ΟΜΑΔΑ ΕΡΓΑΣΙΑΣ ΑΠΕΙΚΟΝΙΣΤΙΚΩΝ ΤΕΧΝΙΚΩΝ, ΜΑΓΝΗΤΙΚΟΥ ΣΥΝΤΟΝΙΣΜΟΥ, ΠΥΡΗΝΙΚΗΣ ΚΑΡΔΙΟΛΟΓΙΑΣ ΚΑΙ ΑΞΟΝΙΚΗΣ ΤΟΜΟΓΡΑΦΙΑΣ ΚΑΡΔΙΑΣ

“Οι απεικονιστικές τεχνικές στη διερεύνηση κλινικών σεναρίων”

“Ασθενής με πιθανή οξεία μυοκαρδίτιδα”

ΚΩΝΣΤΑΝΤΙΝΟΣ ΤΣΟΒΟΛΑΣ
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+ / - ΗΚΓ
+ / - cTn
πρόσφατη λοίμωξη
• **Myocarditis (WHO /ISFC1):**

Inflammatory disease of the myocardium diagnosed by established histological, immunological and immunohistochemical criteria

• **histological Dallas criteria (‘80s):**

historical evidence of inflammatory infiltrates within the myocardium associated with myocyte degeneration and necrosis of non-ischaemic origin

• abnormal inflammatory infiltrate to be defined as follows:

≥14 leucocytes/mm² including up to 4 monocytes/mm² with the presence of CD 3 positive T-lymphocytes ≥7 cells/mm²
Myocarditis is a challenging diagnosis due to the heterogeneity of clinical presentations.
# Clinopathologic Classification of Myocarditis

<table>
<thead>
<tr>
<th></th>
<th>Fulminant</th>
<th>Acute</th>
<th>Chronic Active</th>
<th>Chronic Persistent</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Onset of cardiac symptoms</strong></td>
<td>Distinct</td>
<td>Indistinct</td>
<td>Indistinct</td>
<td>Indistinct</td>
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<tr>
<td><strong>LV function</strong></td>
<td>Severe dysfunction</td>
<td>Dysfunction</td>
<td>Dysfunction</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>Initial endomyocardial biopsy</strong></td>
<td>Multiple foci of active myocarditis</td>
<td>Active or borderline myocarditis</td>
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<td>Active or borderline myocarditis</td>
</tr>
<tr>
<td><strong>Clinical natural history</strong></td>
<td>Complete recovery of death</td>
<td>Incomplete recovery or dilated CM</td>
<td>Dilated CM</td>
<td>Non-CHF symptoms</td>
</tr>
<tr>
<td><strong>Histologic natural history</strong></td>
<td>Complete resolution of active myocarditis</td>
<td>Complete resolution of active myocarditis</td>
<td>Ongoing or resolving myocarditis; fibrosis; giant cells</td>
<td>Ongoing or resolving myocarditis</td>
</tr>
</tbody>
</table>

Myocarditis and Sudden Death

- \( \approx 2\% \) of infant CV sudden deaths
- \( \approx 5\% \) of childhood CV sudden deaths
- \( \approx 4-20\% \) of CV sudden deaths in athletes less than age 35-40
- Males > Females

Fabre A, Sheppard MN. Heart 2006
Maron BJ et al Circulation 2015

A recent study using ICD (9th revision) estimated the global prevalence of myocarditis to be \( \approx 22 / 100 000 \) patients annually

Lancet 2015
• ~20% of patients with myocarditis develop DCM

  D’Ambrosio A et al. Heart 2001

• Lymphocytic myocarditis accounts for around 10% of newly diagnosed adult-onset dilated cardiomyopathy

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

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1Division of Cardiology, Department of Cardiological Thoracic and Vascular Sciences, University of Padua, Padova, Italy; 2Universitätsklinikum Gießen und Marburg GmbH, Standort Marburg, Klinik für Kardiologie, Marburg, Germany; 3Academic Hospital IRCCS Foundation Policlinico, San Matteo, Pavia, Italy; 4Cardiovascular Pathology, Department of Cardiological Thoracic and Vascular Sciences, University of Padua, Padova, Italy; 5Servicio de Cardiología, Hospital U. Virgen de Arrixaca Ctra. Murcia-Cartagena s/n, El Palmar, Spain; 6Medizinische Klinik B, University of Greifswald, Greifswald, Germany; 7Department of Medicine, Heart Failure Unit, Sahlgrenska Hospital, University of Göteborg, Göteborg, Sweden; 8Division of Cardiology, Helsinki University Central Hospital, Heart & Lung Centre, Helsinki, Finland; 9Center for Heart Failure Research, Cardiovascular Research Institute, University Hospital of Maastricht, Maastricht, The Netherlands; 10Department of Internal Medicine, Medizinische Klinik und Poliklinik I, Cardiology, Wuerzburg, Germany; 11Department of Molecular Pathology, University Hospital Tübingen, Tübingen, Germany; 122nd Department of Internal Medicine, 1st School of Medicine, Charles University, Prague 2, Czech Republic; 13The Heart Hospital, University College London, UK; 14Department of Cardiology, Odense University Hospital, Odense, Denmark; 15Department of Cardiology (Heart Failure Research Center), Academic Medical Center, Amsterdam, The Netherlands; 16Department of Cardiology, Clinical Center of Serbia and Belgrade University School of Medicine, Belgrade, Serbia; 17Department of Cardiology and Pneumology, Charité Centrum 11 (Cardiovascular Medicine), Charité–Universitätsmedizin Berlin, Campus Benjamin Franklin, Berlin, Germany; 18Medizinische Klinik 1, Leopoldina Krankenhaus Schweinfurt, Schweinfurt, Germany; 19GVM Care and Research, Maria Cecilia Hospital, Cotignola, RA, Italy; 20Robert-Bosch-Krankenhaus, Stuttgart, Germany; and 21UPMC Univ Paris 6, AP-HP, Hôpital Pitié-Salpêtrière, Centre de Référence Maladies cardiovasculaires héréditaires, Paris, France

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Eur Heart J. 2013
<table>
<thead>
<tr>
<th>Causes of myocarditis/inflammatory cardiomyopathy</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1. Infectious myocarditis</strong></td>
</tr>
<tr>
<td><strong>Bacterial</strong></td>
</tr>
<tr>
<td>Staphylococcus, Streptococcus, Pneumococcus, Meningococcus, Gonococcus, Salmonella, Corynebacterium diphtheriae, Haemophilus influenzae, Mycobacterium (tuberculosis), Mycoplasma pneumoniae, Brucella</td>
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<tr>
<td><strong>Spirochaetal</strong></td>
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<tr>
<td>Borrelia (Lyme disease), Leptospira (Weil disease)</td>
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<tr>
<td><strong>Fungal</strong></td>
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<tr>
<td>Aspergillus, Actinomyces, Blastomyces, Candida, Coccioides, Cryptococcus, Histoplasma, Mucormycoses, Nocardia, Sporothrix</td>
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<tr>
<td><strong>Protozoal</strong></td>
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<tr>
<td>Trypanosoma cruzi, Toxoplasma gondii, Entamoeba, Leishmania</td>
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<tr>
<td><strong>Parasitic</strong></td>
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<tr>
<td>Trichinella spiralis, Echinococcus granulosus, Taenia solium</td>
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<tr>
<td><strong>Rickettsial</strong></td>
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<tr>
<td>Coxiella burnetii (Q fever), R. rickettsii (Rocky Mountain spotted fever), R. tsutsugamushi</td>
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<tr>
<td><strong>Viral</strong></td>
</tr>
<tr>
<td>RNA viruses: Coxsackieviruses A and B, echoviruses, polioviruses, influenza A and B viruses, respiratory syncytial virus, mumps virus, measles virus, rubella virus, hepatitis C virus, dengue virus, yellow fever virus, Chikungunya virus, Junin virus, Lassa fever virus, rabies virus, human immunodeficiency virus-1</td>
</tr>
<tr>
<td>DNA viruses: adenoviruses, parvovirus B19, cytomegalovirus, human herpes virus-6, Epstein-Barr virus, varicella-zoster virus, herpes simplex virus, variola virus, vaccinia virus</td>
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<tr>
<td><strong>2. Immune-mediated myocarditis</strong></td>
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<tr>
<td><strong>Allergens</strong></td>
</tr>
<tr>
<td>Tetanus toxoid, vaccines, serum sickness</td>
</tr>
<tr>
<td>Drugs: penicillin, cefaclor, colchicine, furosemide, isoniazid, lidocaine, tetracycline, sulfonamides, phenytoin, phenylbutazone, methyldopa, thiadiazide diuretics, amitriptyline</td>
</tr>
<tr>
<td><strong>Alloantigens</strong></td>
</tr>
<tr>
<td>Heart transplant rejection</td>
</tr>
<tr>
<td><strong>Autoantigens</strong></td>
</tr>
<tr>
<td>Infection-negative lymphocytic, infection-negative giant cell</td>
</tr>
<tr>
<td>Associated with autoimmune or immune-oriented disorders: systemic lupus erythematosus, rheumatoid arthritis, Churg-Strauss syndrome, Kawasaki’s disease, inflammatory bowel disease, scleroderma, polymyositis, myasthenia gravis, insulin-dependent diabetes mellitus, thyrotoxicosis, sarcoidosis, Wegener’s granulomatosis, rheumatic heart disease (rheumatic fever)</td>
</tr>
<tr>
<td><strong>3. Toxic myocarditis</strong></td>
</tr>
<tr>
<td><strong>Drugs</strong></td>
</tr>
<tr>
<td>Amphetamines, anthracyclines, cocaine, cyclophosphamide, ethanol, fluorouracil, lithium, catecholamines, hemetine, interleukin-2, trastuzumab, clozapine</td>
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<tr>
<td><strong>Heavy metals</strong></td>
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<tr>
<td>Copper, iron, lead (rare, more commonly cause intramyocyte accumulation)</td>
</tr>
<tr>
<td><strong>Miscellaneous</strong></td>
</tr>
<tr>
<td>Scorpion sting, snake, and spider bites, bee and wasp stings, carbon monoxide, inhalants, phosphorus, arsenic, sodium azide</td>
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<tr>
<td><strong>Hormones</strong></td>
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<tr>
<td>Phaeochromocytoma, vitamins: beri-beri</td>
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<tr>
<td><strong>Physical agents</strong></td>
</tr>
<tr>
<td>Radiation, electric shock</td>
</tr>
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Table 3  Clinical presentations of patients with biopsy-proven inflammatory heart muscle disease

(1) Acute coronary syndrome-like
   (a) Acute chest pain
      - Frequently starting within 1–4 weeks of a respiratory or gastrointestinal infection
      - Frequently associated with severe and recurrent symptoms
      - In the absence of angiographic evidence of CAD
   (b) ST/T wave changes
      - ST-segment elevation or depression
      - T-wave inversions
   (c) With or without normal global or regional LV and/or RV dysfunction on echocardiography or CMR
   (d) With or without increased TnT/TnI that may have a time course similar to acute myocardial infarction or a prolonged and sustained release over several weeks or months

(2) New onset or worsening heart failure in the absence of CAD and known causes of heart failure
   (a) New onset or progressive heart failure over 2 weeks to 3 months
      - Dyspnoea
      - Peripheral oedema
      - Chest discomfort
      - Fatigue
   (b) Impaired systolic LV and/or RV function, with or without an increase in wall thickness, with or without dilated LV and/or RV on echocardiography or CMR
   (c) Symptoms possibly started after a respiratory or gastrointestinal infection, or in the peri-partum period
   (d) Non-specific ECG signs, bundle branch block, AV-block, and/or ventricular arrhythmias

(3) Chronic heart failure in the absence of CAD and known causes of heart failure (see point 2 above)
   (a) Heart failure symptoms (with recurrent exacerbations) of >3 months duration
   (b) Fatigue, palpitation, dyspnoea, atypical chest pain, arrhythmia in an ambulant patient
   (c) Impaired systolic LV and/or RV function on echocardiography or CMR suggestive of DCM or non-ischaemic cardiomyopathy
   (d) Non-specific ECG signs, sometimes bundle branch block and/or ventricular arrhythmias and/or AV-block

(4) ‘Life-threatening condition’, in the absence of CAD and known causes of heart failure comprising
   (a) Life-threatening arrhythmias and aborted sudden death
   (b) Cardiogenic shock
   (c) Severely impaired LV function
**Table 4  Diagnostic criteria for clinically suspected myocarditis**

**Clinical presentations**
- 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. Myocardio cytosis markers
   - Elevated TnT/Tnl

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 endocardial thrombi

IV. Tissue characterization by CMR
   - Oedema and/or LGE of classical myocarditic pattern (see text)

---

Clinically suspected myocarditis if ≥1 clinical presentation and ≥1 diagnostic criteria from different categories, in the absence of (1) angiographically detectable coronary artery disease (coronary stenosis ≥ 50%); (2) known pre-existing cardiovascular disease or extra-cardiac causes that could explain the syndrome (e.g. valve disease, congenital heart disease, hyperthyroidism, etc.) (see text). Suspicion is higher with higher number of fulfilled criteria.

*If the patient is asymptomatic ≥ 2 diagnostic criteria should be met.*
Diagnosis of myocarditis

Non-invasive imaging techniques such as cardiac magnetic resonance (CMR) imaging can be useful in making the diagnosis of myocarditis and for monitoring disease progression, but we strongly endorse the concept that EMB should be the gold standard for the diagnosis of definite myocarditis.\(^1\)\(^-\)\(^3\) However, this implies that all patients with suspected myocarditis should undergo an EMB which is not routine practice; moreover, current guidelines recommend EMB only in a limited number of clinical scenarios that do not include some common
Echocardiography

Echocardiography helps to rule out non-inflammatory cardiac disease such as valve disease and to monitor changes in cardiac chamber size, wall thickness, ventricular function, and pericardial effusions. Global ventricular dysfunction, regional wall motion abnormalities, and diastolic dysfunction with preserved ejection fraction may occur in myocarditis. Histologically proven myocarditis may resemble dilated, hypertrophic, and restrictive cardiomyopathy and can mimic ischaemic heart disease. Fulminant myocarditis often presents with a non-dilated, thickened, and hypocontractile left ventricle as the intense inflammatory response results in interstitial oedema and loss of ventricular contractility. The role of newer imaging techniques such as tissue Doppler or strain-rate imaging in the diagnosis of myocarditis remains to be determined.
Nuclear imaging
Data on radionuclide evaluation, including antmyosin antibody imaging, are scarce but suggest that its sensitivity for detecting myocardial inflammation is variable and its specificity low.\textsuperscript{125–128} Due to their limited availability and risk from radiation exposure, nuclear techniques are not routinely recommended for the diagnosis of myocarditis, with the possible exception of sarcoidosis.

Thallium 201 and technetium 99m scintigraphy have been used to detect cardiac sarcoidosis but lack specificity. Gallium-67 scintigraphy and more recently positron emission tomography using 18 fluorodeoxyglucose are probably more sensitive and may be useful in the acute phase of sarcoidosis and to monitor disease progression.\textsuperscript{129–132} The detection of extracardiac disease can suggest a diagnosis of cardiac sarcoidosis.

Recommendations
4. Nuclear imaging is not routinely recommended in the diagnosis of myocarditis, with the possible exception of suspected cardiac sarcoidosis.
Heymans et al. JACC 2016
### Table 4  Diagnostic criteria for clinically suspected myocarditis

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ΡΟΛΟΣ ΤΗΣ CMR στην μυοκαρδίτιδα

1. Ανατομία
   Αριστερή ΚΑΙ δεξιά κοιλία ΚΑΙ κορυφή

2. Λειτουργία (και ήπια δυσλειτουργία)

3. Χαρακτηρισμός ιστών – παθολογία

Περικαρδιακή κοιλότητα – συλλογή (32-57% περιπτώσεων, μη ειδικό)
Cardiovascular Magnetic Resonance (CMR) has become the primary tool for noninvasive assessment of myocardial inflammation in patients with suspected myocarditis. The International Consensus Group on CMR Diagnosis of Myocarditis was founded in 2006 to achieve consensus among CMR experts and develop recommendations on the current state-of-the-art use of CMR for myocarditis. The recommendations include indications for CMR in patients with suspected myocarditis, CMR protocol standards, terminology for reporting CMR findings, and diagnostic CMR criteria for myocarditis (i.e., “Lake Louise Criteria”).

Proposed Diagnostic CMR Criteria (i.e., Lake Louise Consensus Criteria) for Myocarditis

In the setting of clinically suspected myocarditis,* CMR findings are consistent with myocardial inflammation, if at least 2 of the following criteria are present:
- Regional or global myocardial SI increase in T2-weighted images.†
- Increased global myocardial early gadolinium enhancement ratio between myocardium and skeletal muscle in gadolinium-enhanced T1-weighted images.†
- There is at least 1 focal lesion with nonischemic regional distribution in inversion recovery-prepared gadolinium-enhanced T1-weighted images (“late gadolinium enhancement”).§

A CMR study is consistent with myocyte injury and/or scar caused by myocardial inflammation if Criterion 3 is present.

A repeat CMR study between 1 and 2 weeks after the initial CMR study is recommended if
- None of the criteria are present, but the onset of symptoms has been very recent and there is strong clinical evidence for myocardial inflammation.
- One of the criteria is present.

The presence of LV dysfunction or pericardial effusion provides additional, supportive evidence for myocarditis.
Lake Louise Criteria

1. intracellular and interstitial edema – T2w imaging

2. capillary leakage – Hyperemia – T1w early gadolinium enhancement (EGE)

3. cellular necrosis / fibrosis – LGE
CMR Sensitivity Varies With Clinical Presentation and Extent of Cell Necrosis in Biopsy-Proven Acute Myocarditis

Marco Francione, MD, PhD,* Cristina Chimenti, MD, PhD,†† Nicola Galea, MD,* Fernanda Scopelliti, PhD,§ Romina Verardo, PhD,§ Roberto Galea, MD,‖ Iacopo Carbone, MD,* Carlo Catalano, MD,* Francesco Fedele, MD,‖ Andrea Frustaci, MD†§
Clinical applications of multi-parametric CMR in myocarditis and systemic inflammatory diseases

Jakub Lagan¹ ² · Matthias Schmitt¹ · Christopher A. Miller¹ ²

LLC

• sensitivity 80%
• specificity 87%
• diagnostic accuracy 83%

better diagnostic performance in “infarct-like” presentation (sensitivity of 80%) compared to heart failure (sensitivity 57%) or arrhythmias (sensitivity 40%)
Clinical applications of multi-parametric CMR in myocarditis and systemic inflammatory diseases

Jakub Lagan¹² · Matthias Schmitt¹ · Christopher A. Miller¹²

**T2w**
sensitivity 63%
specificity 76%
diagnostic accuracy 68%

**EGE**
sensitivity 66%
specificity 70%
diagnostic accuracy 67%

**LGE**
sensitivity 65%
specificity 95%
diagnostic accuracy 75%
Myocardial infarction - Myocarditis
HYPERENHANCEMENT PATTERNS

Ischemic

A. Subendocardial Infarct

B. Transmural Infarct

Nonischemic

A. Mid-wall HE

- Idiopathic Dilated Cardiomyopathy
- Myocarditis
- Right ventricular pressure overload (e.g., congenital heart disease, pulmonary HTN)
- Hypertrophic Cardiomyopathy
- Sarcoidosis
- Myocarditis
- Anderson-Fabry
- Chagas Disease

B. Epicardial HE

- Sarcoidosis, Myocarditis, Anderson-Fabry, Chagas Disease

C. Global Endocardial HE

- Amyloidosis, Systemic Sclerosis, Post cardiac transplantation
Type of virus

Extend of DE and LV systolic function

Objectives

This study sought to evaluate the long-term mortality in patients with viral myocarditis, and to establish the prognostic value of various clinical, functional, and cardiovascular magnetic resonance (CMR) parameters.

Background

Long-term mortality of viral myocarditis, as well as potential risk factors for poor clinical outcome, are widely unknown.

Methods

A total of 222 consecutive patients with biopsy-proven viral myocarditis and CMR were enrolled. A total of 203 patients were available for clinical follow-up, and 77 patients underwent additional follow-up CMR. The median follow-up was 4.7 years. Primary endpoints were all-cause mortality and cardiac mortality.

Results

We found a relevant long-term mortality in myocarditis patients (19.2% all cause, 15% cardiac, and 9.9% sudden cardiac death [SCD]). The presence of late gadolinium enhancement (LGE) yields a hazard ratio of 8.4 for all-cause mortality and 12.8 for cardiac mortality, independent of clinical symptoms. This is superior to parameters like left ventricular (LV) ejection fraction, LV end-diastolic volume, or New York Heart Association (NYHA) functional class, yielding hazard ratios between 1.0 and 3.2 for all-cause mortality and between 1.0 and 2.2 for cardiac mortality. No patient without LGE experienced SCD, even if the LV was enlarged and impaired. When focusing on the subgroup undergoing follow-up CMR, we found an initial NYHA functional class >1 as the best independent predictor for incomplete recovery (p = 0.03).

Conclusions

Among our population with a wide range of clinical symptoms, biopsy-proven viral myocarditis is associated with a long-term mortality of up to 19.2% in 4.7 years. In addition, the presence of LGE is the best independent predictor of all-cause mortality and of cardiac mortality. Furthermore, initial presentation with heart failure may be a good predictor of incomplete long-term recovery. (J Am Coll Cardiol 2012;59:1604-15) © 2012 by the American College of Cardiology Foundation.
An overview of 19 studies, all with an arrhythmic endpoint, for a total of 2692 patients, indicated that the presence and extension of myocardial fibrosis, documented by LGE, predicted VA both in ischemic and non-ischemic diseases, even in patients with only mildly depressed LVEF.
MRI for Prognosis in Myocarditis

• Clinically suspected myocarditis +LGE on baseline CMR = OR of 10.8 for appropriate ICD tx or CV death

• + LGE - risk of life threatening arrhythmias = 7%/yr

Schumm, J CV Mag Res 2014
Sanguineti et al. J CV Mag Res 2015
T2 mapping and T2* imaging in heart failure

A.S. Lota¹ · P.D. Gatehouse¹ · R.H. Mohiaddin¹
Patient with acute viral myocarditis. a Late enhancement imaging. Epicardial and mid-wall late enhancement (*green arrows*) in mid anterolateral and apical lateral segments. b T1 mapping, MOLLI sequence. Elevated T1 values in mid-wall and epicardial portion of basal—mid anterolateral and apical lateral segments (*green arrows*; T1 values in anterolateral wall: 1152 ms, T1 values in basal inferoseptum: 1031 ms). c T2 mapping, T2-prepared SFFP sequence. Elevated T2 values in epicardial portion of mid anterolateral and apical lateral segments (*green arrows*; T2 values in mid anterolateral segment: 66 ms, T2 values in basal inferoseptum: 47 ms)
Clinical applications of multi-parametric CMR in myocarditis and systemic inflammatory diseases

Jakub Lagan¹² · Matthias Schmitt¹ · Christopher A. Miller¹²

**T2 mapping**

Sensitivity 70%
Specificity 91%
Diagnostic accuracy 79%
Clinical applications of multi-parametric CMR in myocarditis and systemic inflammatory diseases

Jakub Lagan¹² · Matthias Schmitt¹ · Christopher A. Miller¹²

T1 mapping

sensitivity 82%
specificity 91%
diagnostic accuracy 86%
Multimodal assessment of myocarditis using simultaneous positron emission tomography/magnetic resonance imaging

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A 19-year-old man presented with general malaise, retrosternal chest pain, and palpitations. ECG demonstrated a flattening of T-waves in pre-cordial leads. Laboratory measurements showed elevated troponin I (6.6 ng/mL) and creatine kinase (477 U/L). Histopathological analysis of three samples taken from the right ventricle (RV) via endomyocardial biopsy (EMB) was unsuspicious, but polymerase chain reaction revealed DNA of parvovirus B19.
Using the Dallas criteria

LGE less sensitive in “borderline” myocarditis (44%) than in “active” myocarditis (84%).


**Delayed gadolinium-enhanced cardiac magnetic resonance in patients with chronic myocarditis presenting with heart failure or recurrent arrhythmias.**

The sensitivity and specificity of CMR in suspected myocarditis more than 14 days after symptom onset are poor

- Sensitivity 63%
- Specificity 40%

- T1 mapping and ECV in acute symptoms and suspected myocarditis
- only T2 mapping added significantly to LCC when assessing patients with chronic symptoms
Proposed diagnostic CMR criteria (Lake Louise Consensus Criteria) for myocarditis

In the setting of clinically suspected myocarditis\(^a\), CMR findings are consistent with myocardial inflammation, if at least two of the following criteria are present:

1. Regional or global myocardial SI increase in T2-weighted images\(^b\).

2. Increased global myocardial early gadolinium enhancement ratio between myocardium and skeletal muscle in gadolinium-enhanced T1-weighted images\(^c\).

3. There is at least one focal lesion with non-ischemic regional distribution in IR-prepared gadolinium-enhanced T1-weighted images ("late gadolinium enhancement")\(^d\).

A CMR study is consistent with myocyte injury and/or scar caused by myocardial inflammation, if

- criterion 3 is present.

A repeat CMR study between 1 and 2 weeks after the initial CMR study is recommended, if

- none of the criteria are present, but the onset of symptoms has been very recent and there is strong clinical evidence for myocardial inflammation.

- one of the criteria is present.

The presence of LV dysfunction or pericardial effusion provides additional, supportive evidence for myocarditis.
ΣΥΜΠΕΡΑΣΜΑΤΑ

- Η μυοκαρδίτιδα είναι φλεγμονώδης καρδιακή πάθηση με πολύπλοκη κλινική εικόνα και πιθανώς απρόσμενη εξέλιξη.

- Απεικονιστικές τεχνικές ειδικές στην καταγραφή φλεγμονής και νέκρωσης.

- Σημαντικός ο ρόλος της CMR στη διάγνωση της οξείας μυοκαρδίτιδας.

- T2, EGE και LGE αυξάνουν τη διαγνωστική ικανότητα.

- CMR σημαντική στην πρόγνωση μέσω LGE.

- Νεότερες τεχνικές – ακολουθίες σε προοπτική.

- Μειωμένη ακρίβεια μετά τις 14 ημέρες.

- Πυρηνική ιατρική - PET - σαρκοείδωση.

- EMB οριστική διάγνωση.