

Myokarditis

Pleva Martin, Borová Júlia

Nemocnice Podlesí, a.s., Třinec

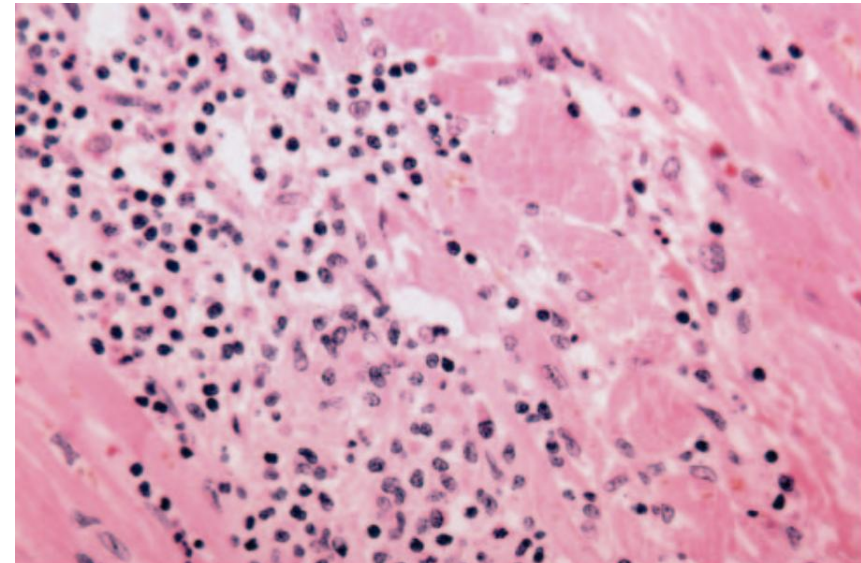
Vítkovická nemocnice, a.s., Ostrava

Akutní myokarditida

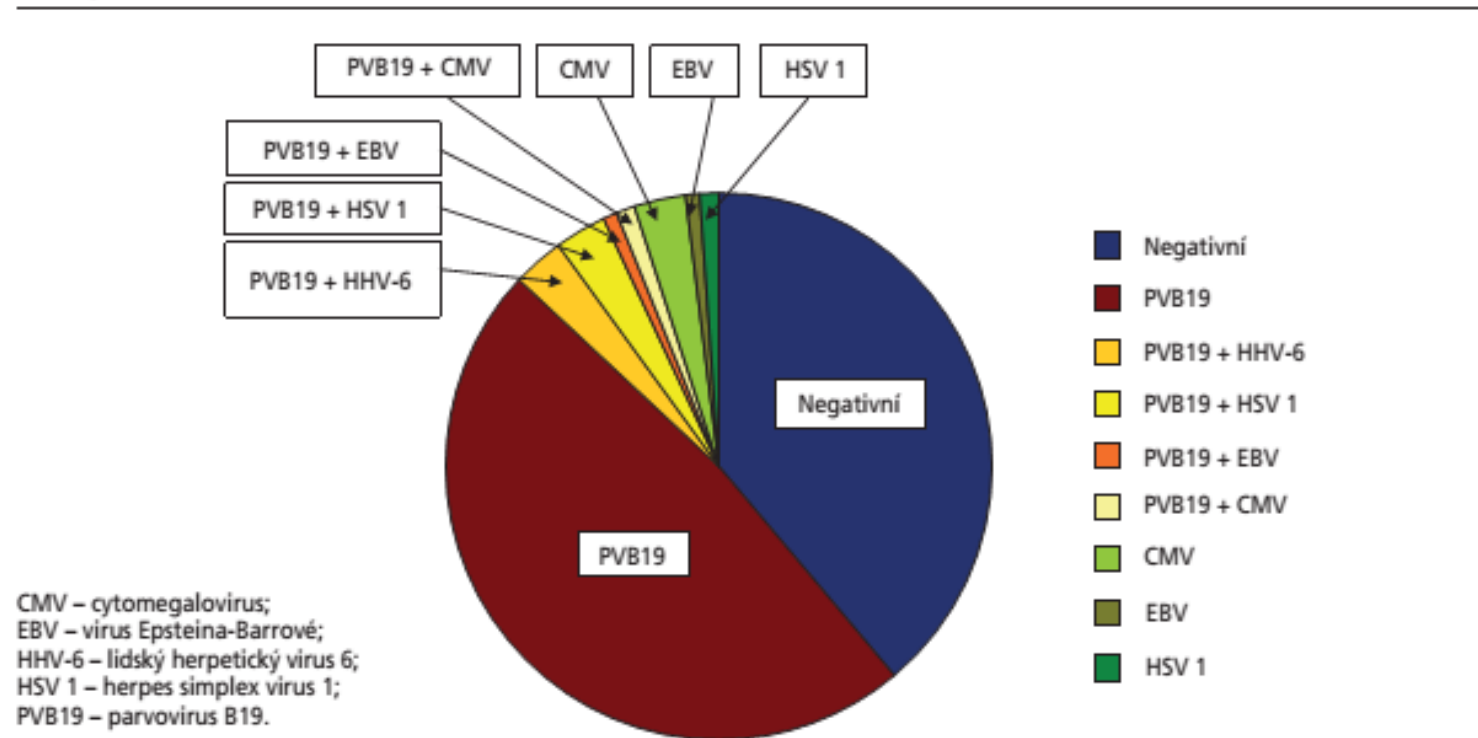
- akutní zánětlivé postižení myokardu
- zánětlivá KMP – dilatační KMP vzniklá v souvislosti s myokarditidou (myokarditida + porucha systolické funkce)

Etiologie:

- infekční
- autoimunní
- toxické poškození



Etiologie



Obr. 1 – PCR nález se zastoupením jednotlivých agens

Etiologie

[Med Microbiol Immunol](#). 2010 May;199(2):139-43. doi: 10.1007/s00430-009-0141-6. Epub 2010 Jan 6.

Presence of *Borrelia burgdorferi* in endomyocardial biopsies in patients with new-onset unexplained dilated cardiomyopathy.

[Palecek T¹](#), [Kuchynka P](#), [Hulinska D](#), [Schramlova J](#), [Hrbackova H](#), [Vitkova I](#), [Simek S](#), [Horak J](#), [Louch WE](#), [Linhart A](#).

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Abstract

Dilated cardiomyopathy (DCM) represents the third most common cause of heart failure and the most frequent cause of heart transplantation. Infectious, mostly viral, and autoimmune mechanisms, together with genetic abnormalities, have been reported as three major causes of DCM. We hypothesized that Lyme disease (LD), caused by spirochete *Borrelia burgdorferi* (Bb), might be an important cause of new-onset unexplained DCM in patients living in a highly endemic area for LD such as the Czech Republic. We performed endomyocardial biopsy (EMB) in 39 consecutive patients presenting with symptomatic unexplained left ventricular (LV) systolic dysfunction lasting no more than 12 months. In eight subjects (21%), Bb was detected in the EMB sample by polymerase chain reaction or by electron microscopy. None of these patients exhibited any form of atrioventricular block or other extracardiac manifestation of Bb infection. Serological testing identified IgG antibodies against Bb in only two cases and IgM antibodies in none. All affected patients were treated with intravenous ceftriaxone for 3 weeks. At 6 months follow-up, LV morphology and function as well as functional status of these patients significantly improved. In conclusion, Bb infection may represent an important cause of new-onset unexplained DCM in patients living in endemic regions such as the Czech Republic. Because the antibiotic treatment appears to be markedly effective and serological examination does not provide a tool for diagnosing the disease, EMB focused on the detection of Bb should be performed in all patients from endemic areas with new-onset unexplained DCM not responding to conventional therapy.

Klinická prezentace

- subklinická forma
- „AKS – like“ průběh – bolest na hrudi s pozitivitou troponinu (\pm ekg změny)
- nově vzniklé srdeční selhání
- život ohrožující arytmie
- kardiogenní šok

Diagnostika

EMB jako zlatý standard

- histologie a imunohistochemie – posouzení zánětlivé infiltrace myokardu
- PCR – přítomnost infekčního agens

CMR jako „zlatý neinvazivní“ standard

- přítomnost zánětlivých změn bez možnosti určení příčiny

Table 4 Definition of clinically suspected myocarditis according to the ESC 2013 Myocarditis Task Force³⁶

Presence of ≥ 1 clinical presentation and ≥ 1 diagnostic criteria from different categories, in the absence of:

- (1) Angiographically detectable CAD (coronary stenosis $\geq 50\%$)
- (2) Known pre-existing cardiovascular disease or extra-cardiac causes that could explain the syndrome (e.g. valve disease, congenital heart disease, etc.).
- (3) If the patient is asymptomatic ≥ 2 diagnostic criteria should be met.

Diagnosis of certainty and aetiologic diagnosis of myocarditis requires EMB (histology, immunohistology, infectious agents by PCR)

Clinical presentations

Acute coronary syndrome-like, with or without normal global or regional left ventricular (LV) and/or right ventricular (RV) dysfunction on echocardiography or CMR, with or without increased troponin (Tn)T/TnI (that may have a time course similar to AMI or a prolonged and sustained release over several weeks or months).

New onset or worsening unexplained heart failure.

Chronic unexplained heart failure of >3 months duration.

Life-threatening unexplained condition (including life-threatening arrhythmias and aborted sudden death, cardiogenic shock, severely impaired left ventricular function).

Diagnostic criteria

- (1) 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, 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, and supraventricular tachycardia
- (2) Myocardial injury markers
elevated cardiac troponins
- (3) Functional and structural abnormalities on cardiac imaging (echo/angio/CMR)
new, otherwise unexplained LV and/or RV structure and function abnormality.
- (4) Tissue characterization by CMR
oedema and/or LGE of classical myocarditic pattern (according to Lake-Louise criteria).³⁷

Zobrazovací metody

- **ECHO**
- **SKG + LVG**
- CT
- **CMR**
- FDG/PET CT (sarkoidóza)

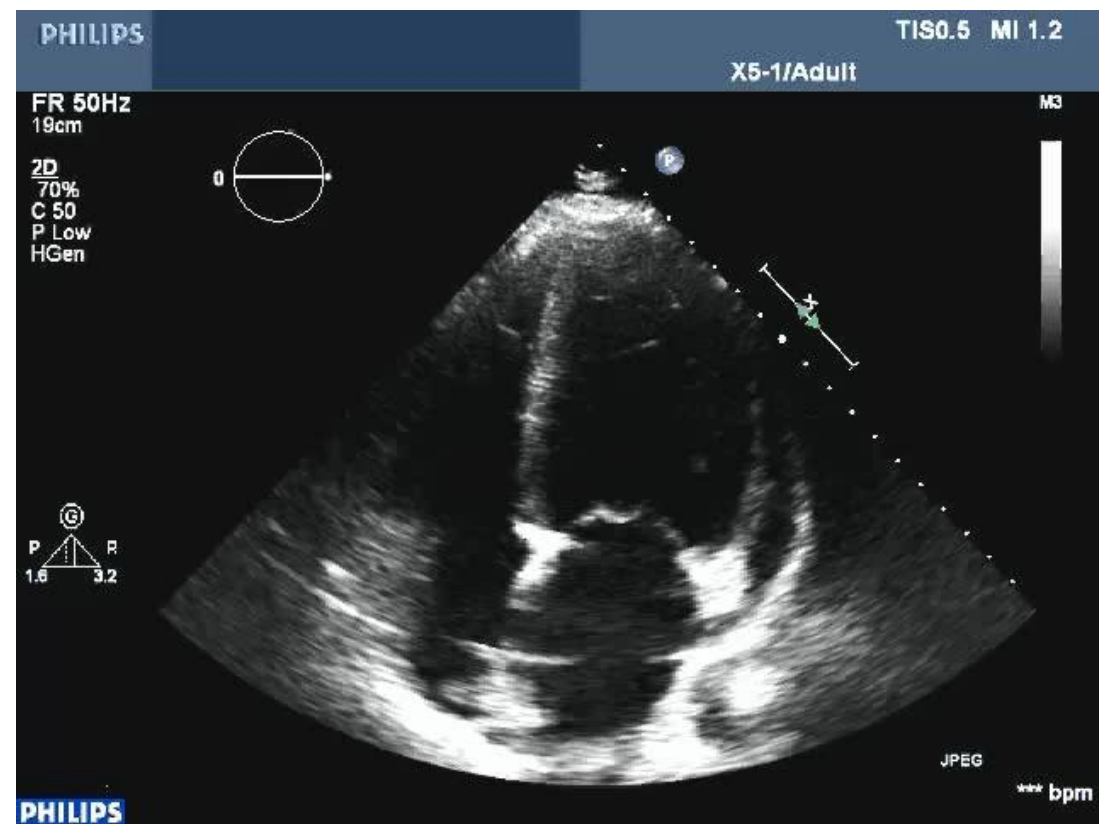
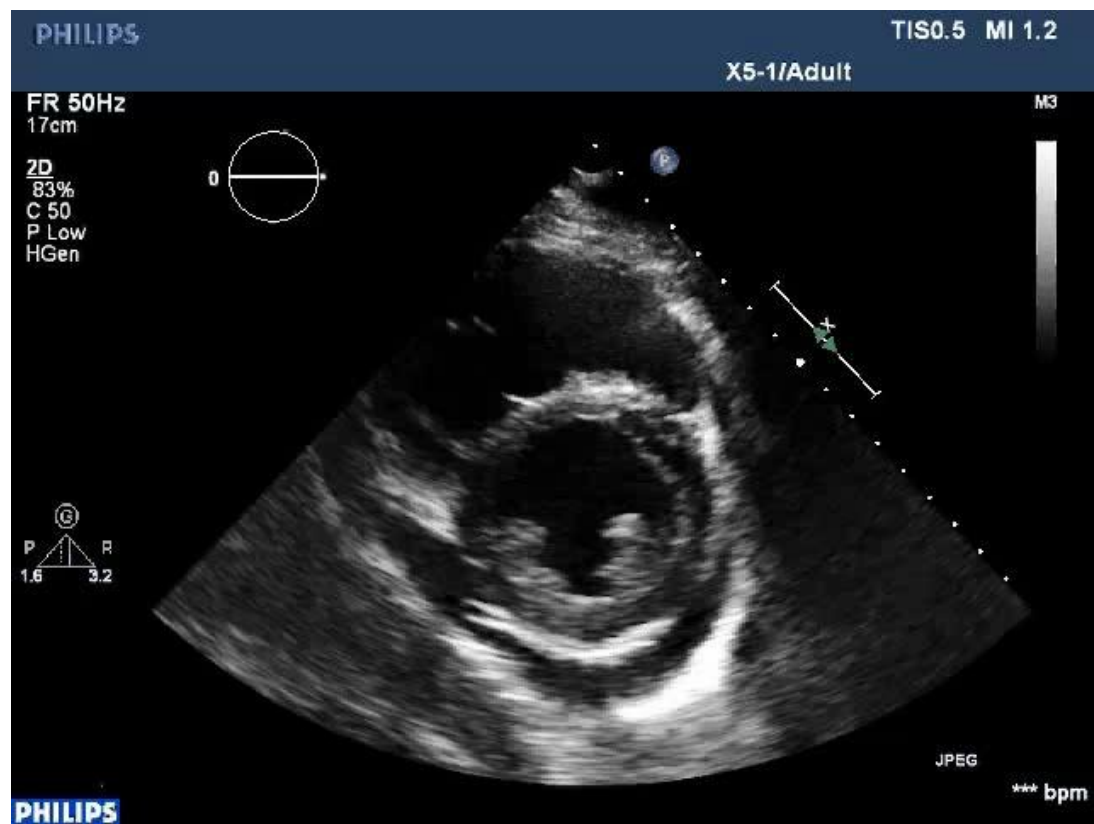
ECHO

- prováděno vždy
- bedside metoda

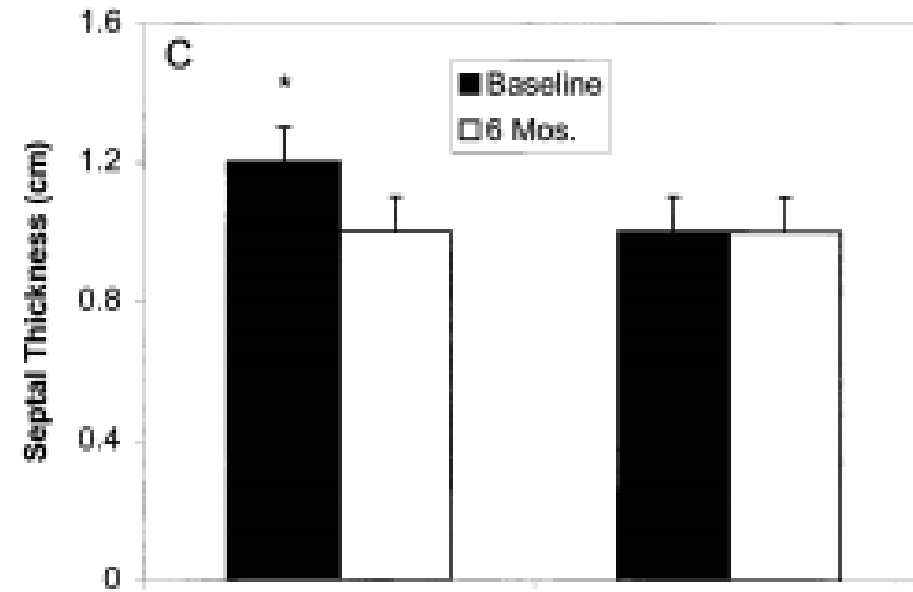
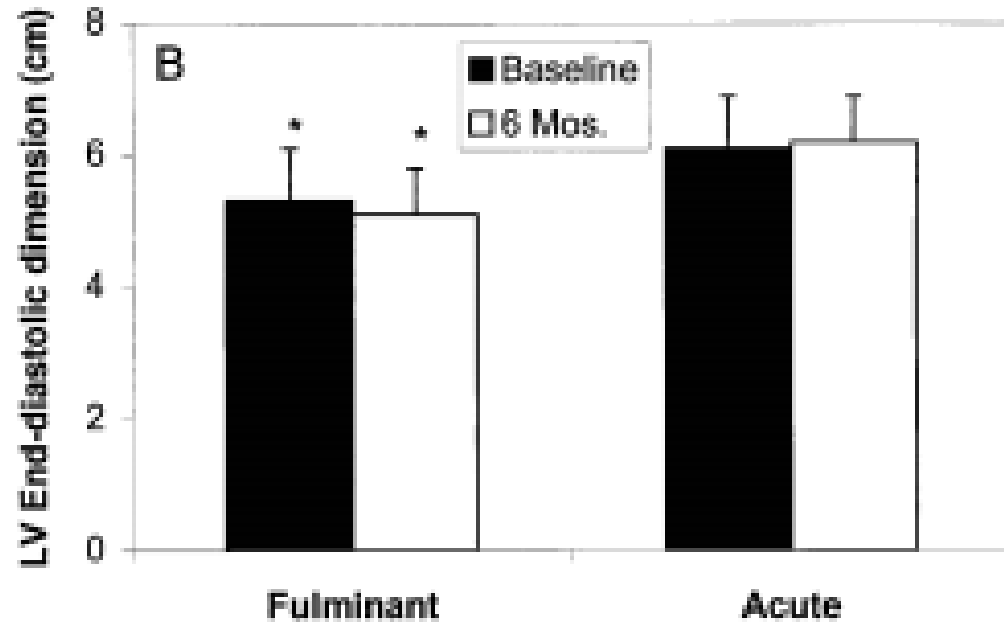
Nespecifické nálezy:

- systolická dysfunkce LK/obou komor
- regionální porucha kinetiky
- ztluštění stěn LK
- perikardiální výpotek
- normální nález

Kardiogenní šok



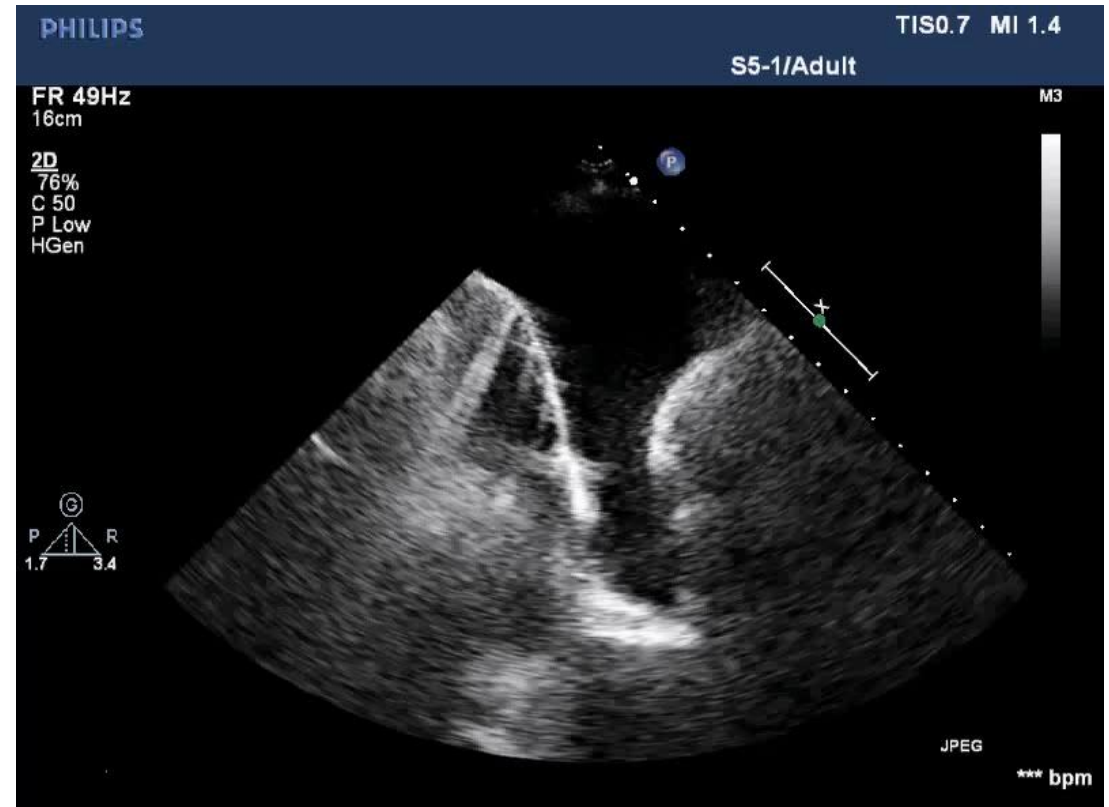
ECHO



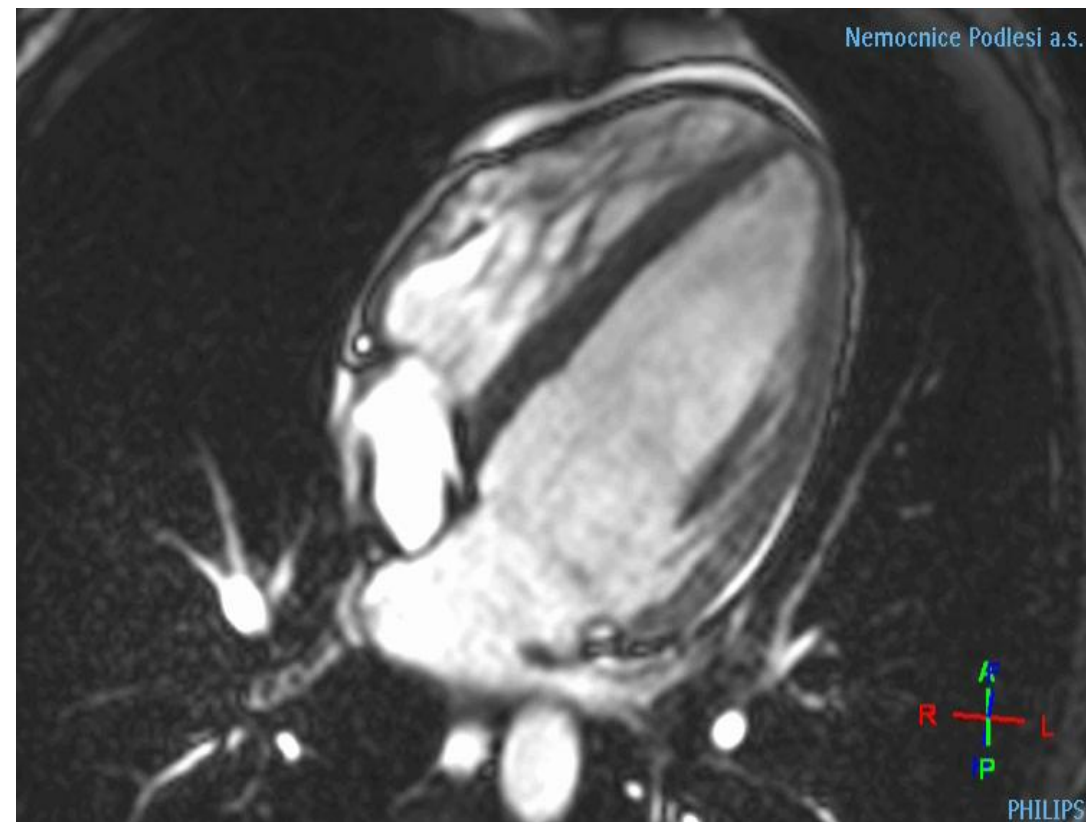
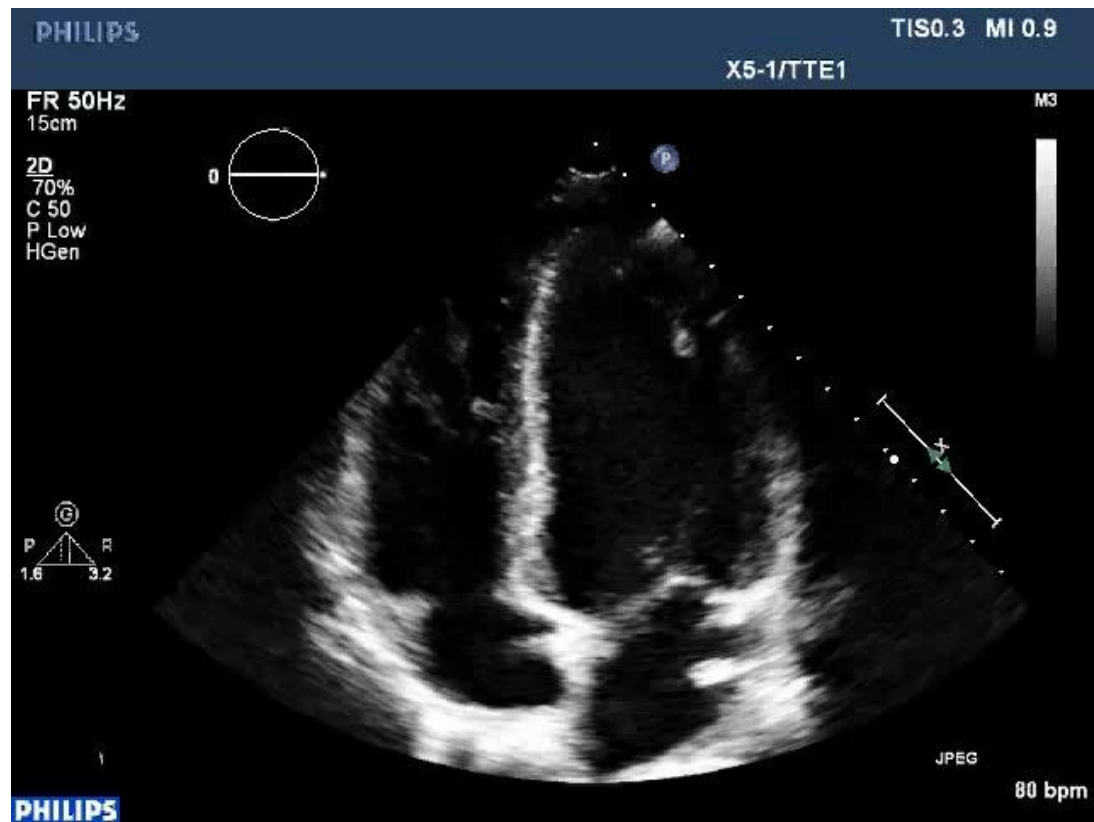
ECHO

Monitorace a průběh:

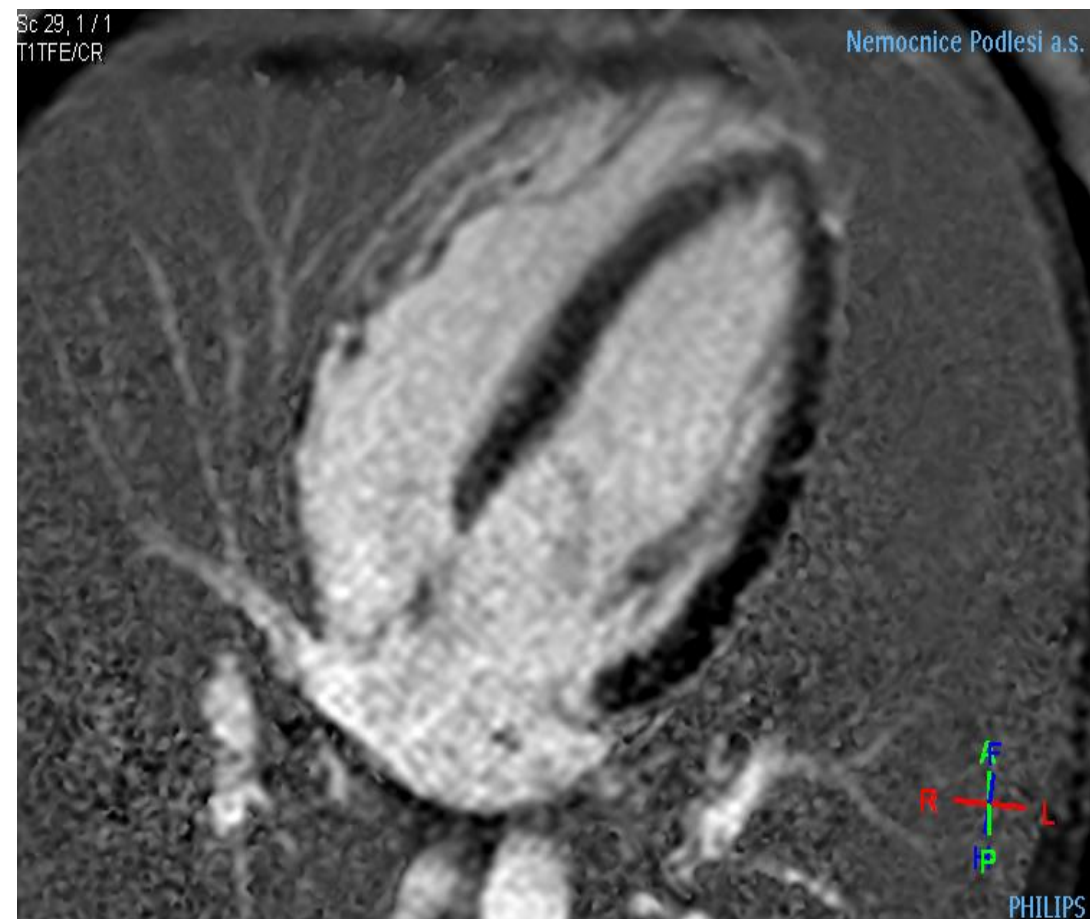
- systolické funkce komor
- odhad plnicích tlaků a CO
- odhad závažnosti plicní hypertenze
- perikardiální/pleurální výpotky
- USG plic



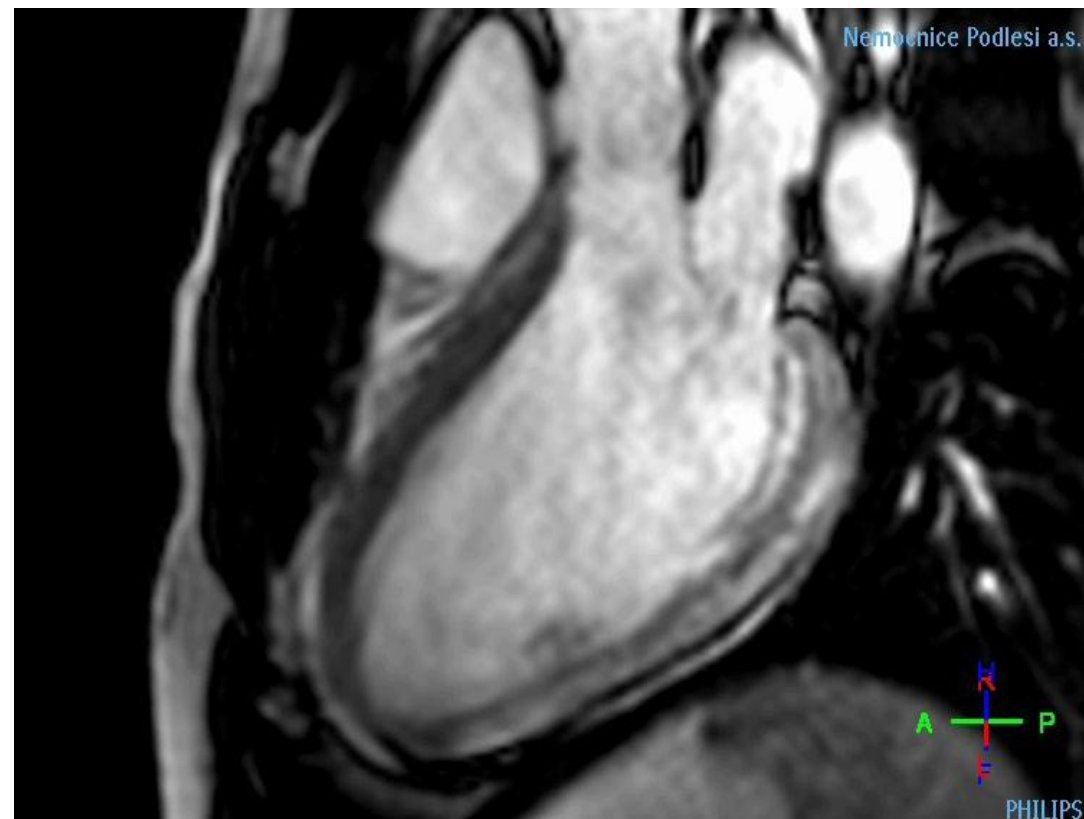
Systolická dysfunkce LK



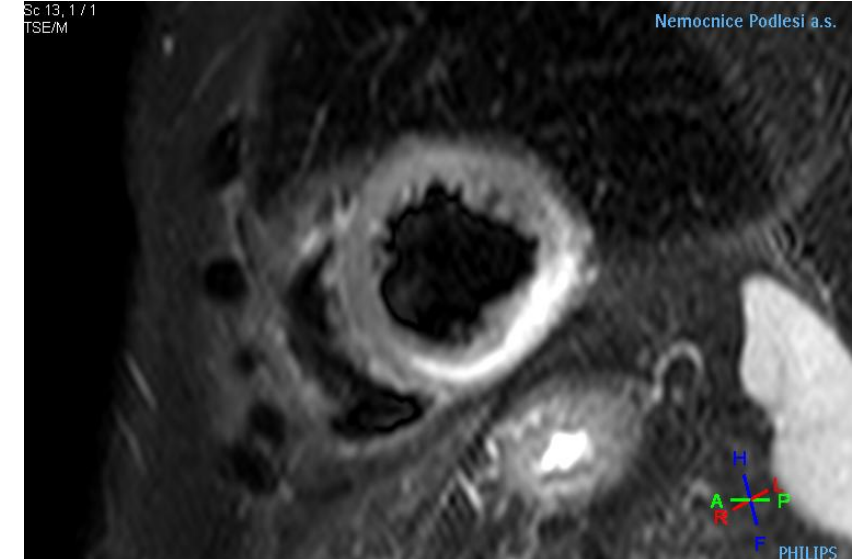
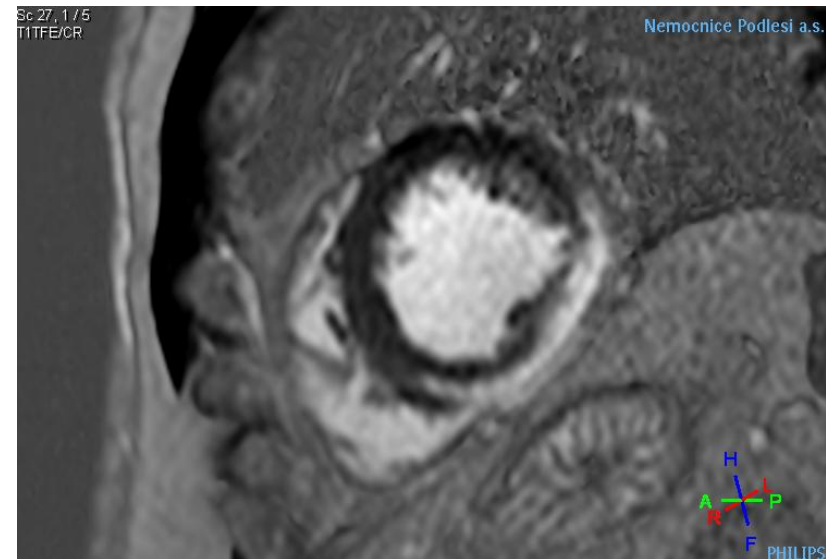
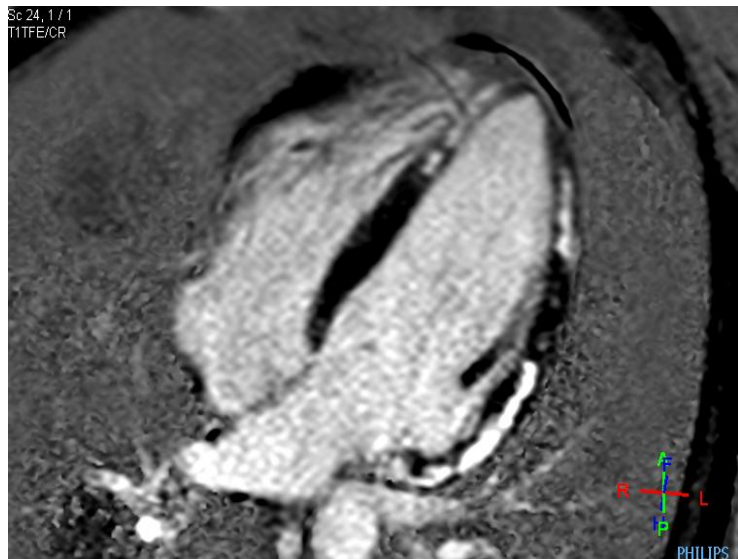
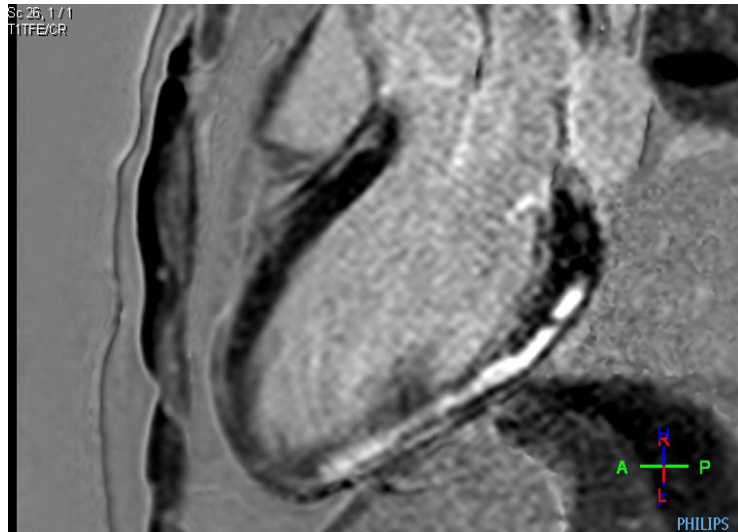
Systolická dysfunkce LK



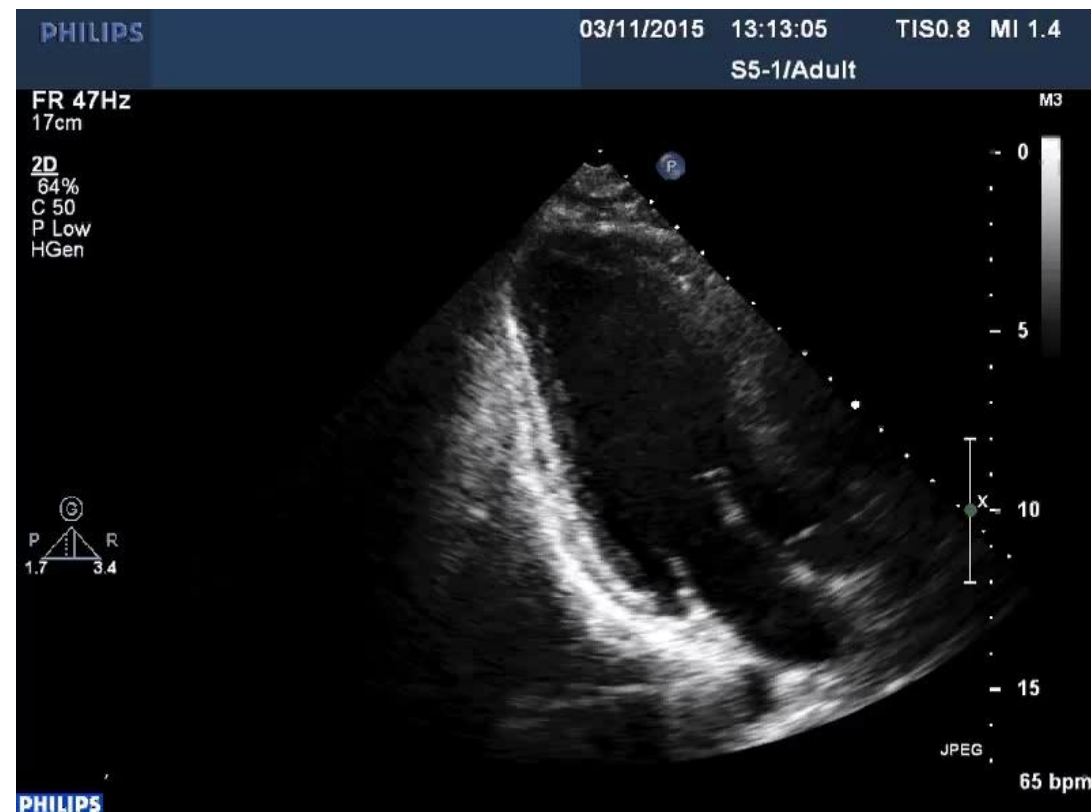
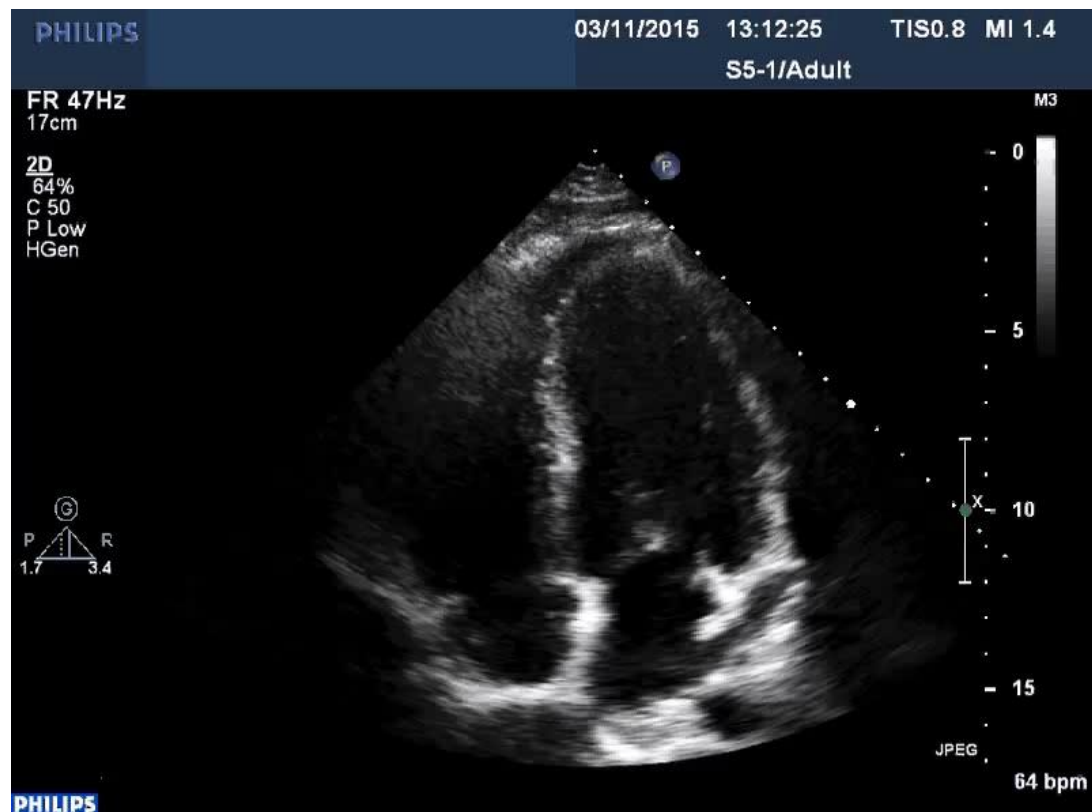
Lokální porucha kinetiky



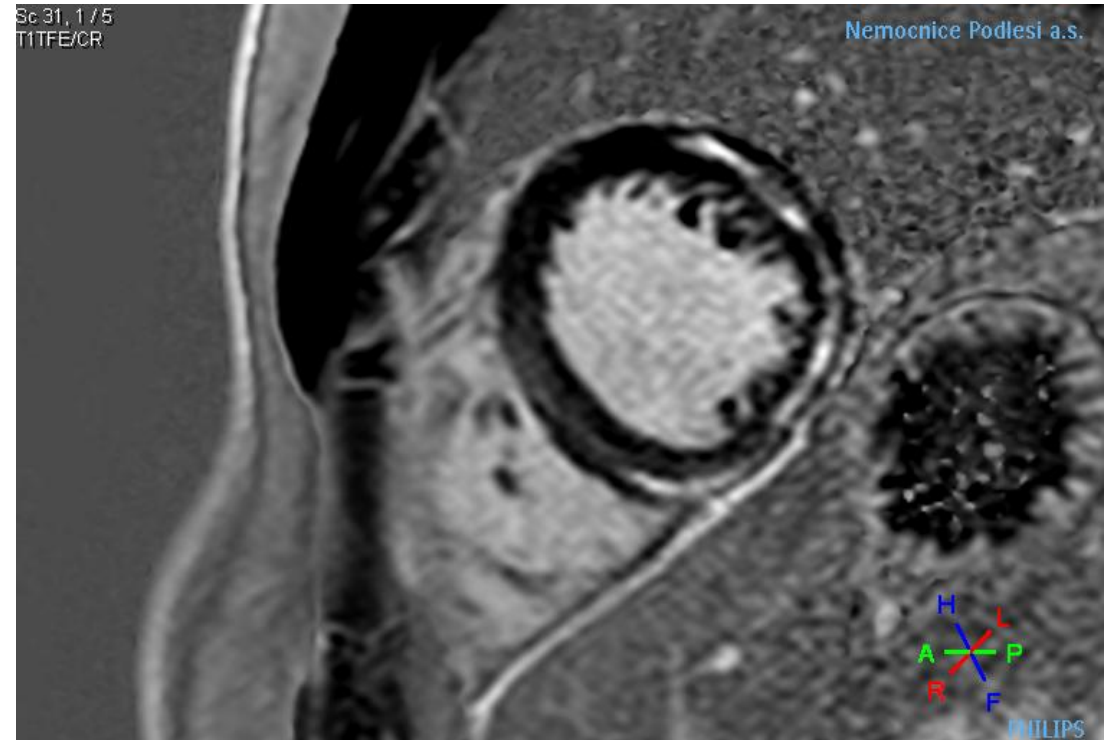
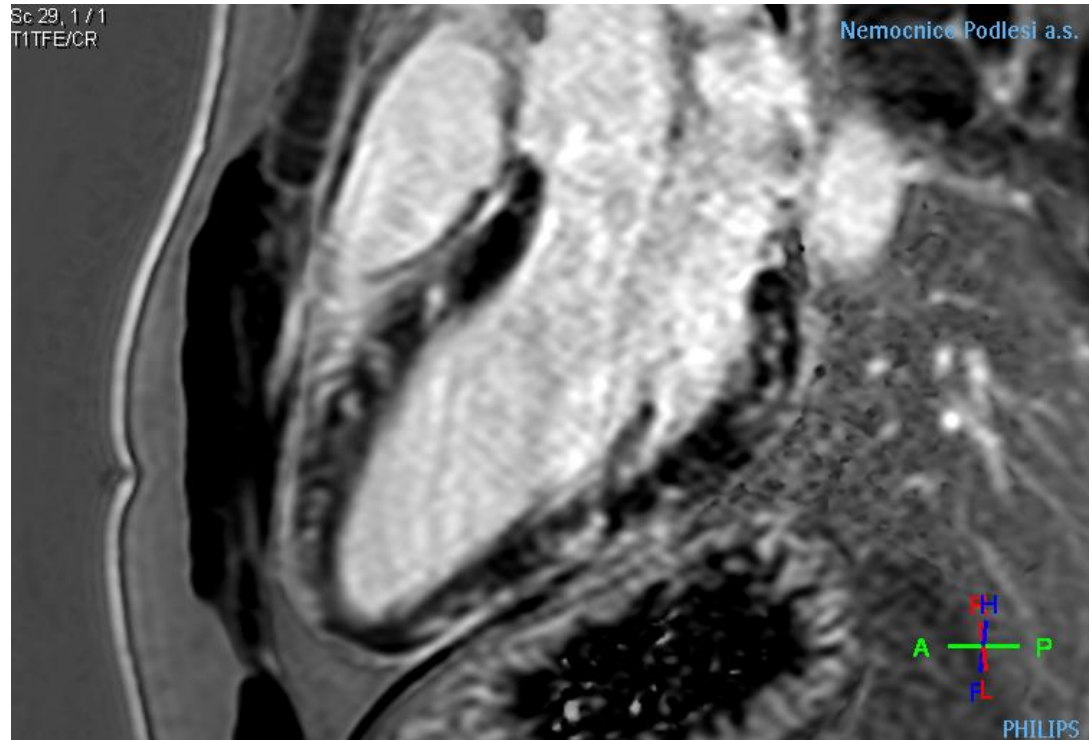
Lokální porucha kinetiky



Normální funkce



Normální funkce



Myocardial oedema in acute myocarditis detected by echocardiographic 2D myocardial deformation analysis

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Aims

The clinical diagnosis of acute myocarditis is based on symptoms, electrocardiography, elevated myocardial necrosis biomarkers, and echocardiography. Often, conventional echocardiography reveals no obvious changes in global cardiac function and therefore has limited diagnostic value. Myocardial deformation imaging by echocardiography is an evolving method used to characterize quantitatively longitudinal systolic function, which may be affected in acute myocarditis. The aim of our study was to assess the utility of echocardiographic deformation imaging of the left ventricle in patients with diagnosed acute myocarditis in whom cardiovascular magnetic resonance (CMR) evaluation was performed.

Methods and results

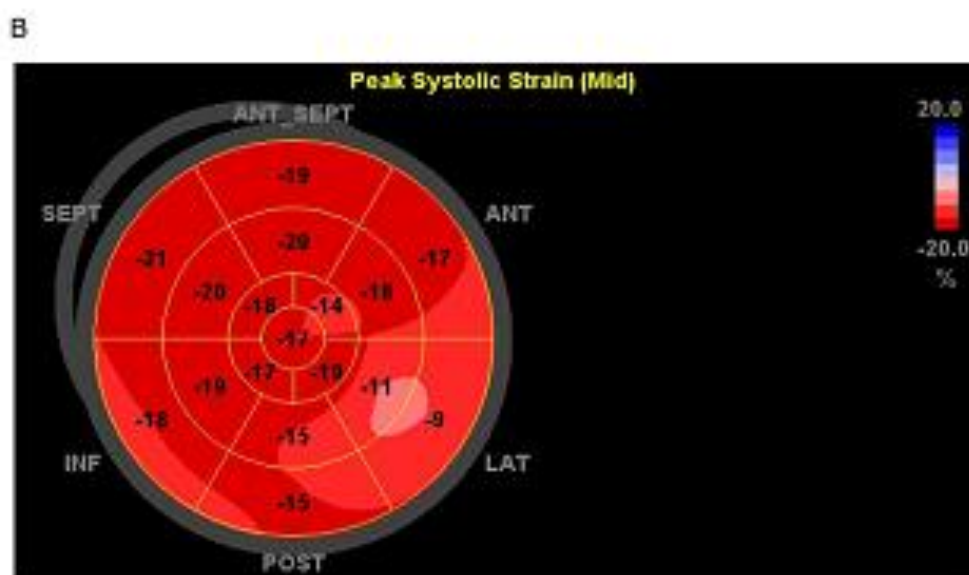
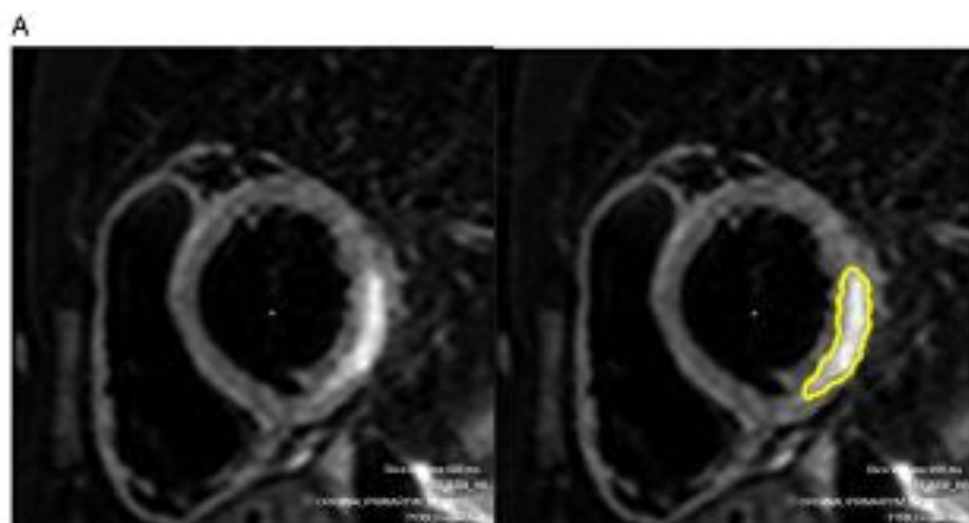
We included 28 consecutive patients (mean age 32 ± 13 years) with CMR-verified diagnosis of acute myocarditis according to the Lake Louise criteria. Cardiac function was evaluated by a comprehensive assessment of left ventricular (LV) function, including 2D speckle-tracking echocardiography. We found no significant correlation between the peak values of cardiac enzymes and the amount of myocardial oedema assessed by CMR (troponin: $r = 0.3$; $P = 0.05$ and CK-MB: $r = 0.1$; $P = 0.3$). We found a larger amount of myocardial oedema in the basal part of the left ventricle [American Heart Association (AHA) segments 1–6] in inferolateral and inferior segments, compared with the anterior, anterolateral, antero-septal, and inferoseptal segments. In the mid LV segments (AHA segments 7–12), this was more pronounced in the anterior, anterolateral, and inferolateral segments. Among conventional echocardiographic parameters, LV function was not found to correlate with the amount of myocardial oedema of the left ventricle. In contrast, we found the wall motion score index to be significantly correlated with the amount of myocardial oedema, but this correlation was only present in patients with an extensive amount of oedema ($> 11\%$ of the total left ventricle). Global longitudinal systolic myocardial strain correlated significantly with the amount of oedema ($r = 0.65$; $P < 0.001$). We found that both the epicardial longitudinal and the endocardial longitudinal systolic strains were significantly correlated with oedema ($r = 0.55$; $P = 0.003$ and $r = 0.54$; $P < 0.001$).

Conclusion

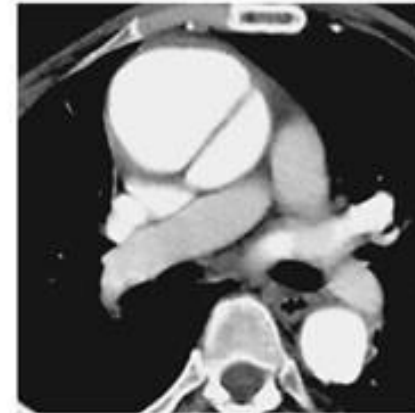
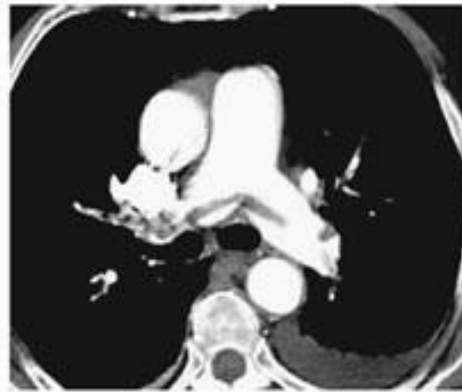
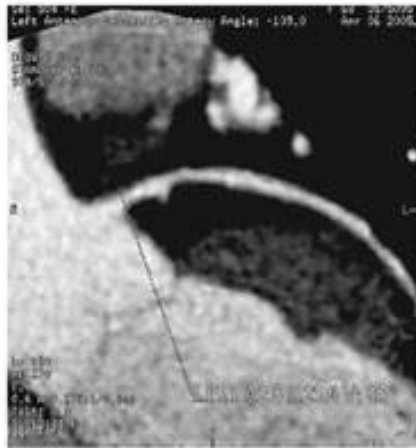
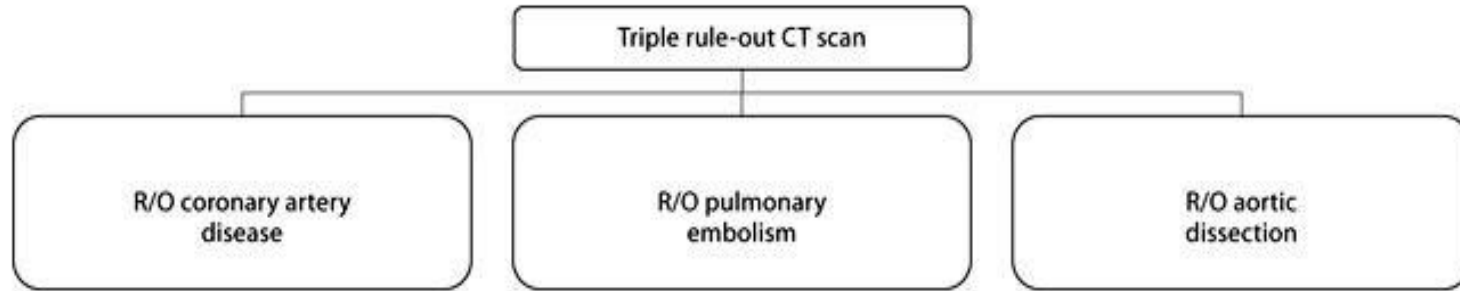
In patients with acute myocarditis, 2D speckle-tracking echocardiography was a useful tool in the diagnostic process of acute myocarditis. Global longitudinal strain adds important information that can support clinical and conventional echocardiographic evaluation, especially in patients with preserved LV ejection fraction in relation to the diagnosis and degree of myocardial dysfunction.

Keywords

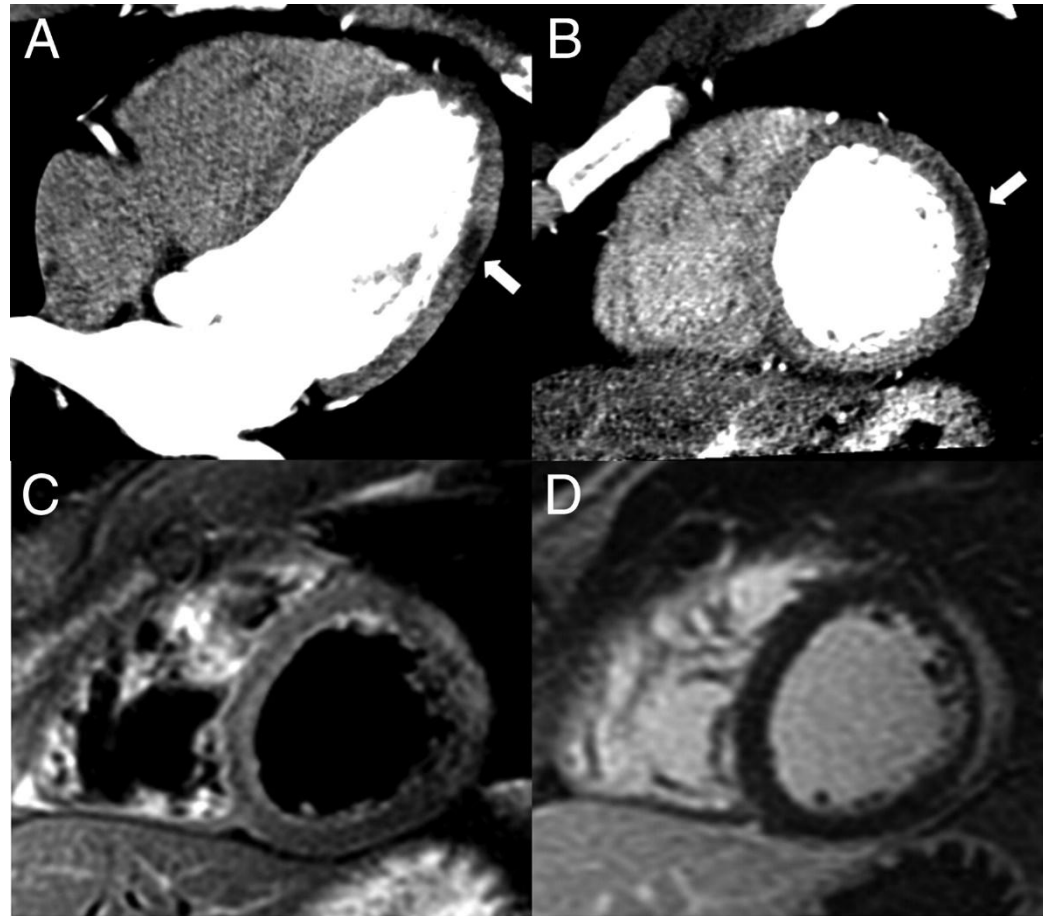
myocarditis • cardiovascular magnetic resonance • speckle tracking • left ventricular function • myocardial oedema



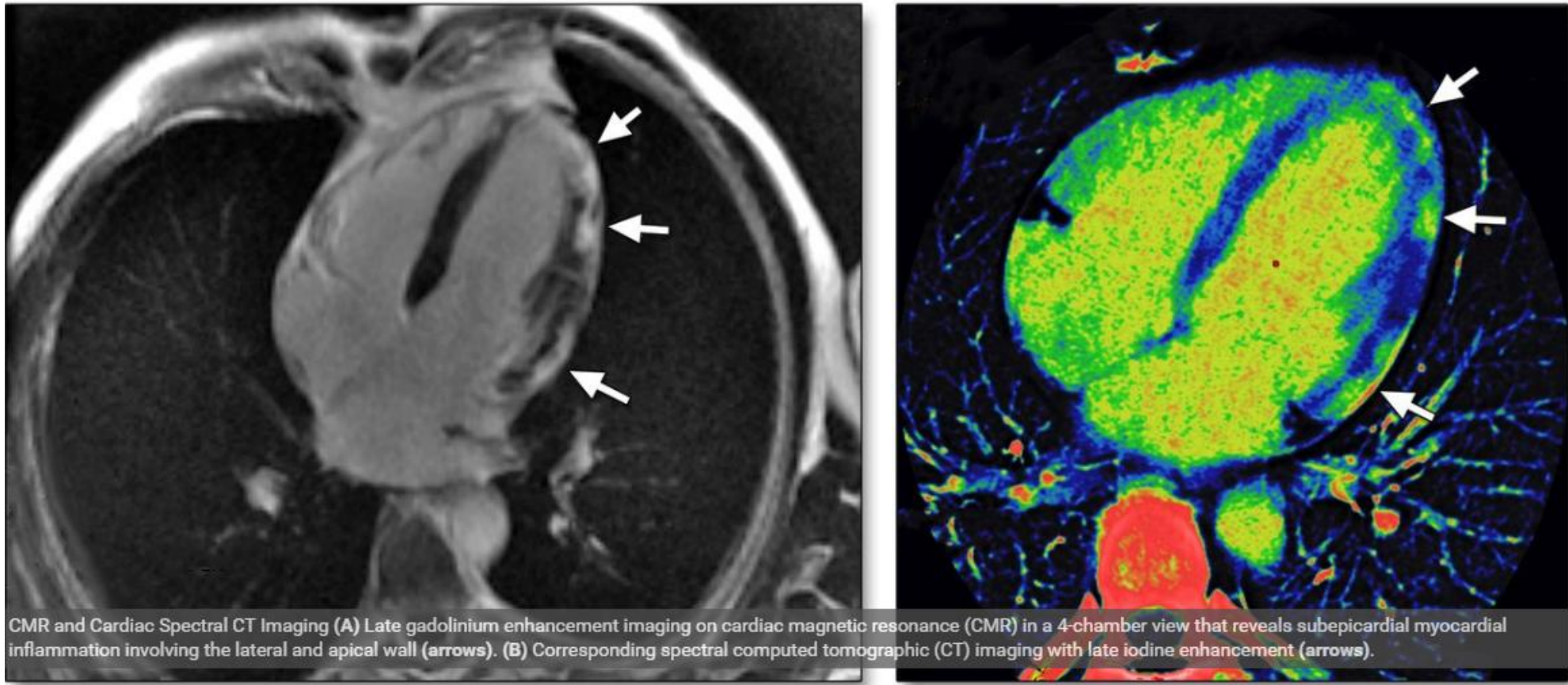
CT – triple rule-out



CT



CT



CMR – „Lake Louise Criteria“

Cardiovascular Magnetic Resonance in Myocarditis: A JACC White Paper

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Hassan Abdel-Aty, MD,§ Matthias Gutberlet, MD,** Sanjay Prasad, MD,††
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Peter Liu, MD,## for the *International Consensus Group on Cardiovascular Magnetic Resonance
in Myocarditis*

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”).

CMR

Table 5

Indications for Cardiovascular Magnetic Resonance in Patients With Suspected Myocarditis

New Onset or Persisting Symptoms Suggestive of Myocarditis	Plus	Evidence for Recent/Ongoing Myocardial Injury	Plus	Suspected Viral Etiology
Dyspnea or orthopnea or palpitations or effort intolerance/malaise or chest pain		Ventricular dysfunction or new or persisting ECG abnormalities or elevated troponin		History of recent systemic viral disease or previous myocarditis or absence of risk factors for coronary artery disease or age <35 yrs or symptoms not explained by coronary stenosis on coronary angiogram or recent negative ischemic stress test

ECG = electrocardiogram.

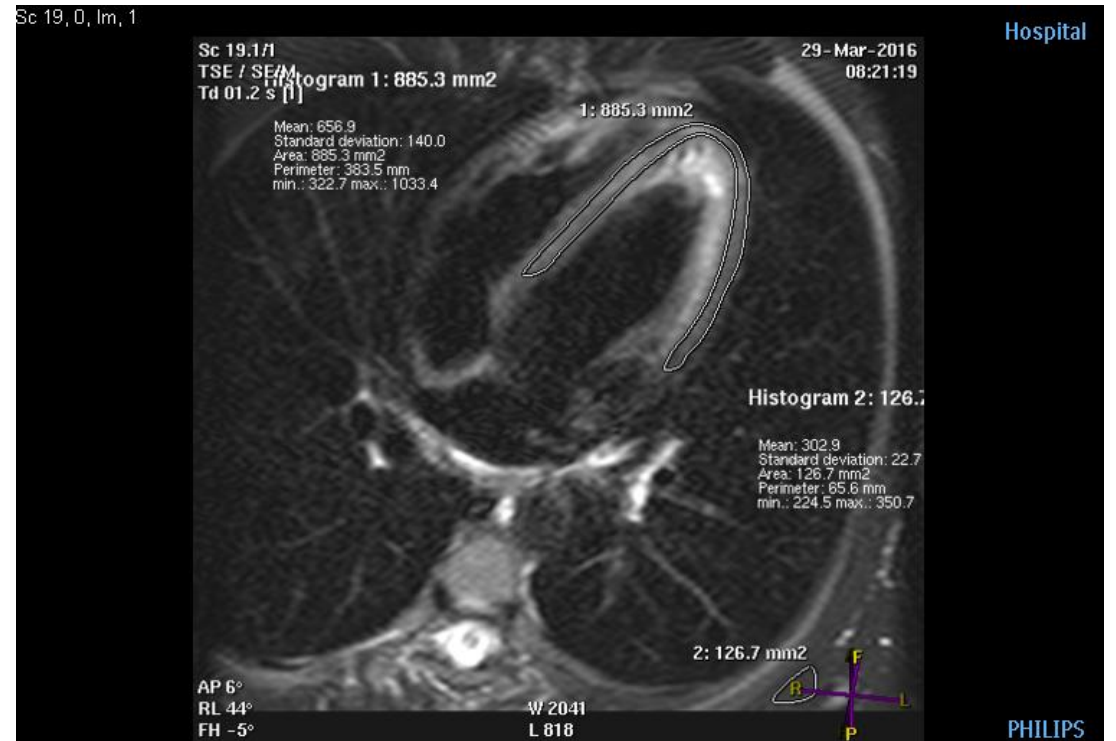
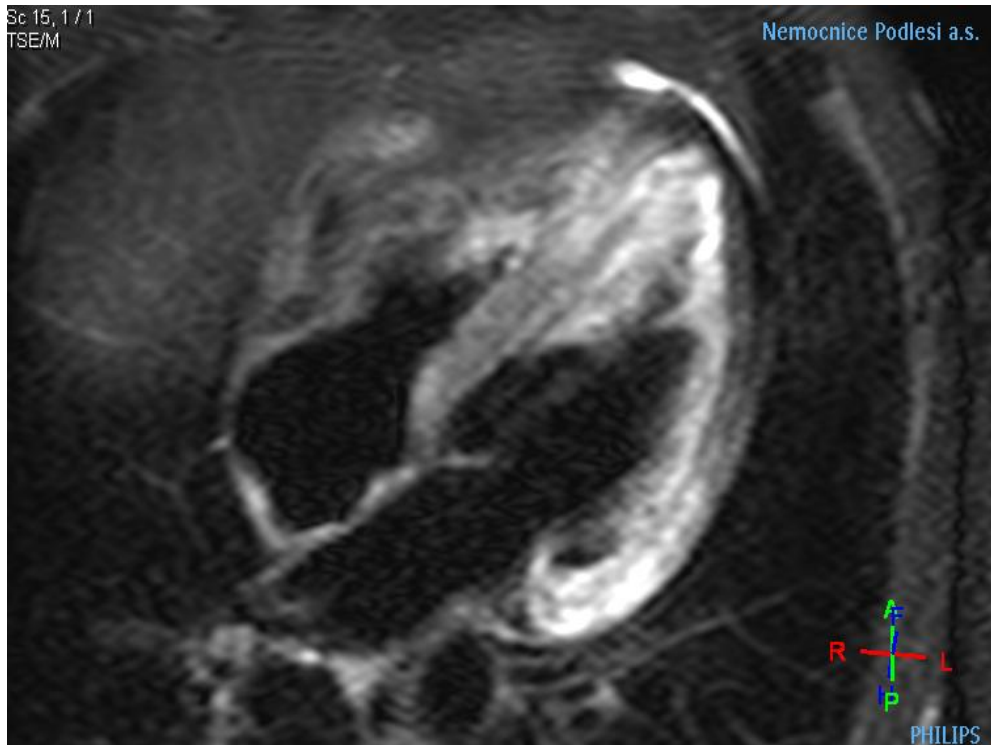
Proposed Terminologies for Describing CMR Findings

	Normal		CMR Findings Consistent With Myocardial Inflammation		
Edema	Lack of evidence for myocardial edema	Patchy areas or regions of high T2 signal intensity indicating focal or regional edema*	Subepicardial or septal layer of high T2 signal intensity indicating regional edema	Transmural high T2 signal intensity indicating regional edema, consistent with but not specific for myocardial inflammation	Global high T2 signal intensity indicating global edema†
Hyperemia Capillary leak	Lack of evidence for increased myocardial early gadolinium enhancement ratio		Increased myocardial early gadolinium enhancement ratio‡		
Irreversible cell injury	Lack of evidence for regional late gadolinium enhancement	Patchy areas of late gadolinium enhancement indicating focal injury	Subepicardial or septal layer of late gadolinium enhancement indicating regional injury	Transmural late gadolinium enhancement, consistent with but not specific for myocardial inflammation	
	Normal		Supportive CMR Findings		
LV dysfunction	Normal LV function	Regional systolic dysfunction		Global systolic dysfunction	
Pericardial effusion	Lack of evidence for pericardial effusion	Small pericardial effusion	Moderately large pericardial effusion	Large pericardial effusion without hemodynamic relevance	Large pericardial effusion with hemodynamic relevance

*To avoid misinterpretation of artifacts, areas with abnormal signal intensity should consist of at least 10 adjacent pixels to be regarded as relevant. †Global high T2 signal is defined by a signal intensity ratio between myocardium and skeletal muscle of ≥ 2.0 . ‡An increased myocardial early gadolinium enhancement ratio is defined by either a signal intensity enhancement ratio between myocardium and skeletal muscle of ≥ 4.0 or an absolute myocardial enhancement of $\geq 45\%$.

CMR = cardiovascular magnetic resonance; LV = left ventricular.

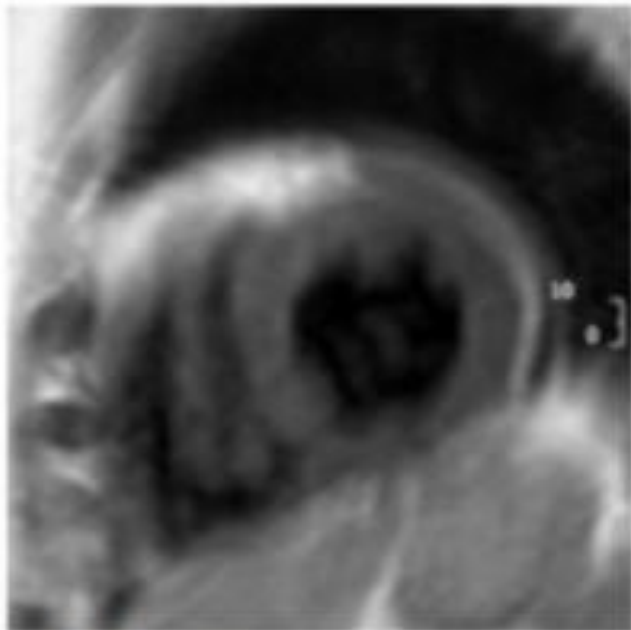
Lokální a globální edém



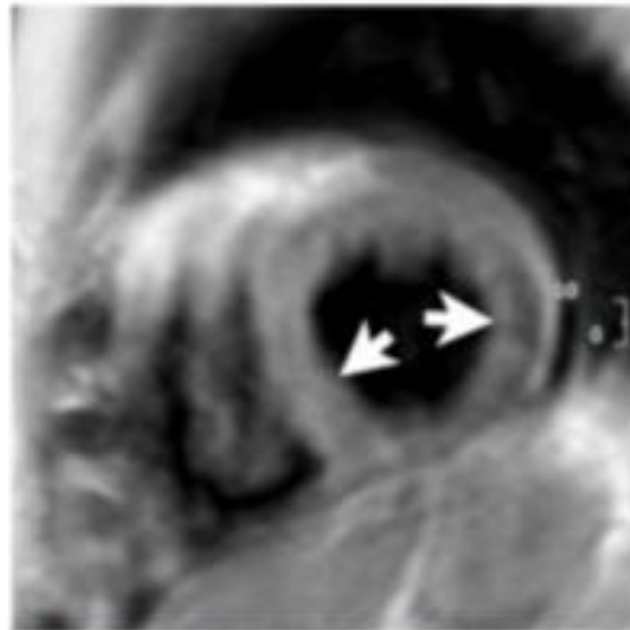
Edema ratio (SI myokardu/SI kosterního svalu) $\geq 2,0$

Early gadolinium enhancement ratio

- zvýšené globální časné sycení myokardu gadoliniem ve srovnání s kosterním svalem (EGEr ≥ 4 nebo absolutní vzestup intenzity signálu myokardu postkontrastně ≥ 45 %)



Early enhancement - pre

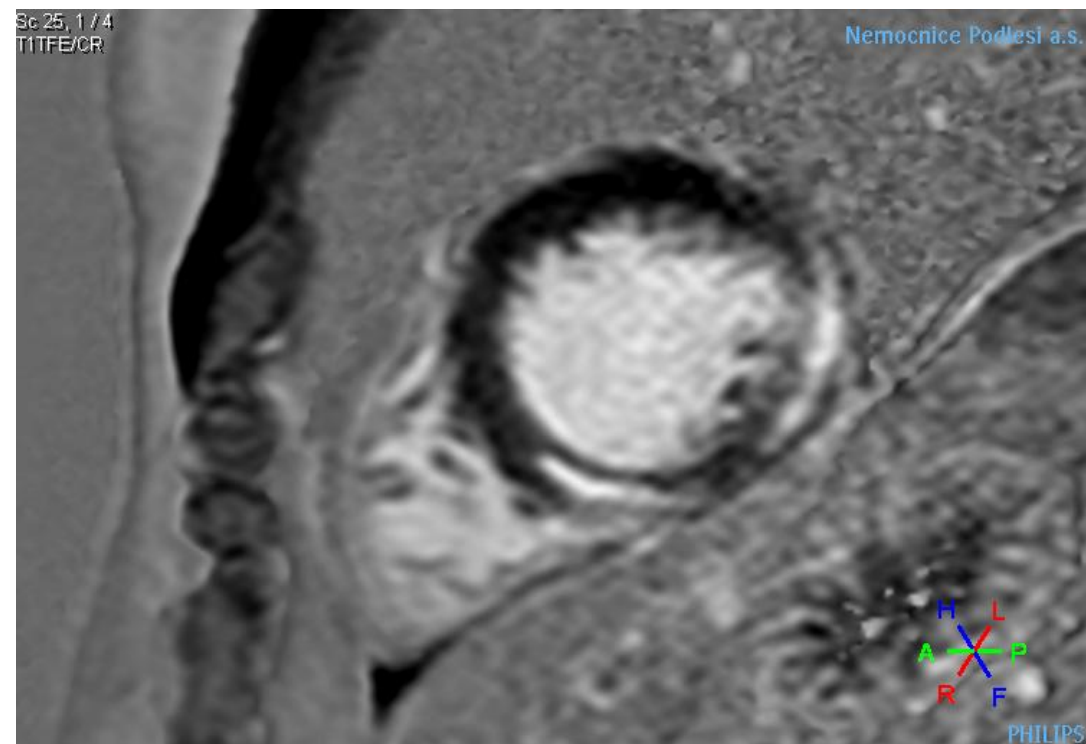
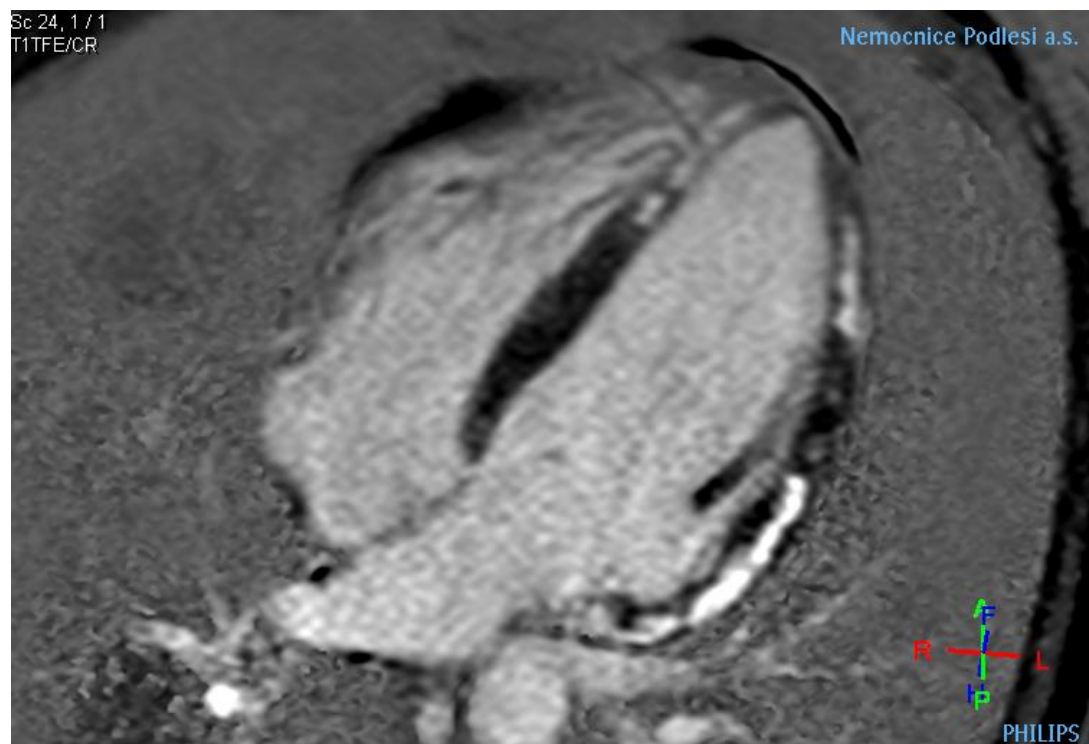


Early enhancement - post

$$\text{EGEr} = \frac{(\text{postSI}_{\text{myo}} - \text{preSI}_{\text{myo}}) / \text{preSI}_{\text{myo}}}{(\text{postSI}_{\text{skm}} - \text{preSI}_{\text{skm}}) / \text{preSI}_{\text{skm}}}$$

(myo – myokard, skm – skeletární sval)

Late gadolinium enhancement



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.

^{*}The clinical suspicion for active myocarditis should be based on the criteria listed in [Table 5](#). [†]Images should be obtained using a body coil or a surface coil with an effective surface coil intensity correction algorithm; global signal intensity (SI) increase has to be quantified by an SI ratio of myocardium over skeletal muscle of ≥ 2.0 . If the edema is more subendocardial or transmural in combination with a colocalized ischemic (including the subendocardial layer) pattern of late gadolinium enhancement, acute myocardial infarction is more likely and should be reported. [‡]Images should be obtained using a body coil or a surface coil with an effective surface coil intensity correction algorithm; a global SI enhancement ratio of myocardium over skeletal muscle of ≥ 4.0 or an absolute myocardial enhancement of $\geq 45\%$ is consistent with myocarditis. [§]Images should be obtained at least 5 min after gadolinium injection; foci typically exclude the subendocardial layer, are often multifocal, and involve the subepicardium. If the late gadolinium enhancement pattern clearly indicates myocardial infarction and is colocalized with a transmural regional edema, acute myocardial infarction is more likely and should be reported.

Abbreviations as in [Table 6](#).

Table 3**Overview of the Diagnostic Accuracy of Individual Tissue Criteria as Assessed in Controlled Trials**

	Validation	Sensitivity (%)	Specificity (%)	Accuracy (%)	PPV (%)	NPV (%)
Early myocardial gadolinium enhancement						
Friedrich et al., <i>Circulation</i> 1998 (9)	Clinical	84	89	86	89	84
Laissy et al., <i>Chest</i> 2002 (11)	Clinical	85	100	89	100	70
Abdel-Aty et al., <i>J Am Coll Cardiol</i> 2005 (13)	Clinical	80	68	74	74	75
Gutberlet et al., <i>Radiology</i> 2008 (34)	Histology	63	86	72	86	63
Pooled data (n = 194)		74	83	78	86	70
T2						
Rieker et al., <i>Rofo</i> 2002 (36)	Clinical	100	50	76	69	100
Laissy et al., <i>Chest</i> 2002 (11)	Clinical	45	100	59	100	39
Abdel-Aty et al., <i>J Am Coll Cardiol</i> 2005 (13)	Clinical	84	74	79	78	81
Gutberlet et al., <i>Radiology</i> 2008 (34)	Histology	67	69	67	74	60
Pooled data (n = 178)		70	71	70	77	63
Late enhancement						
Rieker et al., <i>Rofo</i> 2002 (36)	Clinical	45	60	52	56	50
Abdel-Aty et al., <i>J Am Coll Cardiol</i> 2005 (18)	Clinical	44	100	71	78	62
Mahrholdt et al., <i>Circulation</i> 2006 (40)	Histology	95	96	96	99	81
Gutberlet et al., <i>Radiology</i> 2008 (34)	Histology	27	80	49	65	44
Yilmaz et al., <i>Heart</i> 2008 (43)	Histology	35	83	51	81	38
Pooled data (n = 336)		59	86	68	89	53

Diagnostic Performance of CMR Imaging Compared With EMB in Patients With Suspected Myocarditis

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Leipzig and Tuebingen, Germany

OBJECTIVES The goal of this study was to assess the diagnostic performance of cardiac magnetic resonance (CMR) compared with endomyocardial biopsy in patients with suspected acute myocarditis (AMC) and chronic myocarditis (CMC).

BACKGROUND Several studies have reported an encouraging diagnostic performance of CMR in myocarditis. However, the comparison of CMR with clinical data only and the use of preselected patient populations are important limitations of the majority of these reports.

METHODS One hundred thirty-two consecutive patients with suspected AMC (defined by symptoms ≤ 14 days; $n = 70$) and CMC (defined by symptoms > 14 days; $n = 62$) were included. Patients underwent cardiac catheterization with left ventricular endomyocardial biopsy and CMR, including T_2 -weighted imaging for assessment of edema, T_1 -weighted imaging before and after contrast administration for evaluation of hyperemia, and assessment of late gadolinium enhancement. CMR results were considered to be consistent with the diagnosis of myocarditis if 2 of 3 CMR techniques were positive.

RESULTS Within the total population, myocarditis was the most common diagnosis on endomyocardial biopsy analysis (62.9%). Viral genomes were detected in 30.3% (40 of 132) of patients within the total patient population and significantly more often in patients with AMC than CMC (40.0% vs. 19.4%; $p = 0.013$). For the overall cohort of patients with either suspected AMC or CMC, the diagnostic sensitivity, specificity, and accuracy of CMR were 76%, 54%, and 68%, respectively. The best diagnostic performance was observed in patients with suspected AMC (sensitivity, 81%; specificity, 71%; and accuracy, 79%). In contrast, diagnostic performance of CMR in suspected CMC was found to be unsatisfactory (sensitivity, 63%; specificity, 40%; and accuracy, 52%).

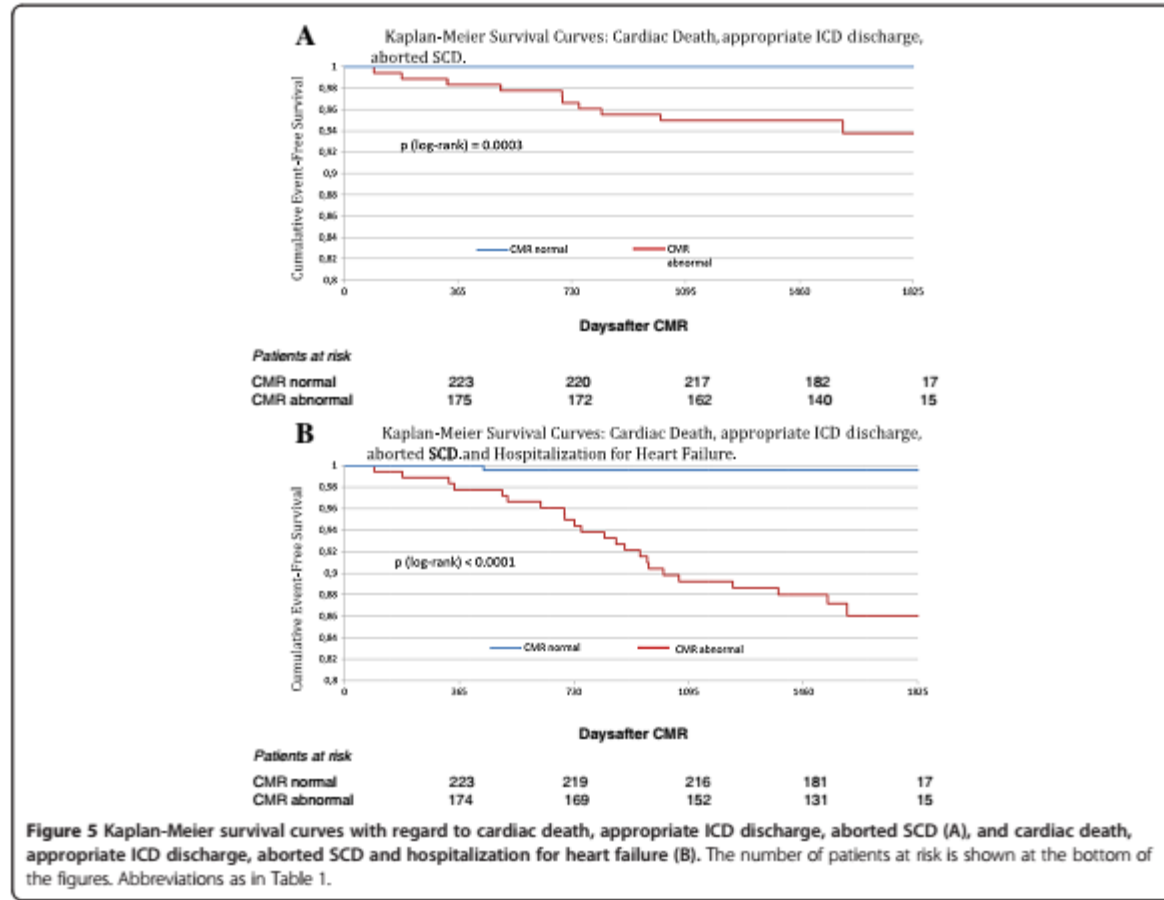
CONCLUSIONS The results of this study underline the usefulness of CMR in patients with suspected AMC. In contrast, the diagnostic performance of CMR in patients with suspected CMC might not be sufficient to guide clinical management. (J Am Coll Cardiol Img 2012;5:513–24) © 2012 by the American College of Cardiology Foundation

CMR

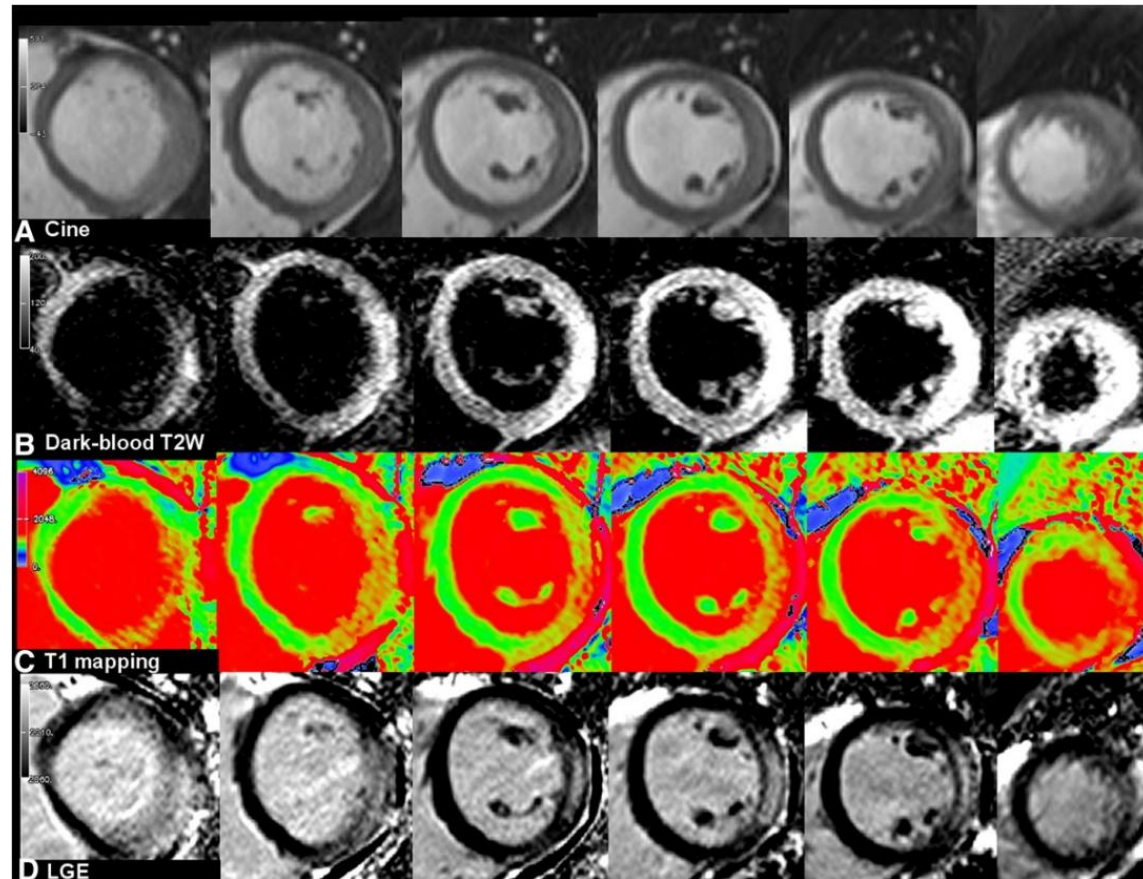
Příčiny falešně negativního nálezu:

- delší odstup od vzniku potíží
- difuzní či velmi diskrétní změny
- chybně či nesprávně provedené či interpretované vyšetření

CMR – prognóza



T1 mapping



T1 mapping

Table 2 Diagnostic performance of CMR tissue characterization methods in the detection of suspected acute myocarditis

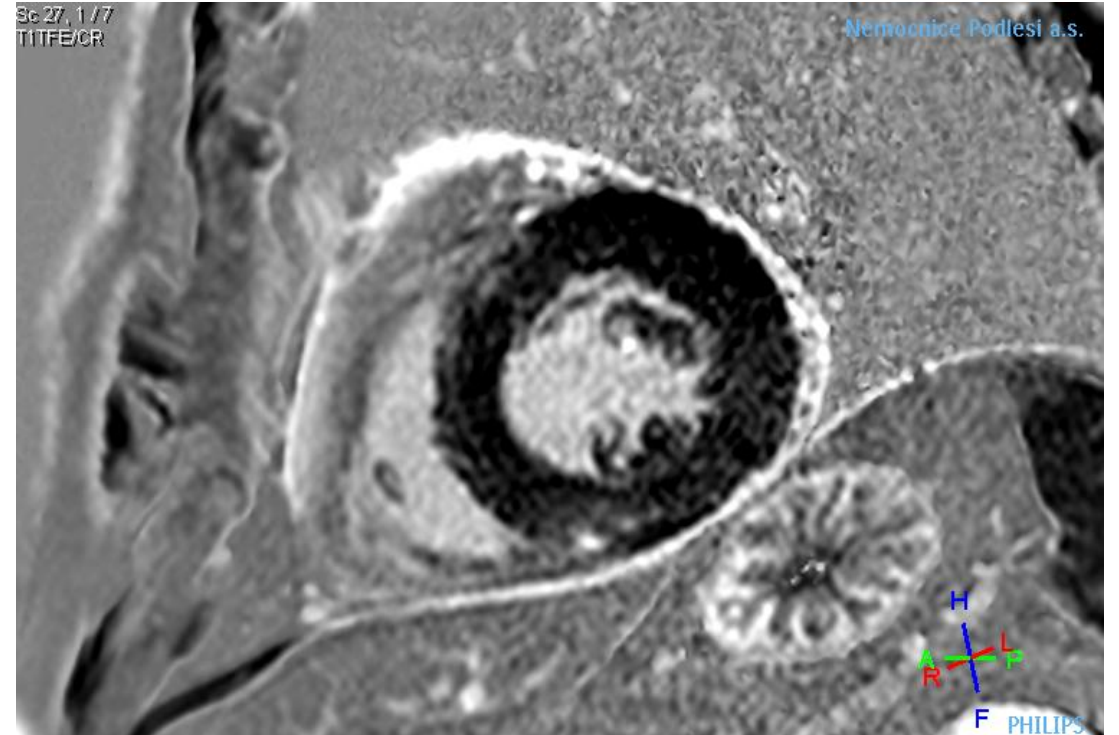
Tissue criteria	Sensitivity (%)	Specificity (%)	Accuracy (%)	PPV (%)	NPV (%)
Individual					
T1-mapping*	90	88	89	90	88
Dark-blood T2*	48	86	66	81	58
LGE	72	97	81	98	67
Combination (with LGE)					
Dark-blood T2 and LGE (2 out of 2) ^{††}	45	97	64	96	51
Dark-blood T2 or LGE (Any 1 of 2)	75	86	79	90	67
T1-mapping and LGE (2 out of 2) [†]	67	97	78	98	63
T1-mapping or LGE (Any 1 of 2)	95	83	91	91	91
T1-mapping, dark-blood T2 or LGE (Any 1 of 3)	95	71	86	85	89
T1-mapping, dark-blood T2 or LGE (Any 2 of 3)	70	97	80	98	65
T1-mapping and dark-blood T2 and LGE (3 out of 3)	45	97	64	96	51
Combination (without LGE)					
T1-mapping and dark-blood T2 (2 out of 2) [†]	48	98	71	97	61
T1-mapping or dark-blood T2 (Any 1 of 2)	90	76	84	82	86

*statistically different ($p < 0.05$); ^{††}no statistical difference ($p = ns$). T1-mapping: myocardial injury is detected when T1 is ≥ 990 ms; Dark-blood T2-weighted imaging: edema is diagnosed when the T2 SI ratio ($T2\ SI_{myocardium} : skeletal\ muscle$) is $\geq 2:1$; Late gadolinium enhancement (LGE) is detected when myocardial SI is ≥ 2 SD above mean SI of remote myocardium. For each technique, only contiguous areas of myocardium $\geq 40\ mm^2$ above the stated threshold were considered relevant; involvement of $\geq 5\%$ of any segment on a per-subject basis was the threshold used for comparison of methods. PPV = positive predictive value; NPV = negative predictive value.

CMR

Unikátní metoda pro stanovení:

- perimyokarditis
- myoperikarditis
- myokarditis pravé komory



myoperikarditis

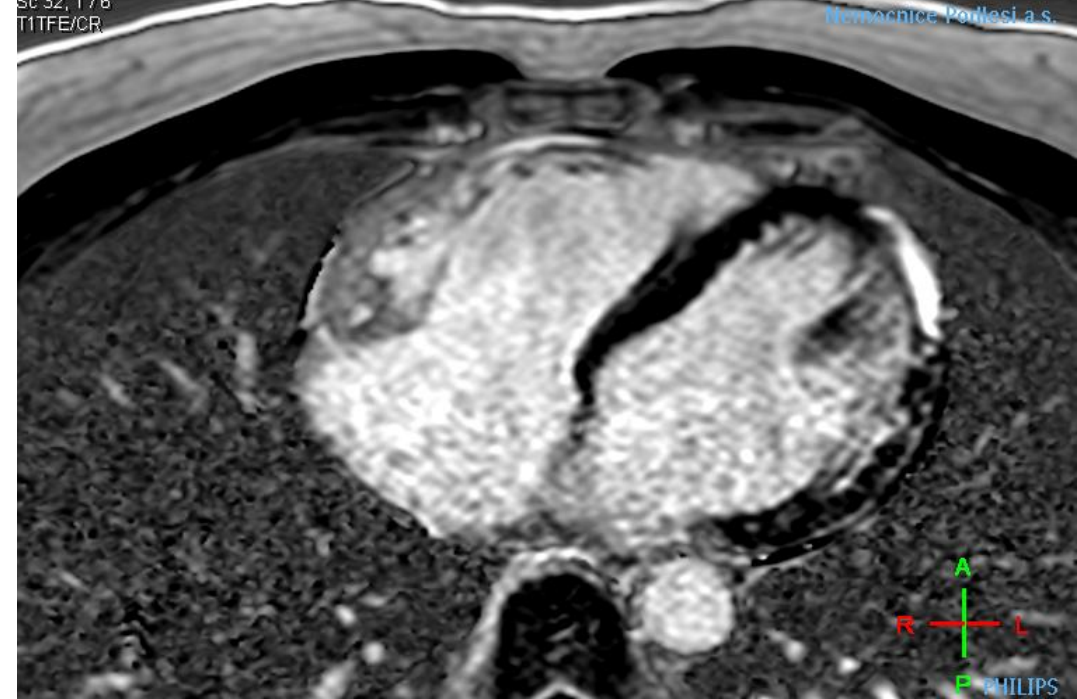
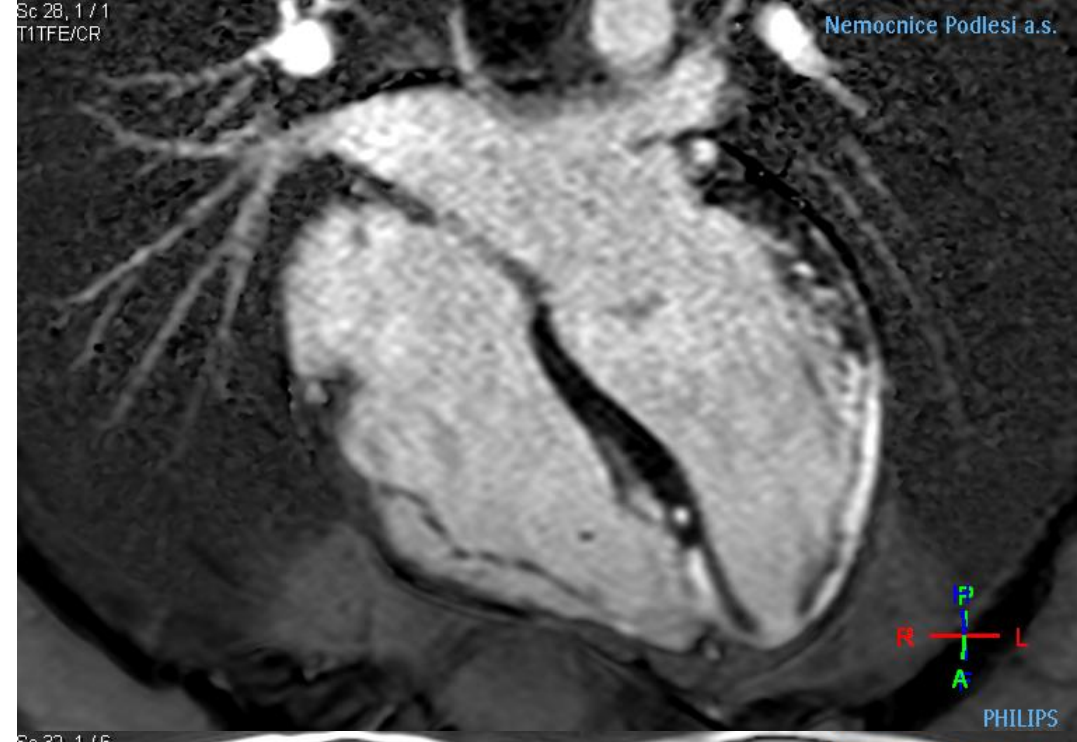
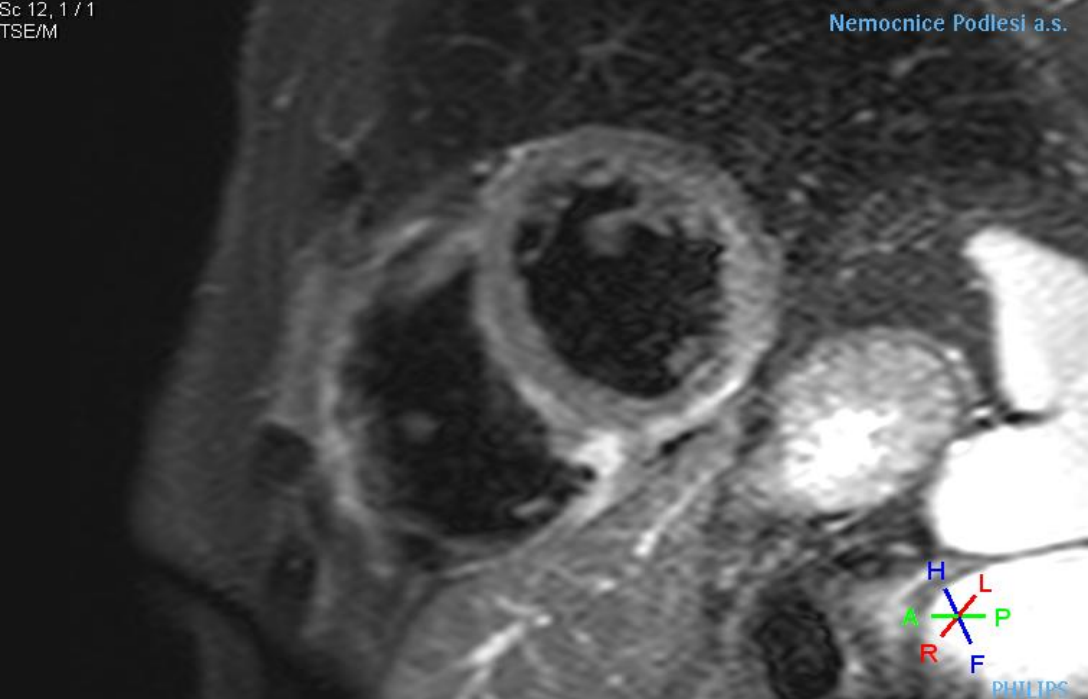
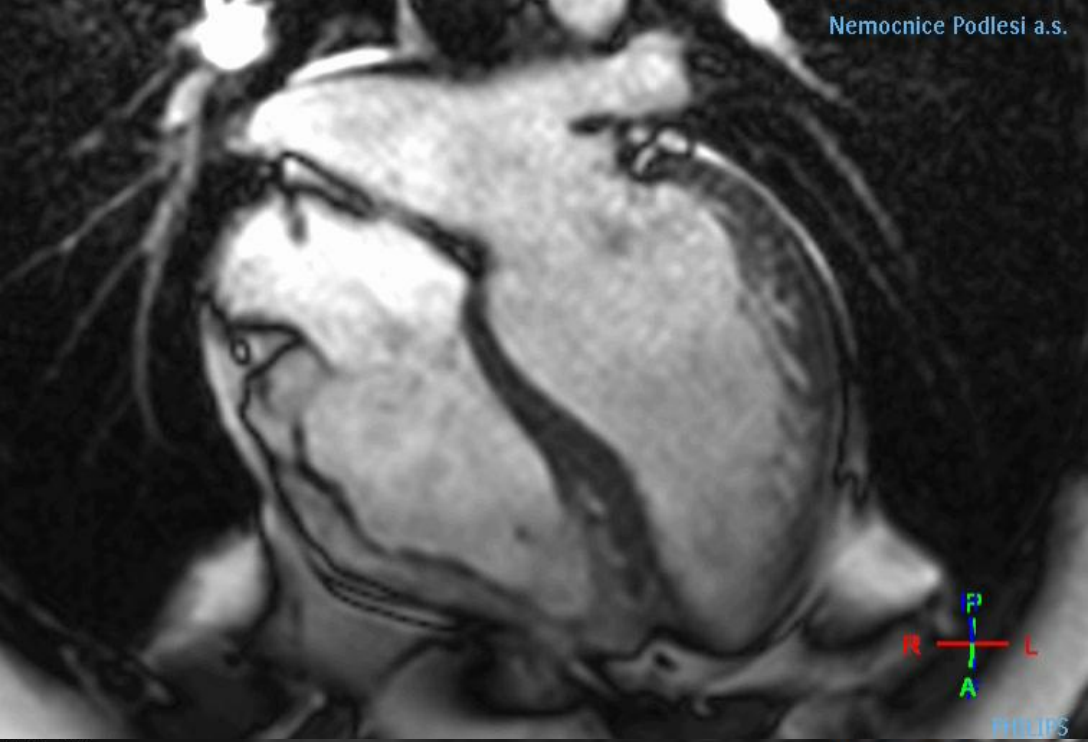


Table 4 Definition of clinically suspected myocarditis according to the ESC 2013 Myocarditis Task Force³⁶

Presence of ≥ 1 clinical presentation and ≥ 1 diagnostic criteria from different categories, in the absence of:

- (1) Angiographically detectable CAD (coronary stenosis $\geq 50\%$)
- (2) Known pre-existing cardiovascular disease or extra-cardiac causes that could explain the syndrome (e.g. valve disease, congenital heart disease, etc.).
- (3) If the patient is asymptomatic ≥ 2 diagnostic criteria should be met.

Diagnosis of certainty and aetiological diagnosis of myocarditis requires EMB (histology, immunohistology, infectious agents by PCR)

Clinical presentations

Acute coronary syndrome-like, with or without normal global or regional left ventricular (LV) and/or right ventricular (RV) dysfunction on echocardiography or CMR, with or without increased troponin (Tn)T/Tnl (that may have a time course similar to AMI or a prolonged and sustained release over several weeks or months).

New onset or worsening unexplained heart failure.

Chronic unexplained heart failure of > 3 months duration.

Life-threatening unexplained condition (including life-threatening arrhythmias and aborted sudden death, cardiogenic shock, severely impaired left ventricular function).

Diagnostic criteria

(1) 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, 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, and supraventricular tachycardia

(2) Myocardiocytolysis markers

elevated cardiac troponins

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

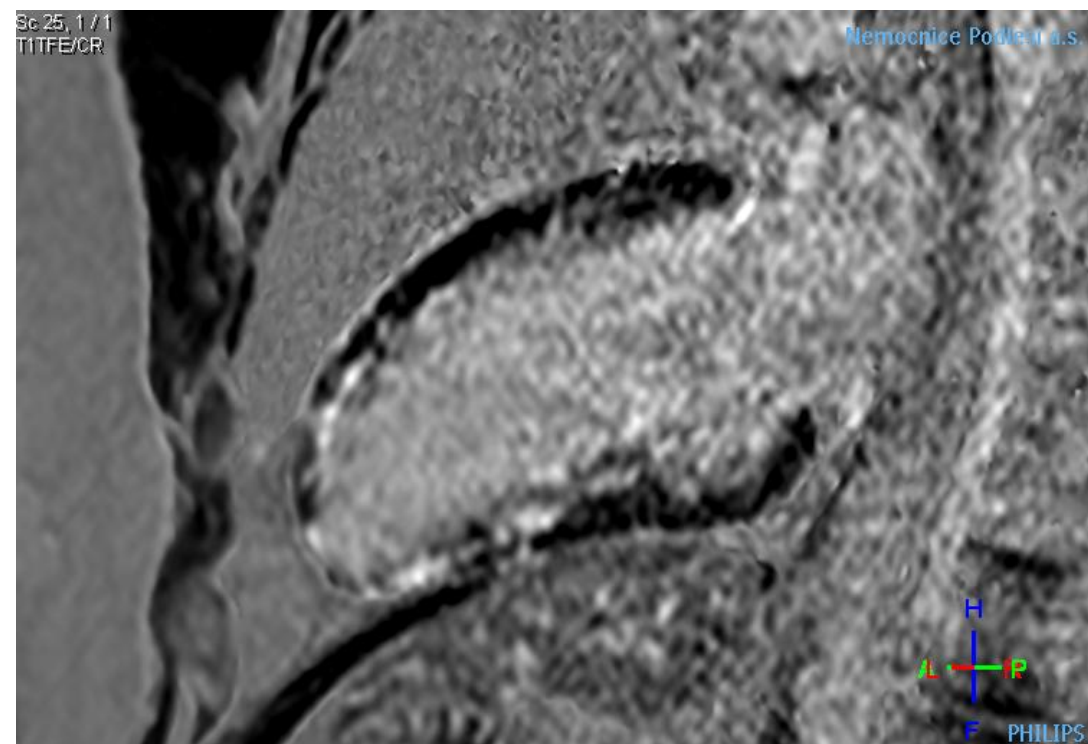
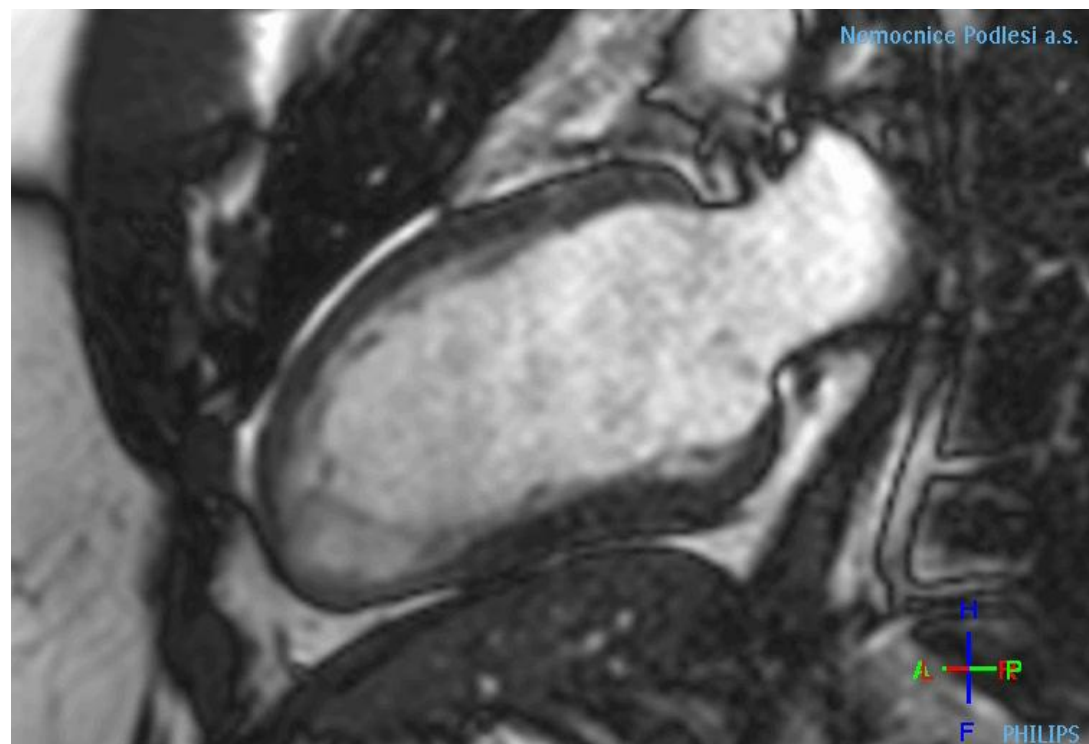
new, otherwise unexplained LV and/or RV structure and function abnormality.

(4) Tissue characterization by CMR

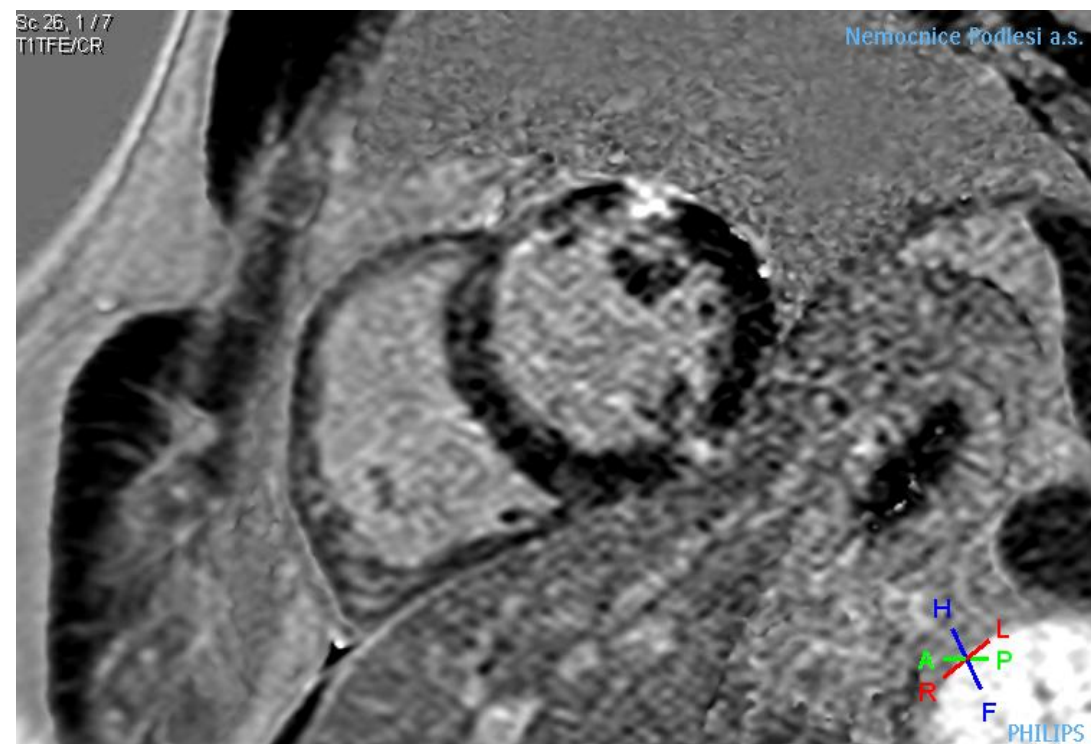
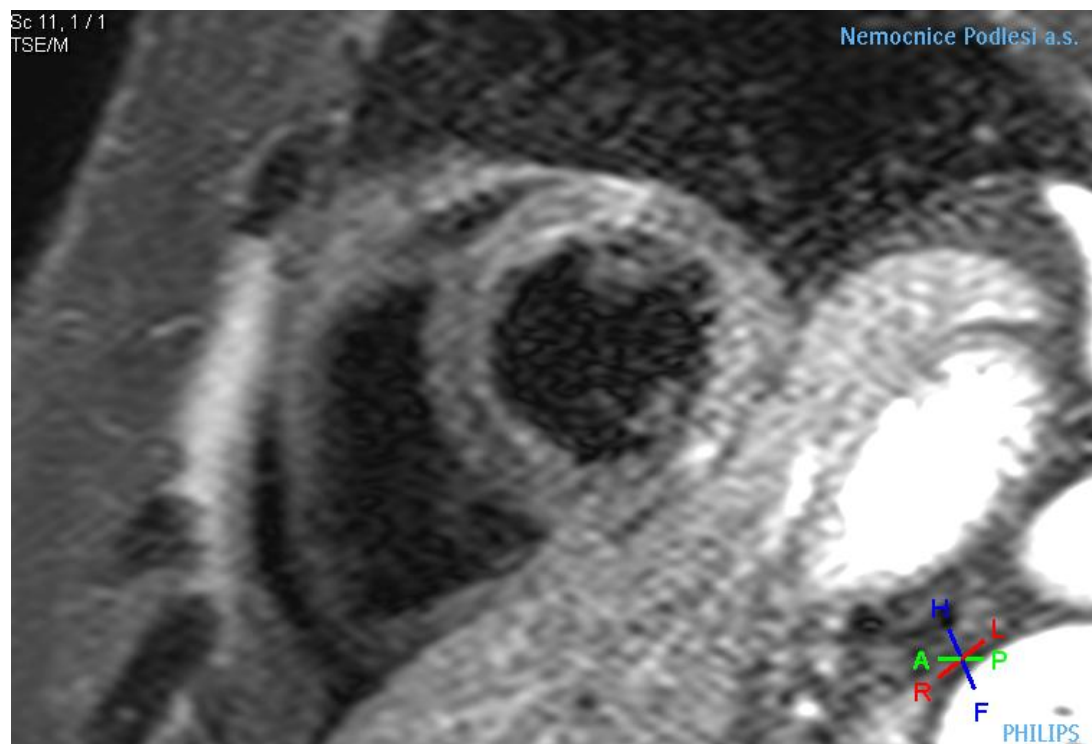
oedema and/or LGE of classical myocarditic pattern (according to Lake-Louise criteria).³⁷

- nemůže mít i pacient s ICHS akutní myokarditidu?

ICHS a akutní myokarditida



ICHS a akutní myokarditida



MINOCA

Table 1 Diagnostic criteria for myocardial infarction with non-obstructive coronary arteries

The diagnosis of MINOCA is made immediately upon coronary angiography in a patient presenting with features consistent with an acute myocardial infarct, as detailed by the following criteria:

(1) AMI criteria.¹

(a) Positive cardiac biomarker (preferably cardiac troponin) defined as a rise and/or fall in serial levels, with at least one value above the 99th percentile upper reference limit.

and

(b) Corroborative clinical evidence of infarction evidenced by at least one of the following:

(i) Symptoms of ischaemia

(ii) New or presumed new significant ST-T changes or new LBBB

(iii) Development of pathological Q waves

(iv) Imaging evidence of new loss of viable myocardium or new RVWMA

(v) Intracoronary thrombus evident on angiography or at autopsy

(2) Non-obstructive coronary arteries on angiography:

- Defined as the absence of obstructive CAD on angiography, (i.e. no coronary artery stenosis $\geq 50\%$), in any potential infarct-related artery.

- This includes both patients with:

- normal coronary arteries (no stenosis $> 30\%$)

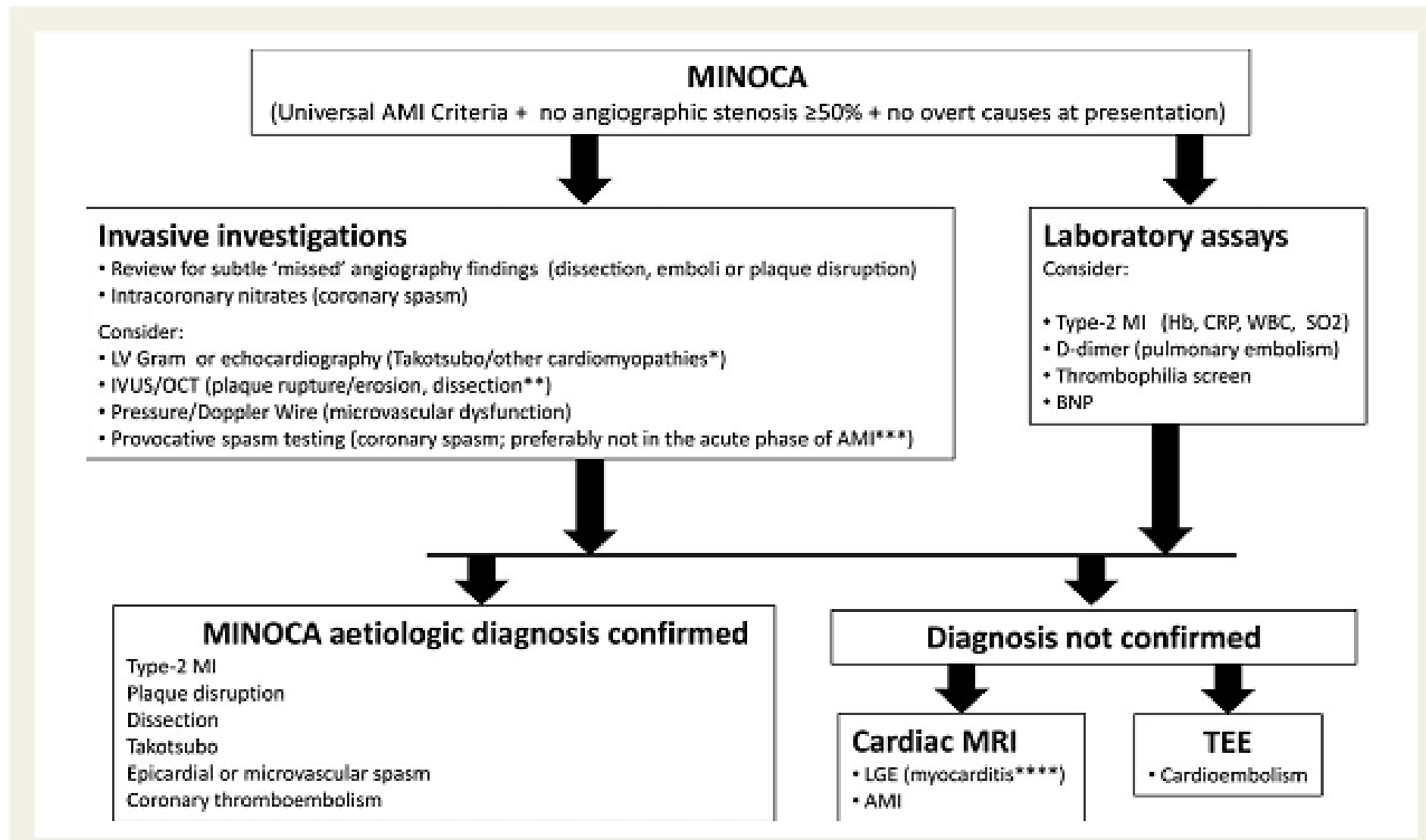
- mild coronary atheromatosis (stenosis $> 30\%$ but $< 50\%$).

(3) No clinically overt specific cause for the acute presentation:

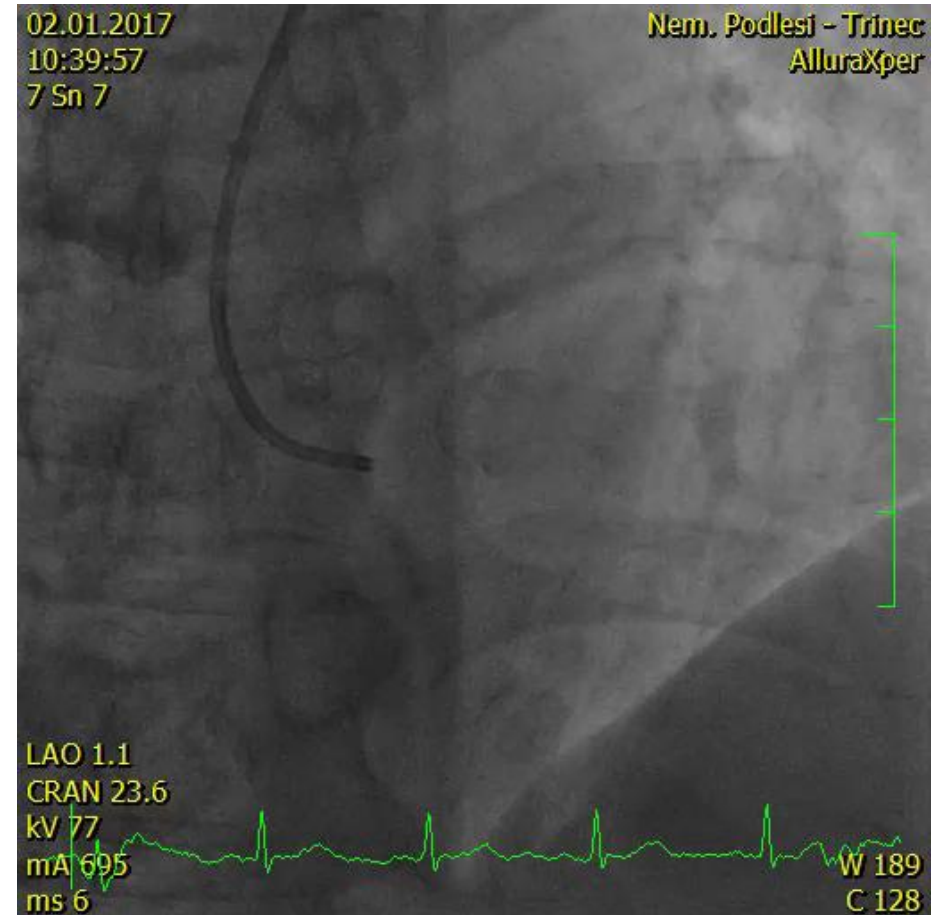
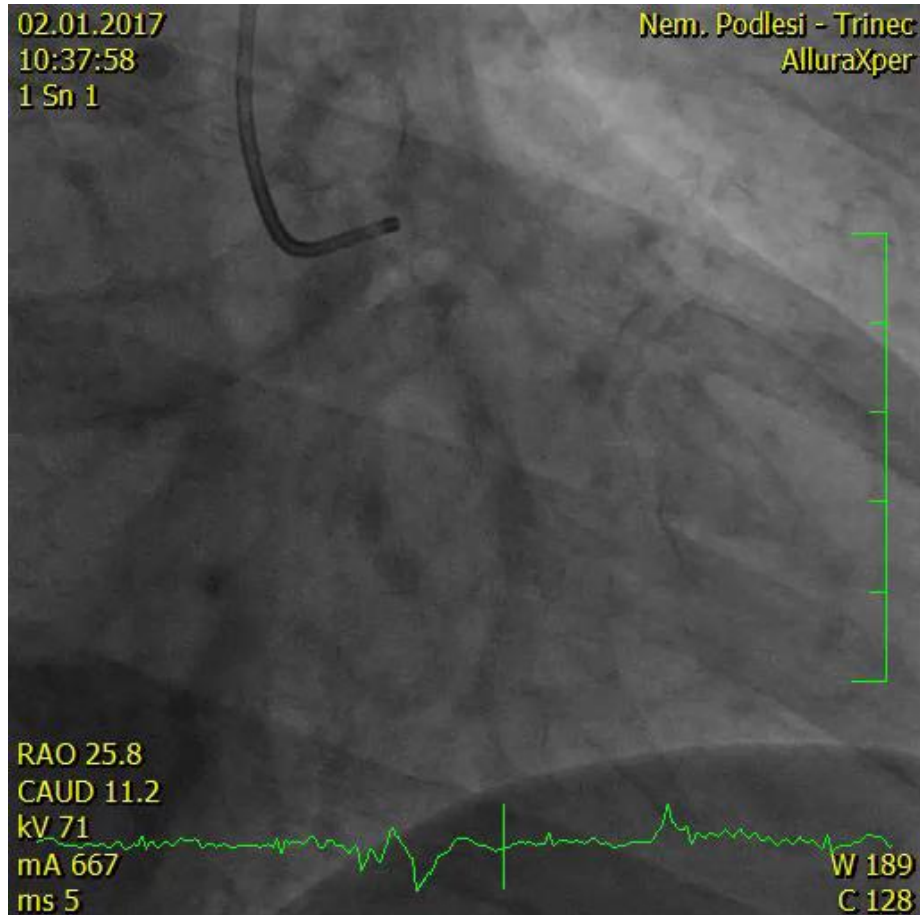
- At the time of angiography, the cause and thus a specific diagnosis for the clinical presentation is not apparent.

- Accordingly, there is a necessity to further evaluate the patient for the underlying cause of the MINOCA presentation.

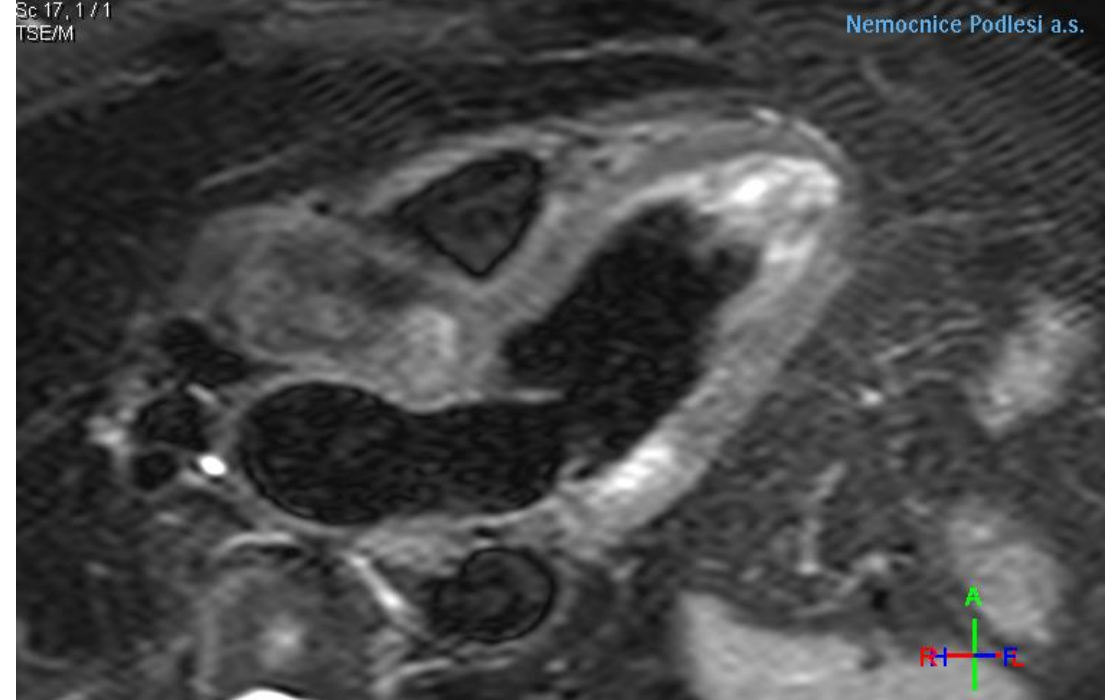
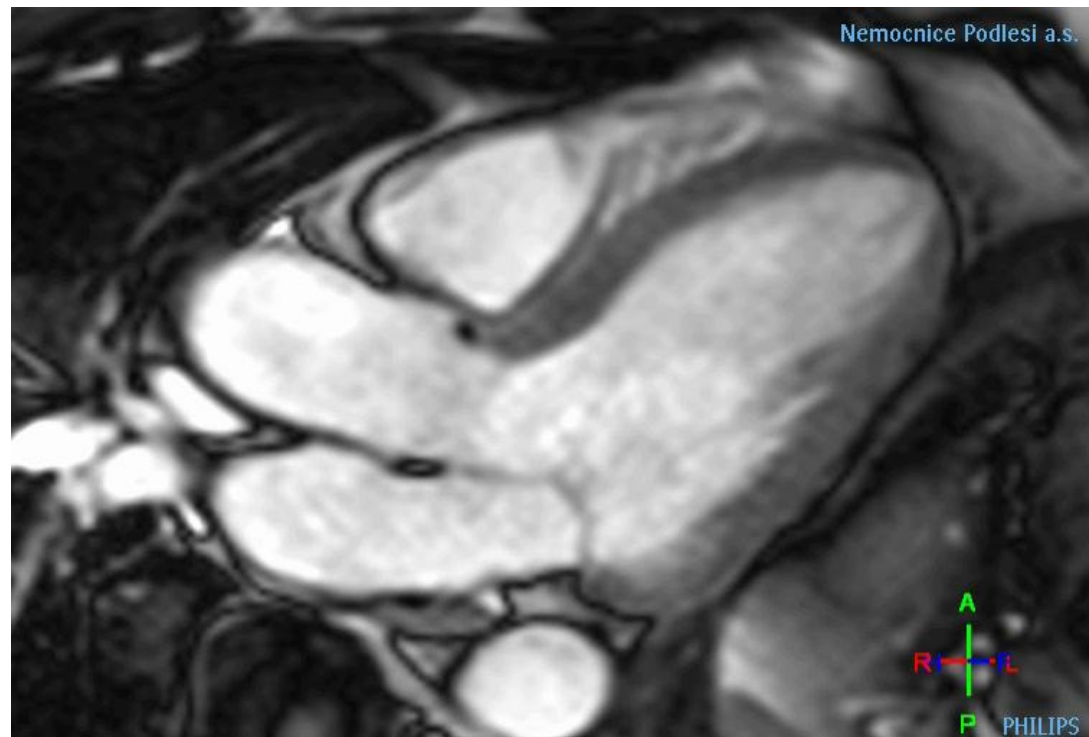
LBBB, left bundle branch block. RVWMA, regional wall motion abnormality.



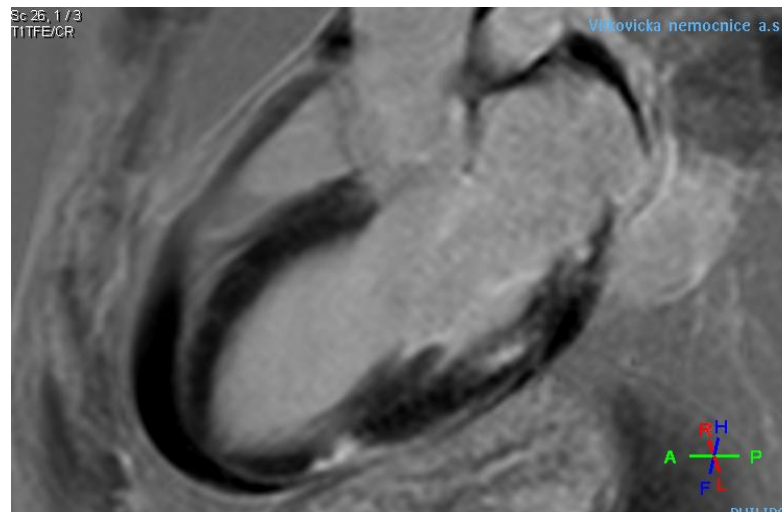
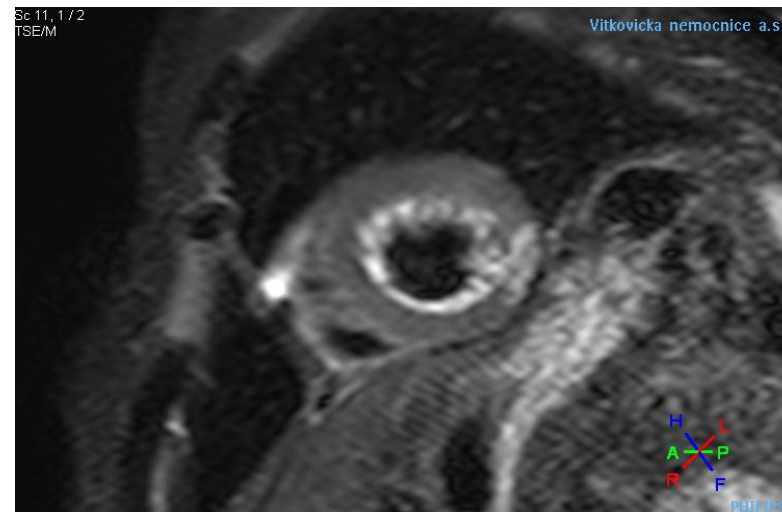
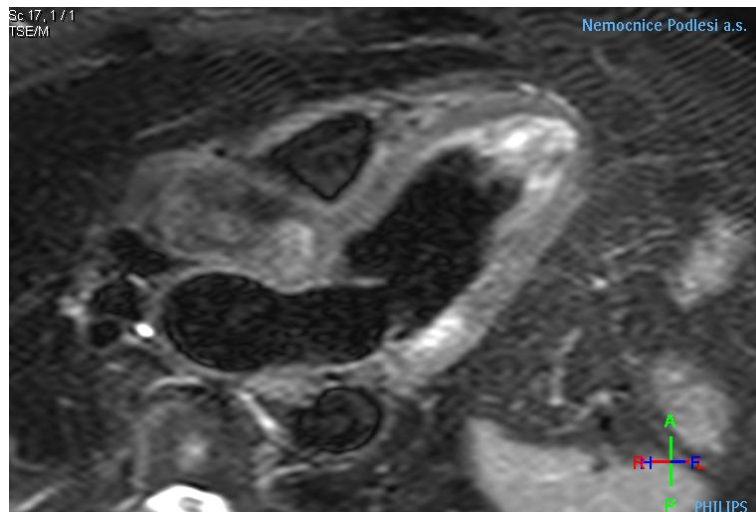
MINOCA



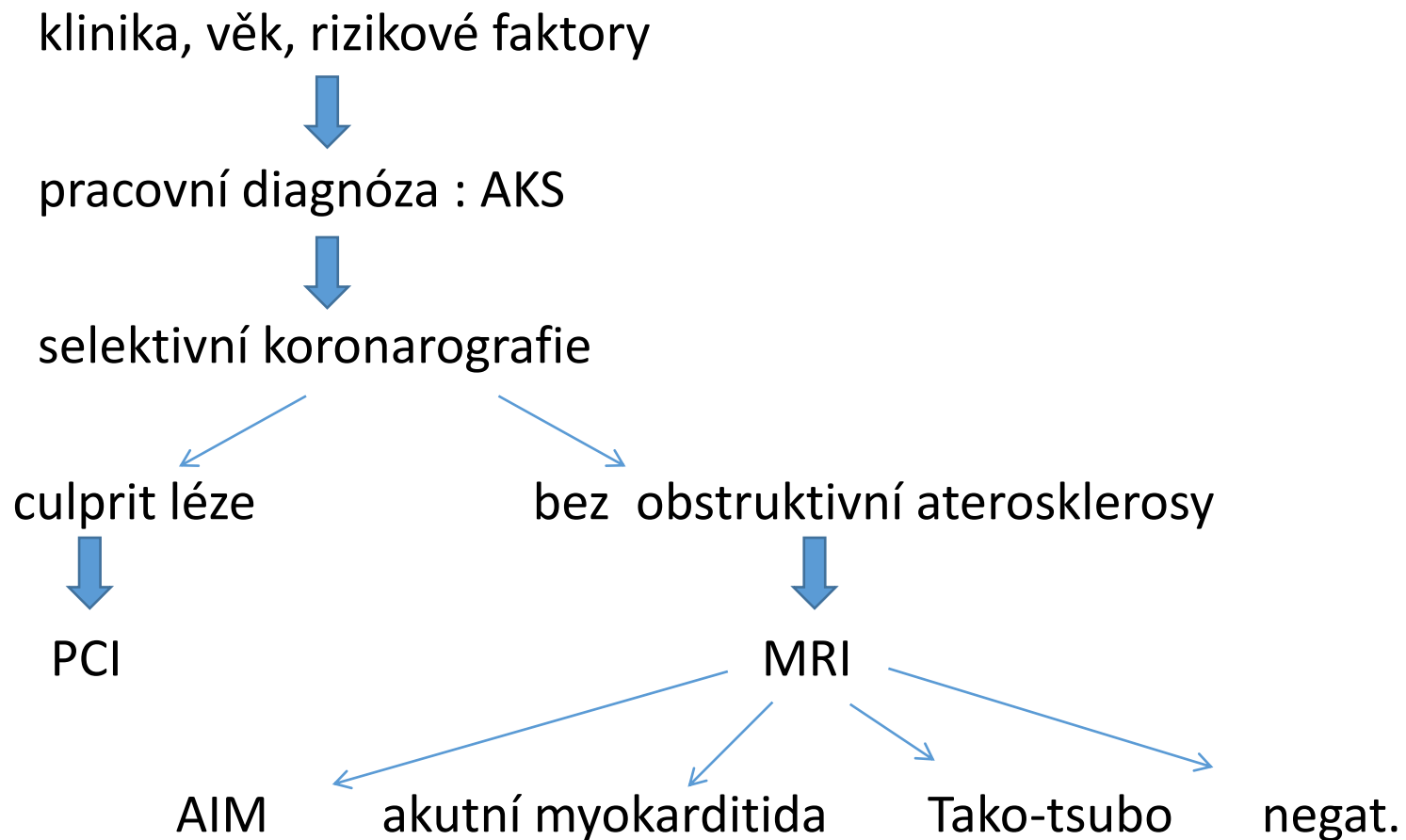
MINOCA



AIM x akutní myokarditis



Bolest na hrudi + dynamika v troponinu



The unique value of cardiovascular magnetic resonance in patients with suspected acute coronary syndrome and culprit-free coronary angiograms

Roman Panovsky, M.D., Ph.D.; Julia Borova; Martin Pleva; Vera Feitova; Petr Novotny; Vladimir Kincl, Ph.D.; Tomas Holecek; Jaroslav Meluzin, Prof., Ph.D.; Ondrej Sochor; Radka Stepanova

Summary:

Background and objective: Making a diagnosis in patients with chest pain and absence of a significant coronary artery stenosis may be difficult. The purpose of this study was to investigate the incremental diagnostic value of cardiovascular magnetic resonance (CMR) in cohort of patients with suspected acute coronary syndrome (ACS) and unobstructed coronary arteries.

Methods: Data files of patients meeting the inclusion criteria in two cardiology centres were searched out and analysed. Special attention was paid to the benefits of CMR in determining the final diagnosis.

Results: In total, 136 patients who underwent coronary angiography for chest pain were analysed. The most frequent underlying causes were myocarditis (38%) and perimyocarditis (18%), followed by angiographically unrecognised acute myocardial infarction (18%), and takotsubo cardiomyopathy (15%). Final diagnosis remained unclear in 6% patients. The contribution of CMR for the final diagnosis determination was crucial in 57% patients. In another 35% patients CMR confirmed the suspicion and, only 8% CMR examinations did not help at all and had no influence on diagnosis and treatment.

Conclusion: CMR provides powerful incremental diagnostic value in the cohort of patients with ACS and unobstructed coronary arteries. CMR should be strongly advised to be an inalienable part of diagnostic algorithms in these patients.

Myokarditis – shrnutí

- ECHO – základní diagnostická zobrazovací metoda
- SKG – vyloučení ischemické etiologie
- CT – vyloučení jiné etiologie
- CMR – „zlatý neinvazivní standard“
- EMB – potvrzení diagnózy a verifikace etiologie

Děkuji Vám za pozornost