4b
edema

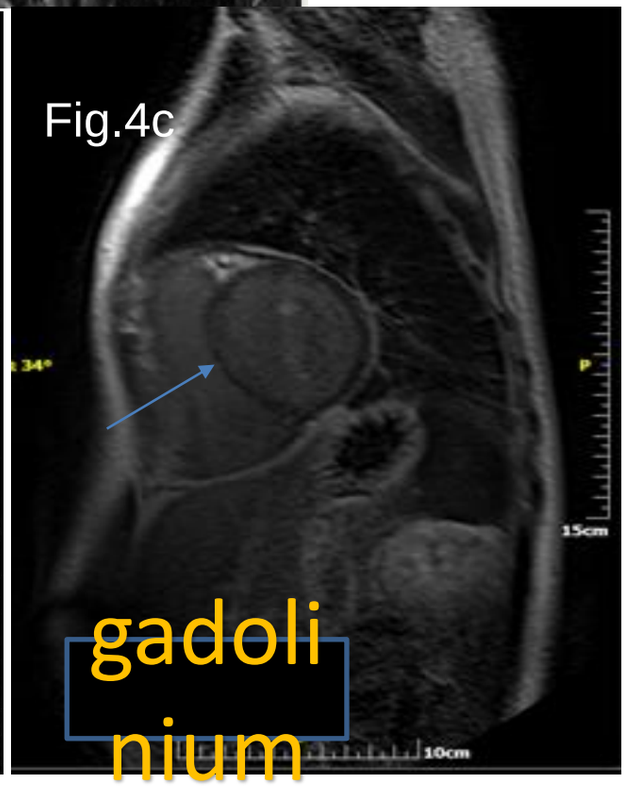


Fig.4c
gadolinium
15cm
10cm

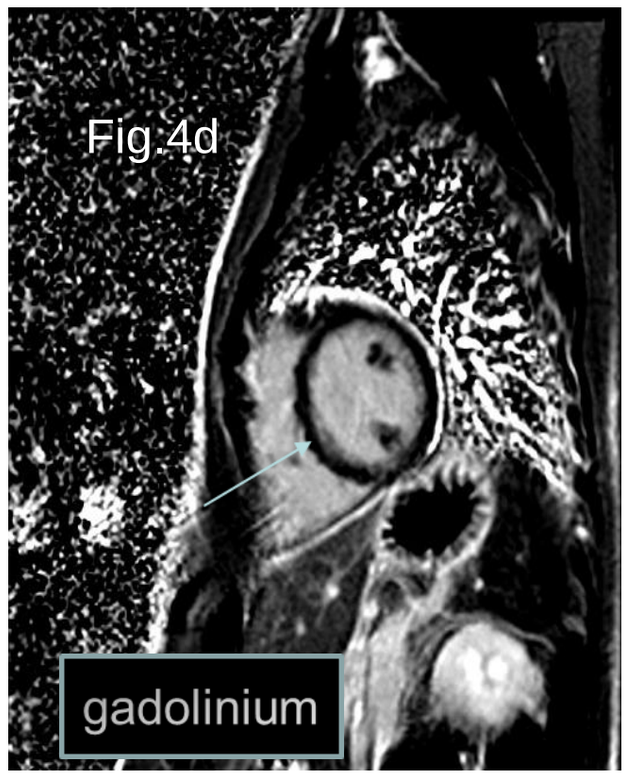


Fig.4d
gadolinium