



Fulminantní myokarditida

Jan Krejčí

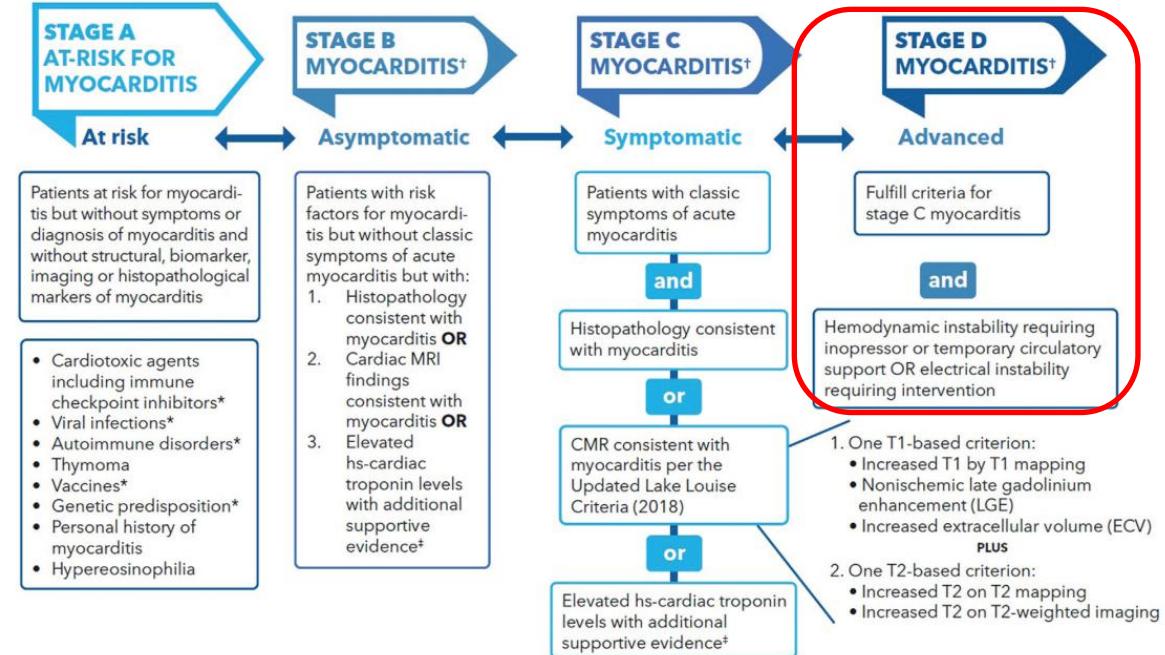


Co je nového - 2024 ACC consensus

2024 ACC Expert Consensus Decision Pathway on Strategies and Criteria for the Diagnosis and Management of Myocarditis

...vytvoření stagingu podobného jako u srdečního selhání

FIGURE 5 Proposed Stages of Myocarditis



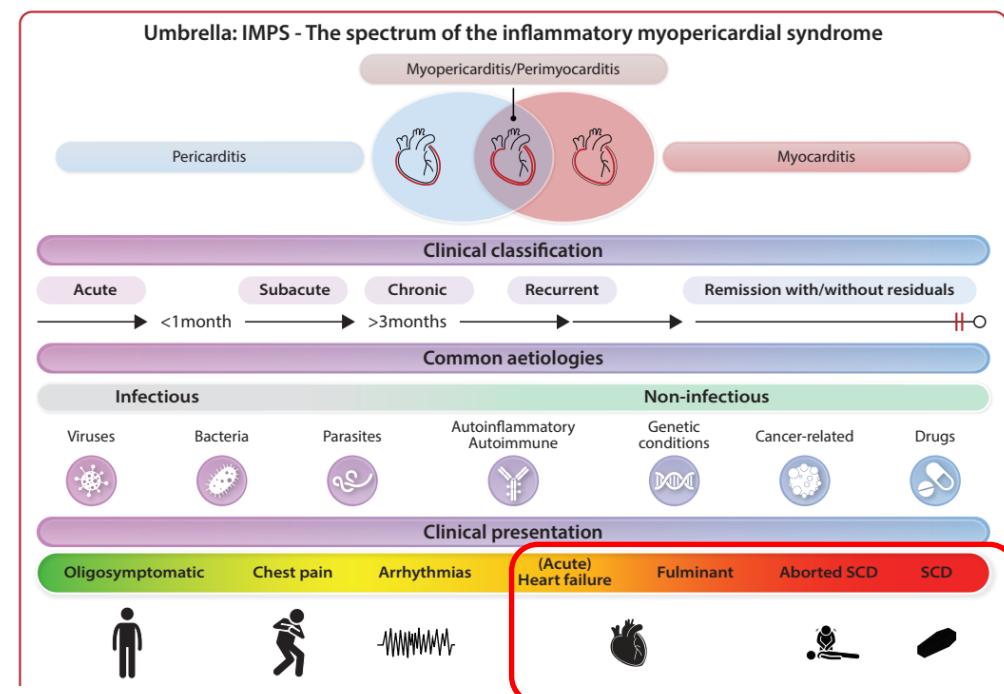
Co je nového - 2025 ESC Guidelines

2025 ESC Guidelines for the management of myocarditis and pericarditis

Developed by the task force for the management of myocarditis and pericarditis of the European Society of Cardiology (ESC)

Endorsed by the Association for European Paediatric and Congenital Cardiology (AEPC) and the European Association for Cardio-Thoracic Surgery (EACTS)

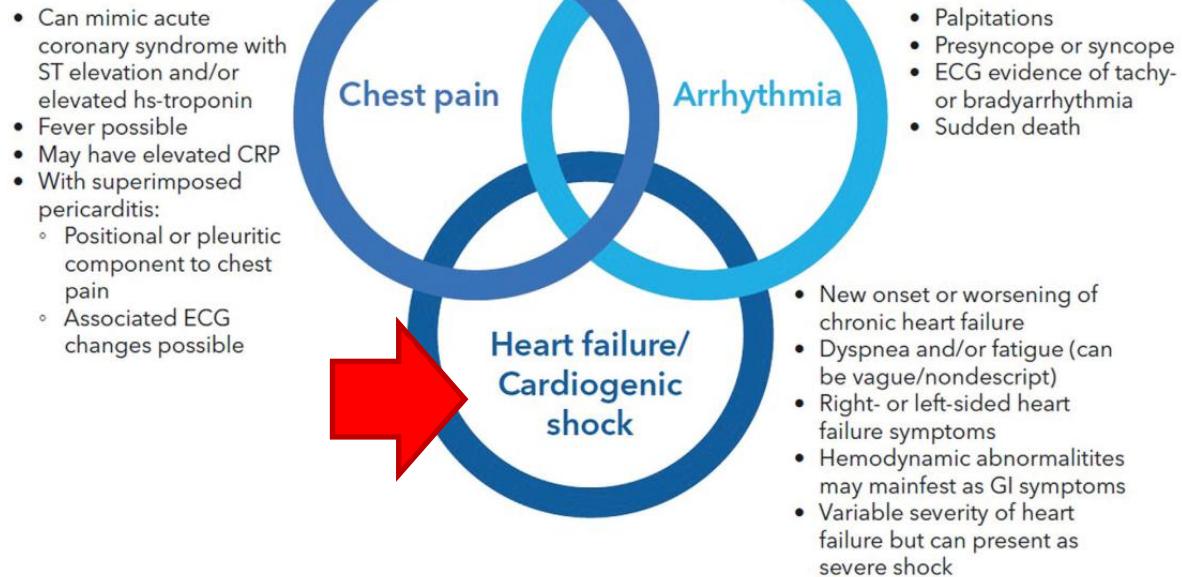
„zánětlivý myoperikardiální syndrom“



Myokarditididy – klinický obraz

2024 ACC Expert Consensus Decision Pathway on Strategies and Criteria for the Diagnosis and Management of Myocarditis

FIGURE 2 Three Classic Presentations of Myocarditis



Fulminantní myokarditida

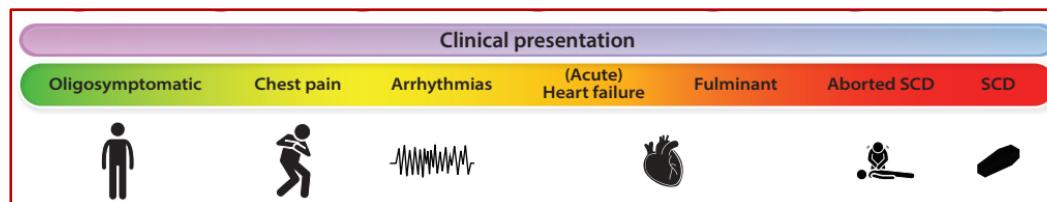
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4.2.2. Fulminant myocarditis

Fulminant myocarditis (occurring in 3%–9%) is characterized by cardiogenic shock at presentation for which haemodynamic support is needed.²⁸ Isolated right ventricle (RV) dysfunction is uncommon; however, biventricular failure occurs frequently in FM. Sustained ventricular arrhythmias (VA) may also occur in these patients (46.9%) and some present with sudden cardiac death (SCD) (25.8%).^{28,56,57}



Diagnostický algoritmus

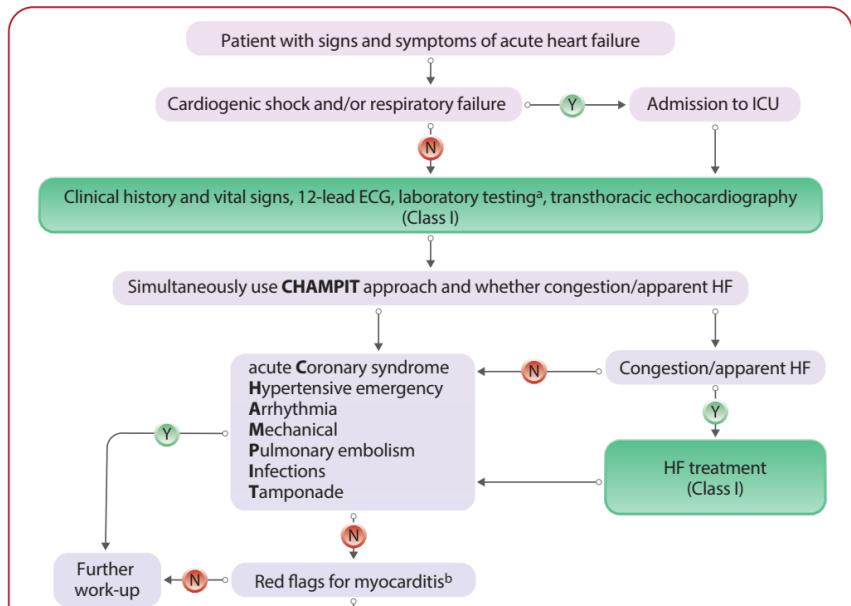


Table 6 Red flags for the clinical diagnosis of myocarditis

Myocarditis

- Recent or concomitant flu-like syndrome or gastroenteritis
- Infarct-like chest pain
- Palpitations
- HF symptoms
- ECG changes^a
- Ventricular arrhythmias (isolated, complex)
- Syncope
- Haemodynamic instability
- Elevated markers of myocardial lesion (hs-Tn, CK-MB elevation)
- Elevated markers of HF (NT-proBNP)
- Abnormal wall motion, increased wall thickness and/or impaired systolic function on imaging
- CMR imaging with myocardial oedema and/or LGE

Diagnostický algoritmus

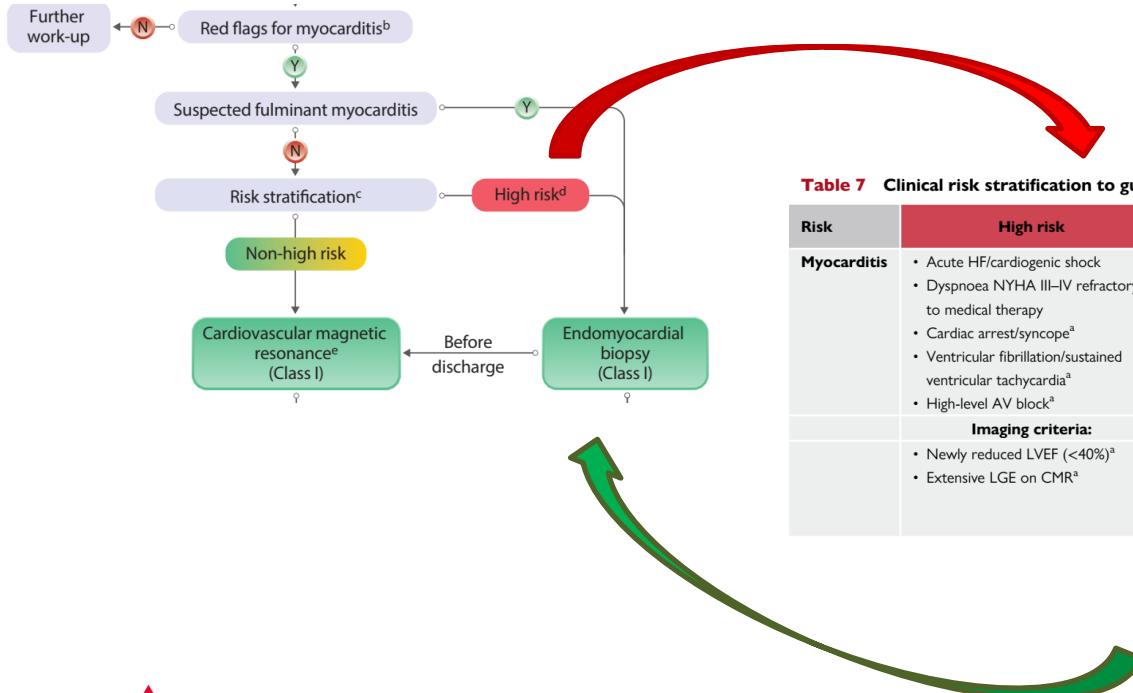
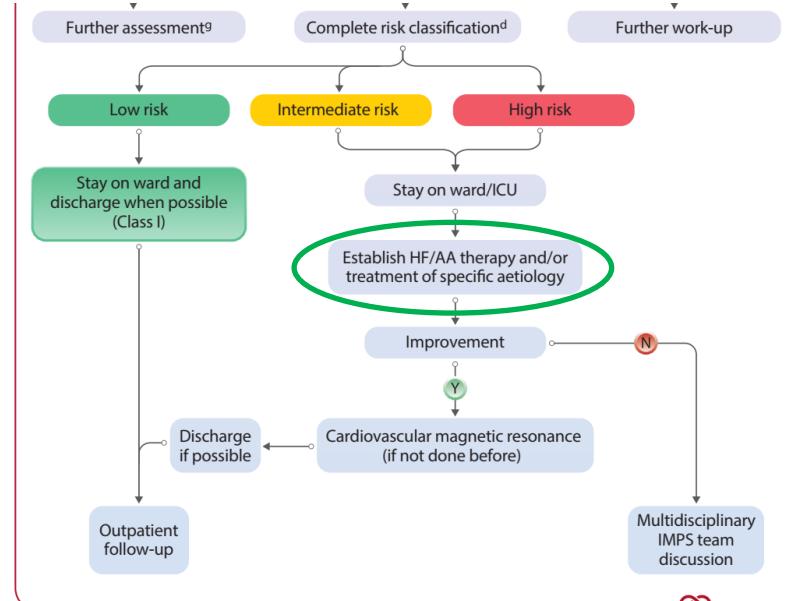
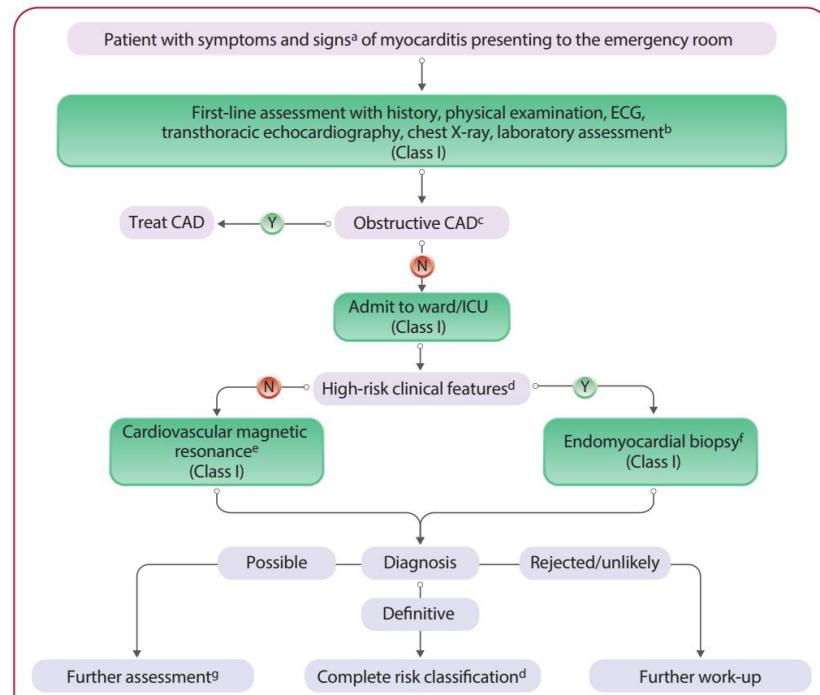


Table 7 Clinical risk stratification to guide

Risk	High risk
Myocarditis	<ul style="list-style-type: none"> • Acute HF cardiogenic shock • Dyspnoea NYHA III–IV refractory to medical therapy • Cardiac arrest/syncope^a • Ventricular fibrillation/sustained ventricular tachycardia^a • High-level AV block^a <p>Imaging criteria:</p> <ul style="list-style-type: none"> • Newly reduced LVEF (<40%)^a • Extensive LGE on CMR^a
	<p>High risk</p> <ul style="list-style-type: none"> • Acute HF cardiogenic shock • Dyspnoea NYHA III–IV refractory to medical therapy • Cardiac arrest/syncope^a • Ventricular fibrillation/sustained ventricular tachycardia^a • High-level AV block^a <p>Imaging criteria:</p> <ul style="list-style-type: none"> • Newly reduced LVEF (<40%)^a • Extensive LGE on CMR^a

Diagnosticko-terapeutický algoritmus

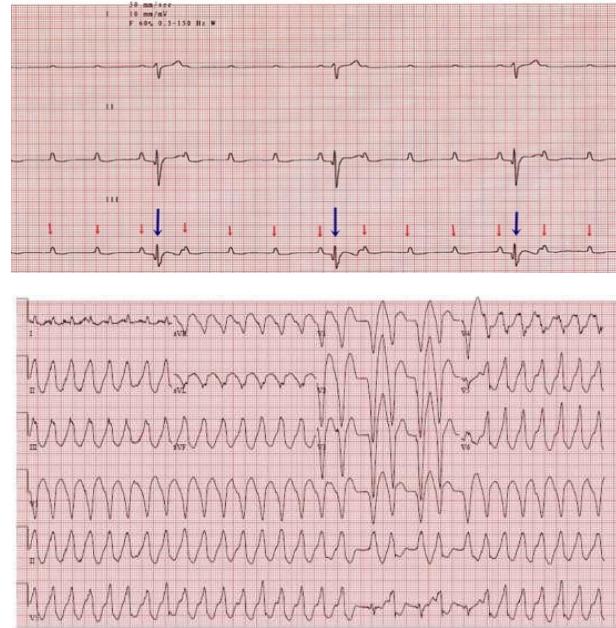


EKG v akutní fázi myokarditidy

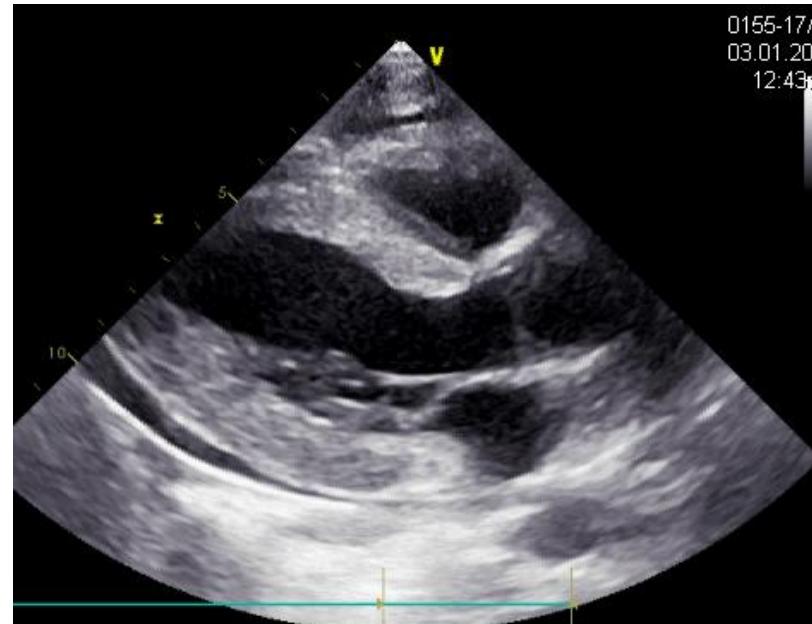
2024 ACC Expert Consensus Decision Pathway on Strategies and Criteria for the Diagnosis and Management of Myocarditis

4.2.1. Electrocardiogram

Electrocardiogram (ECG) is widely used as an initial screening tool for diagnosis of myocarditis, despite its **low sensitivity of 47%**.⁹ ECGs of patients with myocarditis often display a **myriad of nonspecific findings**, including **sinus tachycardia**, **nonspecific ST/T-wave changes**, **low voltage**, and **PR-segment depression**, **none of which are pathognomonic for myocarditis**.¹⁰ A normal ECG or ECG with nonspecific changes do not rule out myocarditis but **presence of pathological Q waves**, **left bundle branch block**, **wide complex QRS ≥ 120 ms**, **prolonged QT interval**, **high-grade atrioventricular (AV) block**, **malignant tachyarrhythmias**, **fragmented QRS**, and **T-wave inversion** are often associated with **decreased left ventricular function**, **presence of left ventricular scar** and overall poor clinical prognosis in patients with myocarditis.¹⁰⁻¹³ Involvement of the conduction system also raises the suspicion for **sarcoidosis**, **GCM**, or **Lyme disease**. Ongoing research,



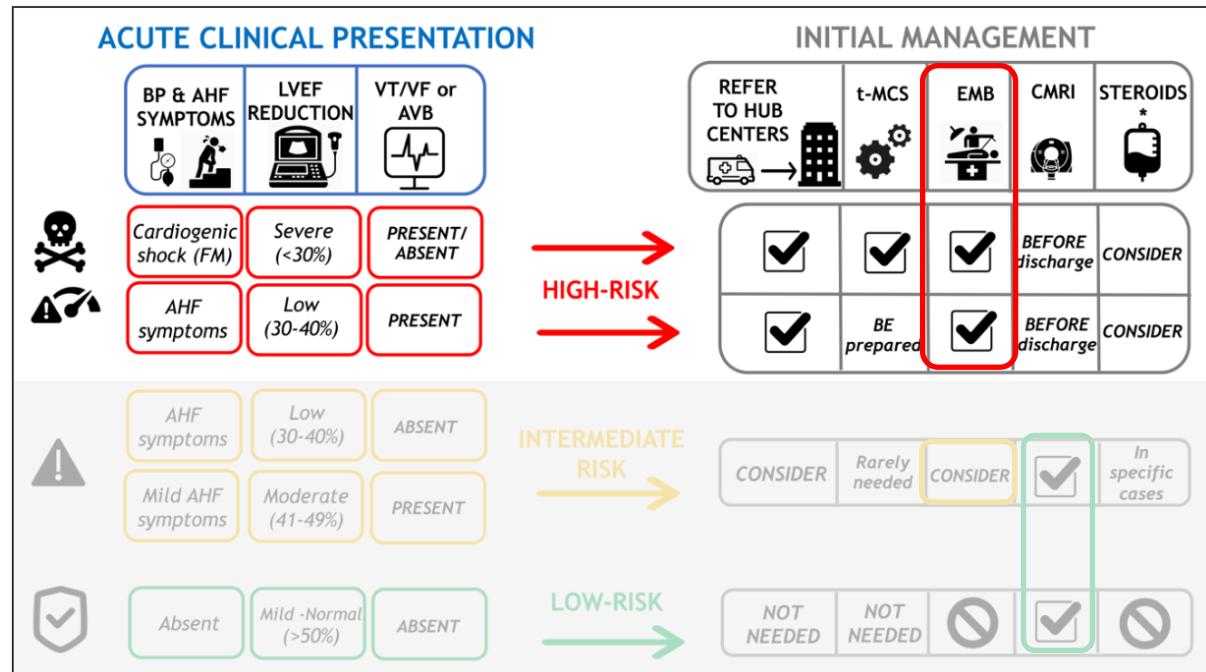
Echokardiografie v akutní fázi myokarditidy



Kdy indikovat EMB u nemocných s podezřením na myokarditidu?

Management of Acute Myocarditis and Chronic Inflammatory Cardiomyopathy

An Expert Consensus Document



Circ Heart Fail. 2020 Nov;13(11):e007405.

Kdy indikovat EMB u nemocných s podezřením na myokarditidu?

Recommendation Table 6 — Recommendations for endomyocardial biopsy (see Evidence Table 6)

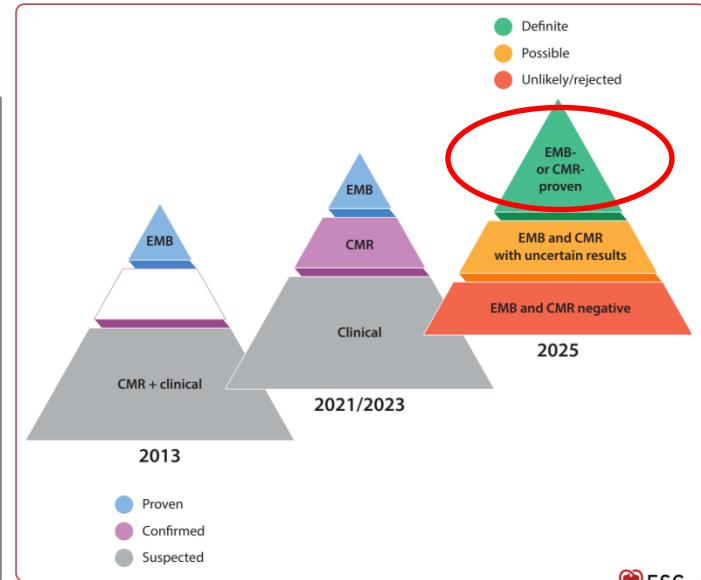
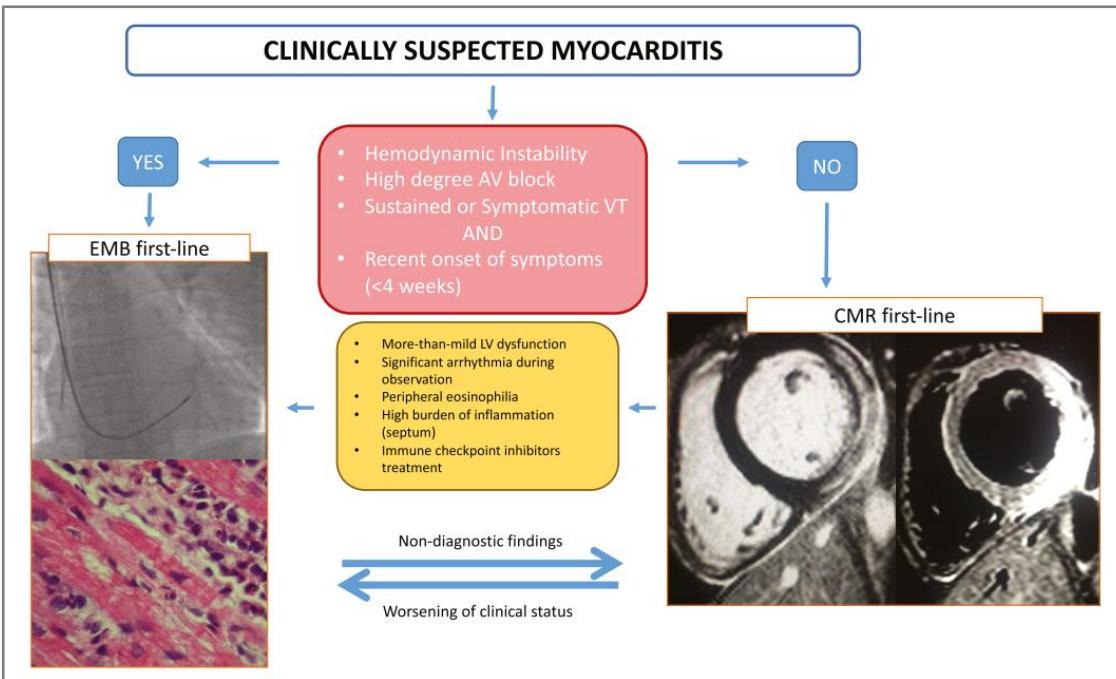
Recommendations	Class ^a	Level ^b	© ESC 2025
EMB ^c is recommended in patients with high-risk myocarditis ^d , and/or haemodynamic instability, and/or in patients with intermediate-risk myocarditis not responding to conventional therapy in order to detect a specific histologic subtype and to assess the presence of viral genome for treatment. ^{34,63,73,131}	I	C	

Table 7 Clinical risk stratification to guide work-up in inflammatory myopericardial syndrome

Risk	High risk	Intermediate risk	Low risk
Myocarditis	<ul style="list-style-type: none">Acute HF/cardiogenic shockDyspnoea NYHA III–IV refractory to medical therapyCardiac arrest/syncope^aVentricular fibrillation/sustained ventricular tachycardia^aHigh-level AV block^a <p>Imaging criteria:</p> <ul style="list-style-type: none">Newly reduced LVEF (<40%)^aExtensive LGE on CMR^a	<ul style="list-style-type: none">New/progressive dyspnoeaNon-sustained ventricular arrhythmiasPersistent release or relapsing troponin	Stable symptoms or oligosymptomatic
		<p>Imaging criteria:</p> <ul style="list-style-type: none">Newly mildly reduced LVEF (41%–49%) and/or WMAPreserved LVEF (≥50%) and LGE ≥2 segments on CMR	<p>Imaging criteria:</p> <ul style="list-style-type: none">Preserved LVEF (≥50%) without LGE or limited LGE (<2 segments) on CMR

Invazivní a neinvazivní dg nestojí proti sobě, ale doplňují se!

State-of-the-Art of Endomyocardial Biopsy on Acute Myocarditis and Chronic Inflammatory Cardiomyopathy



Potřebujeme před nasazením imunosuprese vždy EMB ?

FIGURE 10 Key Points Regarding the Use of Immunosuppressive Therapies in Myocarditis

Not all patients with myocarditis require immunosuppressive therapy

General consensus is to administer immunosuppressive therapy for the following conditions:

- Eosinophilic myocarditis
- Giant cell myocarditis
- Granulomatous myocarditis (sarcoid)
- Associated with immune checkpoint inhibitor therapy
- In setting of other autoimmune conditions

There remains lack of broad consensus but myocarditis experts from certain centers advise:

- Perform viral PCR on endomyocardial biopsy tissue to exclude active infection prior to initiation of immunosuppressive therapy
- Treat chronic lymphocytic myocarditis (with negative viral PCR) with immunosuppressive therapy

Implementation of immunosuppressive therapy

- Typically start with methylprednisolone boluses (7-14 mg/kg per day for 3 days) followed by oral prednisone taper (start at 1 mg/kg)
- Giant cell myocarditis requires higher level of immunosuppression than IV steroids, typically including a calcineurin inhibitor (cyclosporine or tacrolimus)
- Involve other specialty experts in setting of autoimmune conditions (eg, systemic lupus, vasculitis) as immunosuppressive strategy may be altered based on other organ involvement.

IVIG can be considered in the setting of inflammatory, antibody-mediated, or autoimmune disorders



Historické okénko

Immunosuppressive Therapy for Active Lymphocytic Myocarditis

Virological and Immunologic Profile of Responders Versus Nonresponders

Andrea Frustaci, MD; Cristina Chimenti, MD, PhD; Fiorella Calabrese, MD; Maurizio Pieroni, MD;
Gaetano Thiene, MD; Attilio Maseri, MD

Methods and Results—Out of 652 biopsied patients, 112 had a histological diagnosis of active lymphocytic myocarditis; 41 of these 112 patients were characterized by progressive heart failure despite conventional therapy and were treated with prednisone and azathioprine for 6 months. All were resubmitted to cardiac catheterization, angiography, and endomyocardial biopsy at 1 and 6 months and followed-up for 1 year. A total of 21 patients responded with prompt improvement in left ventricular ejection fraction from $25.7 \pm 4.1\%$ to $47.1 \pm 4.4\%$ and showed evidence of healed myocarditis at control biopsy. Conversely, 20 patients failed to respond and showed a histological evolution toward dilated cardiomyopathy: 12 remained stationary, 3 underwent cardiac transplantation, and 5 died. We retrospectively performed a polymerase chain reaction on frozen endomyocardial tissue for the most common cardiotropic viruses and assessed circulating serum cardiac autoantibodies. Viral genomes were present in biopsy specimens of 17 nonresponders (85%), including enterovirus (n=5), Epstein-Barr virus (n=5) adenovirus (n=4), both adenovirus and enterovirus (n=1), influenza A virus (n=1), parvovirus-B19 (n=1), and in 3 responders, who were all positive for hepatitis C virus. Cardiac autoantibodies were present in 19 responders (90%) and in none of the nonresponders.

Conclusions—In patients with active lymphocytic myocarditis, those with circulating cardiac autoantibodies and no viral genome in the myocardium are the most likely to benefit from immunosuppression. The beneficial effect of

Co je tzv. „viral shift“ u myokarditid podle ChatGPT?

Stručné shrnutí (hlavní body)

1. Historicky (20. stol., zejména 1970.–1990.) byly nejčastěji spojované s virovou myokarditidou **enteroviry** (zejména Coxsackievirus B). [Nature](#)
2. Po zavedení molekulárních metod (PCR) v 90.–00. letech začaly studie častěji detekovat **Parvovirus B19 (B19V)** a **human herpesvirus 6 (HHV6)** v endomyokardiálních biopsiích; v mnoha evropských sériích se B19V stal nejčastěji nalezeným virusem. [PubMed+1](#)
3. Kritická diskuse: u **B19V** (a částečně i HHV6) probíhá debata, zda přítomnost virového genomu v srdci vždy znamená, že virus aktivně způsobuje onemocnění, nebo je někdy „bystander“ (perzistence DNA v tkáni bez patogenetické role). [Wiley Online Library+1](#)

Potřebujeme před nasazením imunosuprese vždy EMB ?

Viral genome changes and the impact of viral genome persistence in myocardium of patients with inflammatory cardiomyopathy

Dalibor Mlejnek¹, Jan Krejci¹, Petr Hude¹,
Iva Svobodová¹, Tomas Freiberger³, Eva N.

to myocardial inflammation. We evaluated the change in viral presence after administration of immunosuppression therapy and compared it with the results of the group without immunosuppression. The only virus detected in the myocardium in this group was PVB19. To our best knowledge, such a study has never been published before. However, our pilot results from a small group of patients suggest that the administration of immunosuppression does not lead to a change in the viral presence or an increase in the viral load in follow-up biopsy samples.

Material and methods: We investigated the changes in viral presence and the impact of viral genome persistence in the myocardium on echocardiographic parameters, functional status and some laboratory parameters in a 6-month follow-up. Fifty-four patients with recent onset DCM, left ventricular ejection fraction < 40% and biopsy-proven myocarditis (> 14 mono-

tes/mm² and/or > 7 T-lymphocytes/mm²) were enrolled. In reaction (PCR) was performed to detect pathogens in the patients were divided according to the administered therapy: failure medication (46 patients) and immunosuppressive agents).

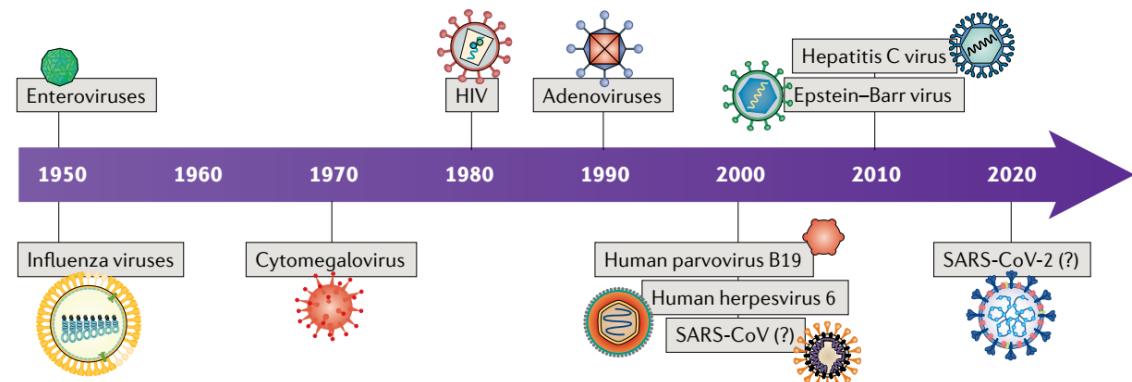
standard heart failure medication group viral clearance was patients and viral persistence in 24 patients in the follow-up in both groups, there was no statistically significant difference of 12.0 ±11.4% vs. 18.3 ±12.6%, decrease in NYHA 7 vs. 1.0 ±0.7, decline in NT-proBNP of 1335 ±1933 ng/l vs. 11 and decrease in infiltrating leukocytes of 11.1 ±15.8 vs. 6.7 and T-lymphocytes of 5.8 ±15.1 vs. 1.8 ±10.9 cells/mm² (all in PCR positive patients from 37 to 29 was observed. The 19 positive PCR findings decreased from 5 to 4 in patients ppressive therapy.

decrease in the number of positive PCR findings in control endomyocardial biopsy was observed. Viral genome persistence was not associated with worse outcome in short-term follow-up.

Viral shift

Myocarditis and inflammatory cardiomyopathy: current evidence and future directions

Carsten Tschöpe^{1,2,3}✉, Enrico Ammirati⁴, Biykem Bozkurt^{5,6}, Alida L. P. Caforio¹⁰⁷,
Leslie T. Cooper⁸, Stephan B. Felix^{9,10}, Joshua M. Hare¹¹, Bettina Heidecker¹²,
Stephane Heymans^{13,14}, Norbert Hübner¹⁰^{2,15,16}, Sebastian Kelle^{2,3,17}, Karin Klingel¹⁸,
Henrike Maatz¹⁵, Abdul S. Parwani³, Frank Spillmann³, Randall C. Starling¹⁰¹⁹,
Hiroyuki Tsutsui²⁰, Petar Seferovic²¹ and Sophie Van Linthout^{1,2}



Tschöpe C et al, *Nat Rev Cardiol.* 2021 Mar;18(3):169-193

Platí stále „stará dogmata“ z jiné éry?

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Implementation of immunosuppressive therapy

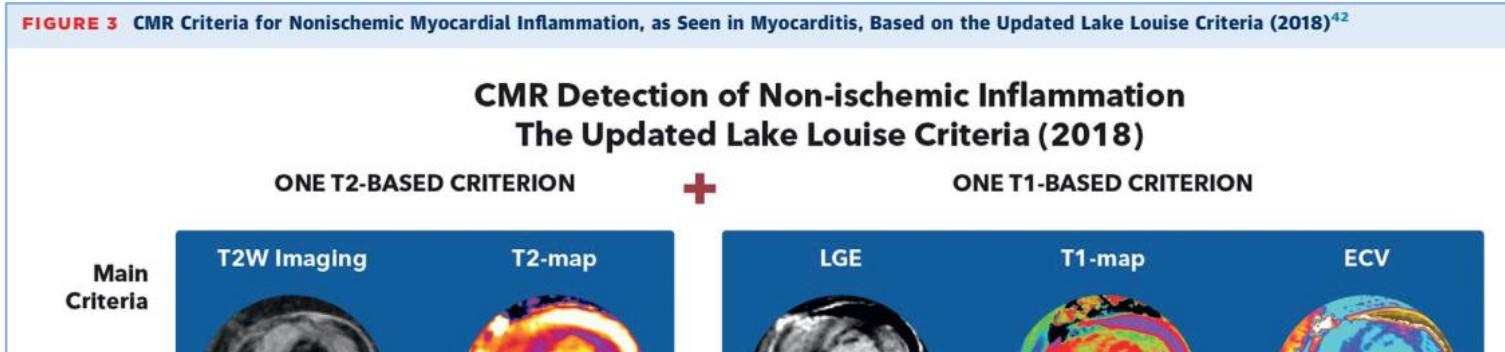
- Typically start with methylprednisolone boluses (7-14 mg/kg per day for 3 days) followed by oral prednisone
- Giant cell myocarditis: Immunosuppressive therapy (eg, cyclophosphamide, rituximab) including a calcineurin inhibitor
- Involve other specialty experts in setting of autoimmune conditions (eg, systemic lupus, vasculitis) as immunosuppressive strategy may be altered based on other organ involvement.

U PVB19, ev. u HHV6 v drtivé většině nikoliv!

IVIG can be considered in the setting of inflammatory, antibody-mediated, or autoimmune disorders



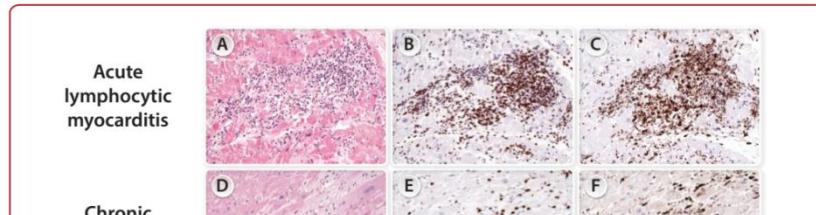
Fulminantní myokarditida – neinvazivní diagnostika – MRI



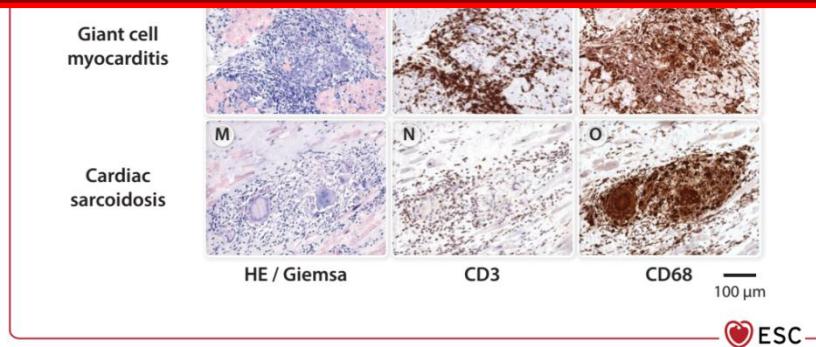
**MRI je zcela klíčovým vyšetřením při neinvazivní diagnostice MC.
Role u fulminantní myokarditidy je ale vedlejší – až po EMB!**



Jen EMB s (imuno)histologií určí podtyp myokarditidy...



Léčba v akutní fázi je podobná...ale u specifických forem myokarditidy je třeba individualizace a doplnění kortikoterapie dalšími skupinami imunomodulačních léků



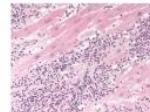
IS/IM terapie u susp. fulminantní myokarditidy

Immunomodulating Therapies in Acute Myocarditis and Recurrent/Acute Pericarditis

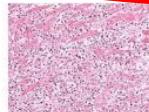
FIRST-LINE

SUSPECTED FULMINANT OR COMPLICATED ACUTE MYOCARDITIS

↓
i.v. pulse methylprednisolone 7–14 mg/kg/day for 3 d, then 1 mg/kg/day



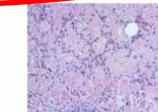
LYMPHOCYTIC



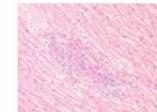
ICI-ASSOCIATED



Giant cell



EOSINOPHILIC



SARCOIDOSIS

ADDITIONAL

- If associated systemic autoimmune disorders (eg. SLE and APS): add aggressive treatment of associated conditions

- Hold ICI therapy
- Confirm ICI-myocarditis via definitive imaging and/or endomyocardial biopsy

- If hemodynamically unstable pts: ATG, from 1 mg/kg, usually single-dose to 300 mg in 3 days or (alternative) i.v. alemtuzumab (anti-CD52 antibody) single dose of 30 mg plus oral CyA, BID, target trough levels 150–250 ng/mL
- If hemodynamically stable pts: only oral CyA, BID, target trough levels 150–250 ng/mL

- If EGPA: consider i.v. cyclophosphamide (especially in ANCA-positive pts), 600 mg/m² at days 1, 15, and 30
- If clonal (myeloproliferative) HES: imatinib 100–400 mg OD
- If helminthic infection: albendazole 400 mg BID for 2–4 wk
- If hypersensitivity reaction: withdraw suspected drug

SECOND-LINE

IVIG (2 g/kg), single continuous infusion in 24–48 h or divided in 4 d or plasmapheresis, 3–5 sessions in 5–10 d

i.v. abatacept (a CTLA-4 agonist) or ATG, 1 mg/kg, usually single dose or i.v. alemtuzumab (anti-CD52 antibody), 30 mg, single dose

i.v. rituximab 375 mg·m² (BSA) mg (once a wk for 4 wk and then every 4 mo as maintenance therapy) for 1 yr

- If DRESS, EGPA or idiopathic HES: anti-ILS agents (e.g., benralizumab 30 mg s.c./4–8 wk or mepolizumab 100–300 mg/4wk)

s.c. methotrexate 15–20 mg/wk or i.v. infliximab 5 mg/kg (up to 500 mg) at time 0 and after 2 and 4 wk and then every 6–8 wk or s.c. adalimumab 40 mg/2wk

IS/IM terapie u susp. fulminantní myokarditidy

Table 12 Therapy for specific forms of myocarditis

Lymphocytic myocarditis (virus-negative)	
1st line therapy	<u>Non-severe:</u> prednisone 1 mg/kg/day p.o. then tapered <u>Severe:</u> i.v. methylprednisolone 7–14 mg/kg/day for 3 days, then 1 mg/kg/day p.o.
2nd line therapy	Oral corticosteroids + azathioprine ^a or mycophenolate mofetil ^b , cyclosporine ^c , methotrexate ^d
3rd line therapy	IVIG ^e or plasmapheresis ^f
Eosinophilic myocarditis	
1st line therapy	Same as lymphocytic myocarditis + Treat EM-associated condition if identified
2nd line therapy	Same as lymphocytic myocarditis + Treat EM-associated condition if identified
3rd line therapy	–
Giant-cell myocarditis	
1st line therapy	<u>Non-severe:</u> prednisone 1 mg/kg/day p.o. then tapered <u>Severe:</u> i.v. methylprednisolone 7–14 mg/kg/day for 3 days, then 1 mg/kg/day p.o. + immunosuppressive (azathioprine ^a or mycophenolate mofetil ^b , cyclosporine ^c)
2nd line therapy	Antithymocyte Globulin (ATG) ^g cyclophosphamide ^h , rituximab ⁱ
3rd line therapy	–
Cardiac sarcoidosis	
1st line therapy	<u>Non-severe:</u> prednisone 1 mg/kg/day p.o., tapering from 40–60 mg daily <u>Severe:</u> i.v. methylprednisolone 7–14 mg/kg/day for 3 days, then 1 mg/kg/day p.o.
2nd line therapy	Methotrexate ^d (1st choice), or azathioprine ^a mycophenolate mofetil ^b , cyclophosphamide ^h
3rd line therapy	Infliximab ^j or adalimumab ^k , rituximab ⁱ

Myokarditidy a ZKMP – IS léčba

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Immunosuppressive therapy

Corticosteroids should be considered in patients with fulminant, non-infectious forms of myocarditis to stabilize the patients.

IIa

C

Corticosteroids may be considered in patients with acute myocarditis with impaired LVEF if refractory to standard HF therapy to stabilize patients.

IIb

C

Routine use of immunosuppressive therapy is not recommended in acute myocarditis with preserved LV function because no outcome benefit has been shown.

III

C

Časná invazivní strategie a IS/IM th je lepší!



ESC

European Society
of Cardiology

European Heart Journal (2023) 44, 5110–5124
<https://doi.org/10.1093/eurheartj/ehad707>

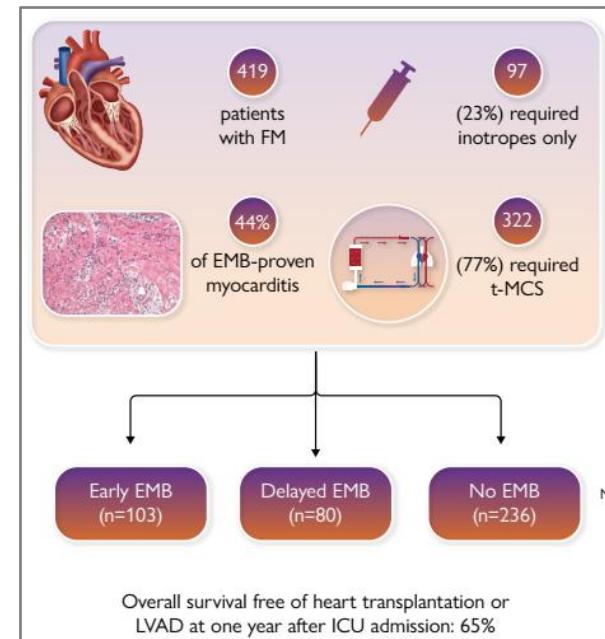
CLINICAL RESEARCH

Acute cardiovascular care

Fulminant myocarditis proven by early biopsy and outcomes

Background and Aims While endomyocardial biopsy (EMB) is recommended in adult patients with fulminant myocarditis, the clinical impact of its timing is still unclear.

Methods Data were collected from 419 adult patients with clinically suspected fulminant myocarditis admitted to intensive care units across 36 tertiary centres in 15 countries worldwide. The diagnosis of myocarditis was histologically proven in 210 (50%) patients, either by EMB ($n = 183$, 44%) or by autopsy/explanted heart examination ($n = 27$, 6%), and clinically suspected cardiac magnetic resonance imaging confirmed in 96 (23%) patients. The primary outcome of survival free of heart transplantation (HTx) or left ventricular assist device (LVAD) at 1 year was specifically compared between patients with early EMB (within 2 days after intensive care unit admission, $n = 103$) and delayed EMB ($n = 80$). A propensity score-weighted analysis was done to control for confounders.



Huang *et al*, EHJ 2023

Histologická charakteristika

Table 1 Baseline characteristics, clinical, echocardiography, and biological findings at intensive care unit admission according to endomyocardial biopsy timing after intensive care unit admission

	Available data, N	All patients (N = 419)	EMB ≤ 2 days (n = 103)	EMB > 2 days (n = 80)	No EMB (n = 236)	P-value
Age, years	419	40 (29–52)	44 (31–55)	41 (28–52)	38 (28–49)	.091
Woman, n (%)	419	220 (53)	58 (56)	33 (41)	129 (55)	.078
EMB finding consistent with myocarditis^b	183	183 (44)				
Lymphocytic infiltrate		125 (68)	71 (69)	54 (68)	-	.836
Giant cell infiltrate		20 (11)	11 (11)	9 (11)	-	.902
Eosinophilic infiltrate		20 (11)	11 (11)	9 (11)	-	.902
Other infiltrate^c		18 (10)	10 (10)	8 (10)	-	.948
Histologic demonstration of myocarditis on autopsy/explanted heart/myocardial specimen after LVAD	419	27 (6)	0 (0)	0 (0)	27 (11)	<.001
Myocarditis proven by CMRI	419	158 (38)	36 (35)	22 (28)	100 (42)	.048

Jaká byla léčba?

Table 2 In-intensive care unit management, complications, and outcomes according to endomyocardial biopsy timing after intensive care unit admission

	Available data, N	All patients (N = 419)	EMB ≤ 2 days (n = 103)	EMB > 2 days (n = 80)	No EMB (n = 236)	P-value
Inotropes/vasopressors						
Inotropes/vasopressors duration, days	419	7 (3–14)	8 (4–13)	16 (7–25)	6 (2–10)	<.001
Dobutamine	419	332 (79)	80 (78)	65 (81)	187 (79)	.839
Norepinephrine	419	316 (75)	77 (75)	60 (75)	179 (76)	.973
Epinephrin	419	159 (38)	42 (41)	30 (38)	87 (37)	.789
Levosimendan	419	45 (11)	11 (11)	14 (18)	20 (9)	.079
Only inotropes/vasopressors without t-MCS	419	97 (23)	24 (23)	14 (18)	59 (25)	.389
Temporary MCS						
Any t-MCS	419	322 (77)	79 (77)	66 (82)	177 (75)	.389
IABP stand-alone	419	27 (6)	16 (16)	1 (1)	10 (4)	<.001
VA-ECMO/Impella®	419	295 (70)	63 (61)	65 (81)	167 (71)	.013
t-MCS duration, days	322	6 (4–11)	6 (4–10)	12 (7–19)	5 (3–8)	<.001
Invasive mechanical ventilation	419	334 (80)	73 (71)	64 (80)	197 (84)	.030
Duration, days	334	7 (4–15)	7 (5–15)	14 (8–24)	7 (3–13)	<.001

Časná invazivní strategie a IS/IM th je lepší!



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CLINICAL RESEARCH Acute cardiovascular care

Fulminant myocarditis proven by early biopsy and outcomes

Table 5 Immunomodulatory therapy and one-year outcome in the group of endomyocardial biopsy-proven myocarditis ($n = 183$) according to main histologic subtypes

	One-year outcome		P-value
	Alive without HTx or LVAD	Death, HTx or LVAD	
Lymphocytic myocarditis ($n = 125$)	<i>n</i> = 78	<i>n</i> = 47	
Any IMT	54 (69)	26 (55)	.117
Early use of IMT ^a	50 (64)	16 (34)	.001
Corticosteroids	54 (69)	23 (49)	.024
Pulse therapy	50 (64)	18 (38)	.005
Other IMT ^b	10 (13)	6 (13)	.993
Intravenous immunoglobulins	9 (12)	6 (13)	.838
Multiple IMT	15 (19)	8 (17)	.757

	One-year outcome		P-value
	Alive without HTx or LVAD	Death, HTx or LVAD	
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Multiple IMT	15 (19)	8 (17)	.757

Other IMT	4 (6)	11 (23)	.768
Intravenous immunoglobulins	2 (40)	4 (27)	.573
Multiple IMT	4 (80)	11 (73)	.766

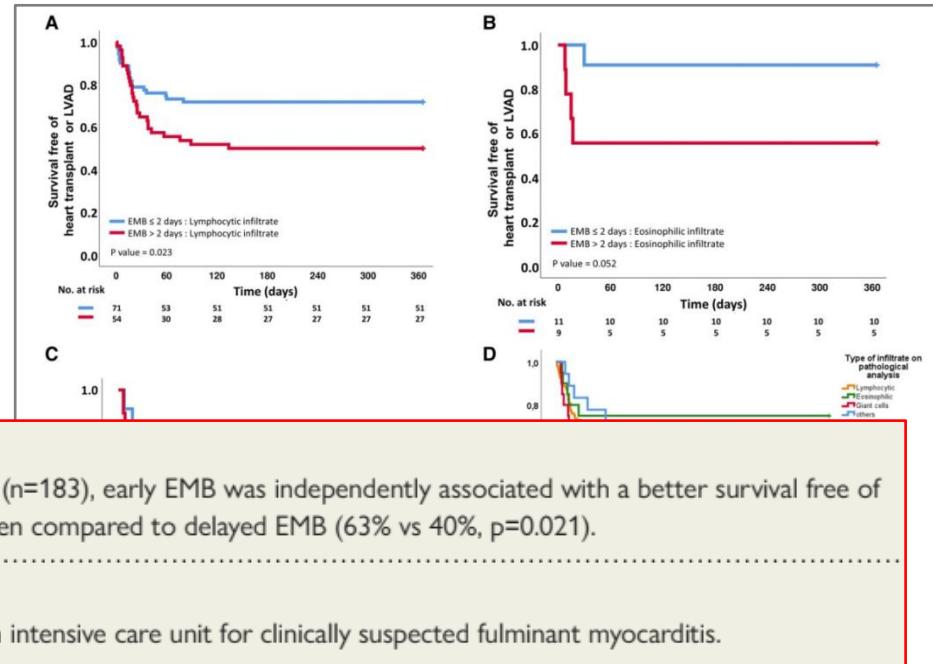
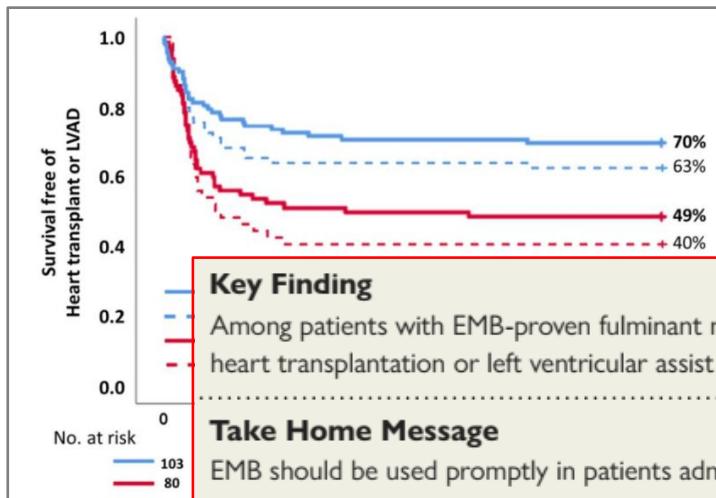
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CLINICAL RESEARCH
Acute cardiovascular care

Fulminant myocarditis proven by early biopsy and outcomes



Léčba fulminantní myokarditidy

Recommendations	Class ^a	Level ^b
A timely and dedicated Shock Team discussion is recommended in patients with myocarditis in the presence of haemodynamic compromise, to decide on the need for escalation to MCS and to determine a long-term management plan.	I	C
Temporary MCS ^c should be considered in patients with myocarditis and cardiogenic shock or acute decompensation in chronic myocarditis to stabilize the patients.	IIa	C

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6.3. Interventional techniques including circulatory support

6.3.1. Myocarditis

6.3.1.1. Short-term mechanical circulatory support

Myocarditis patients who present with rapid deterioration in haemodynamic status and rapidly progressive myocardial dysfunction are amongst those who respond best to temporary MCS.²⁹³ Among temporary MCS, veno-arterial extracorporeal membrane oxygenation (VA-ECMO) represents the most frequently applied or recommended approach, ranging from 75% to 85% of AM cases.^{75,294–296} Despite the

Léčba fulminantní myokarditidy

Diagnosis and management of patients with fulminant myocarditis

Nicoletta D'Ettore  ^{1*}, Kaveh Eghbalzadeh ², Mehmet Oezkur  ³,
Letizia F. Bertoldi ⁴, Matthias Bossard ⁵, and Federico Pappalardo  ⁶

Ventricular unloading via MCS devices can play a crucial role in mitigating the adverse effects of viral injury and autoimmune responses in myocarditis. The benefits of ventricular unloading include the following:

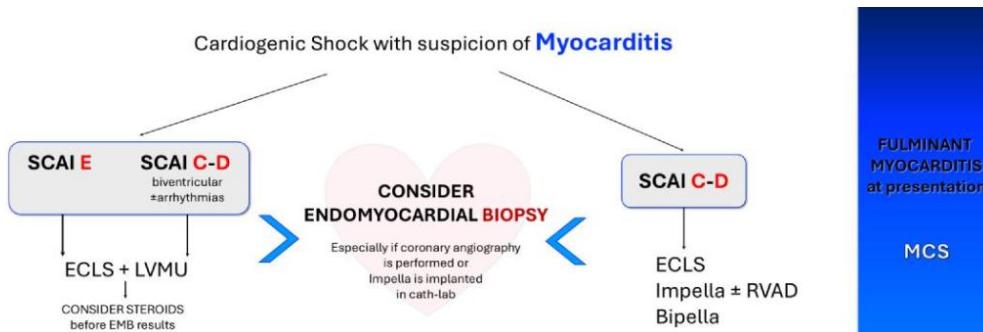
- (1) **Decreased workload of the heart:** Ventricular unloading reduces myocardial stress and oxygen demand, which can limit further myocyte damage caused by the inflammatory response and preserve myocardial function.
- (2) **Improved coronary perfusion:** Devices like microaxial flow pumps enhance coronary blood flow, ensuring better oxygen and nutrient delivery to the myocardium, which aids in the repair of damaged myocardial tissue, and reduces the extent of injury.

- (3) **Modulation of the immune response:** Reducing myocardial stress and improving perfusion can indirectly modulate the immune response by decreasing the levels of stress-related inflammatory cytokines, mitigating the autoimmune component of myocarditis, and preventing further myocardial damage.
- (4) **Enhanced recovery environment:** Ventricular unloading creates a more favourable environment for myocardial recovery by minimizing further injury and providing the heart with a chance to heal. This can be particularly beneficial in the context of viral myocarditis, where ongoing viral replication and immune-mediated injury are common.

Léčba fulminantní myokarditidy – kterou st-MCS použít?

Diagnosis and management of patients with fulminant myocarditis

Nicoletta D'Ettore  ^{1*}, Kaveh Eghbalzadeh ², Mehmet Oezkur  ³, Letizia F. Bertoldi ⁴, Matthias Bossard ⁵, and Federico Pappalardo  ⁶

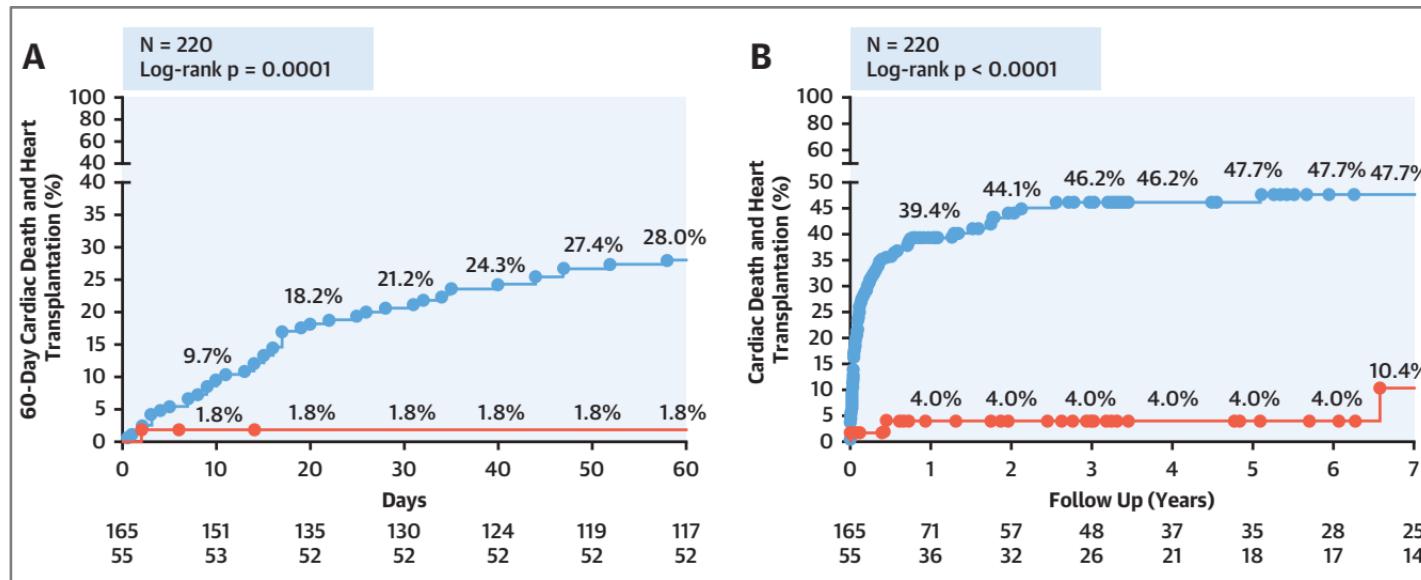


experience. The most frequently used short-term MCS in FM patients is VA-ECMO. However, during VA-ECMO support, LV distention and increased afterload may cause pulmonary oedema and hinder myocardial recovery. An early and effective unloading strategy should be associated. The combined ECMELLA (VA-ECMO + Impella) provides potent haemodynamic support, oxygenation, and ventricular unloading and is associated with lower mortality compared to VA-ECMO alone, despite higher complication rates. The combination in ECMELLA or BIPELLA should be considered when the right ventricular function is compromised.^{31,32}

Prognóza fulminantní vs nefulminantní myokarditidy

Fulminant Versus Acute Nonfulminant Myocarditis in Patients With Left Ventricular Systolic Dysfunction

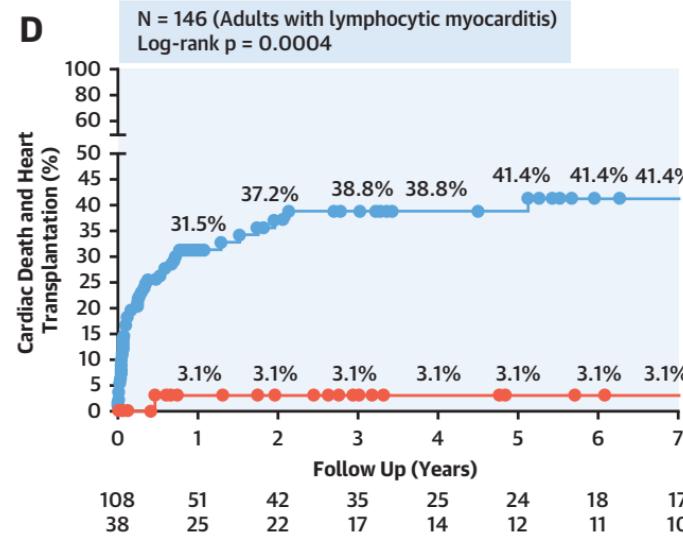
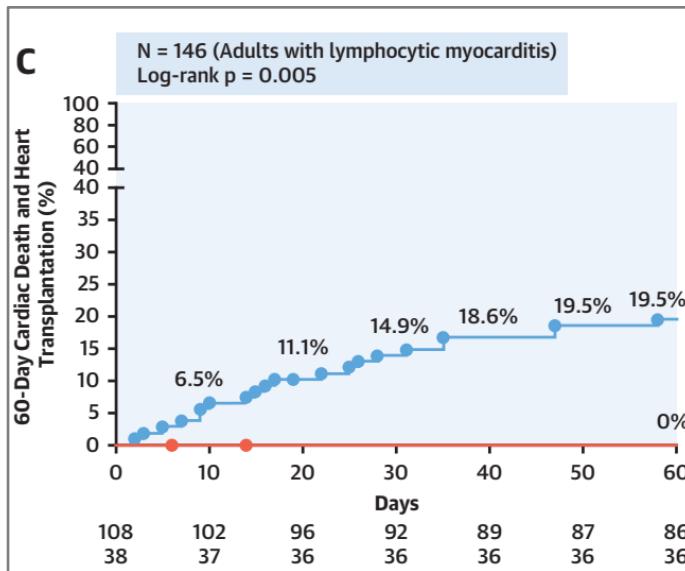
● Fulminant Myocarditis ● Nonfulminant Myocarditis



Prognóza fulminantní vs nefulminantní lymfocytární myokarditidy

Fulminant Versus Acute Nonfulminant Myocarditis in Patients With Left Ventricular Systolic Dysfunction

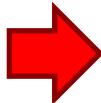
● Fulminant Myocarditis ● Nonfulminant Myocarditis



Restrikce fyzické zátěže

STAGE D MYOCARDITIS

- ICU admission
- Refer to an advanced HF center with a myocarditis team



- Treat arrhythmia
- Hemodynamic support including temporary circulatory support, as needed
- Endomyocardial biopsy
- Pharmacological treatment
 - Immunosuppression
 - Directed at etiology
- GDMT for HF and shock
- Consider durable LVAD or heart transplant if no recovery
- **Restrict strenuous physical activity for 3-6 months (avoid excessive sedentary behavior)**

Recommendation	Class ^a	Level ^b
Restriction of physical exercise until remission, for at least 1 month, is recommended in athletes and non-athletes after IMPS using an individualized approach to accelerate recovery.	I	C

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Prevence SCD u nemocných s akutní myokarditidou

2025 ESC Guidelines for the management of myocarditis and pericarditis

Developed by the task force for the management of myocarditis and pericarditis of the European Society of Cardiology (ESC)

Endorsed by the Association for European Paediatric and Congenital Cardiology (AEPC) and the European Association for Cardio-Thoracic Surgery (EACTS)

ICD in myocarditis

Secondary prevention

ICD implantation is recommended in patients with non-active^c myocarditis and haemodynamically not-tolerated sustained VT to prevent SCD.^{78,79,322,336}

I	C
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ICD implantation should be considered in patients with non-active^c myocarditis and haemodynamically tolerated sustained VT to prevent SCD.^{78,79,322,336}

IIa	C
-----	---

ICD implantation may be considered in patients with acute myocarditis and sustained VA (VT/VF) in the acute phase to prevent SCD.^{71,79,89,222,323–325}

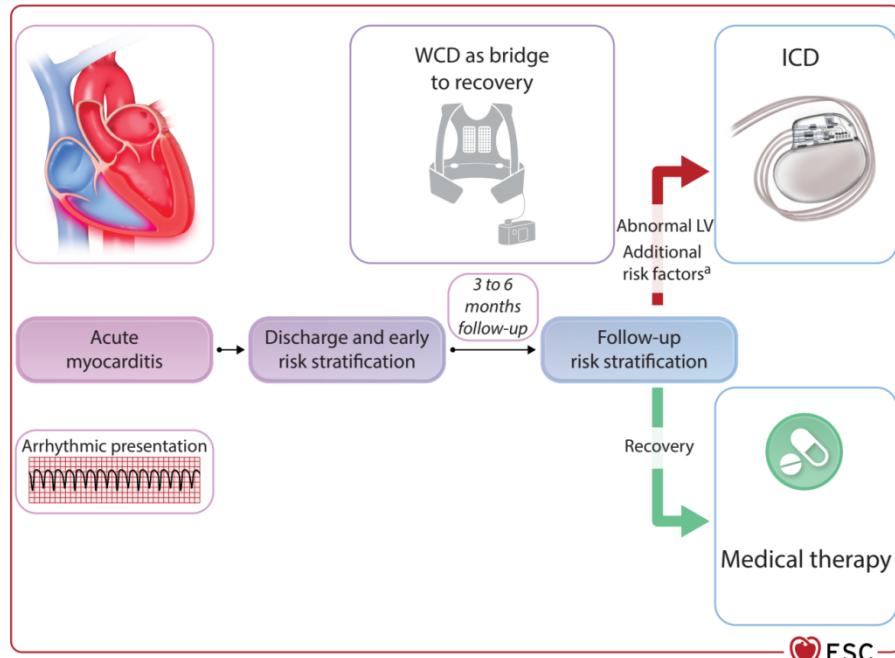
IIb	C
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Primary prevention

ICD implantation may be considered in patients with myocarditis after the acute phase (3–6 months) and persistent risk factors for VA^d to prevent SCD.^{89,332–334,336}

IIb	C
-----	---

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...snaha o zdrženlivost v akutní fázi myokarditidy...

Dlouhodobé sledování nemocných po FM je nezbytné!

Recommendations	Class ^a	Level ^b
Follow-up with clinical assessment, biomarkers ^c , ECG, exercise test, Holter-ECG monitoring, echocardiography, and CMR at least within 6 months after the index hospitalization is recommended in all patients with myocarditis to identify a potential progression or new risk factors. ⁶²	I	C
Long-term follow-up is recommended for patients with complicated myocarditis ^d to identify a potential progression or new complications. ^{28,74}	I	C

		Within 1 month	Within 3–6 months	12 months	>1 year and long-term FU ^a
Clinical evaluation and ECG	Myocarditis	×	×	×	×
Biomarkers (TnI, C-reactive protein)	Myocarditis	×	×	(X)	(X)
Rhythm (stress and/or Holter-ECG)	Myocarditis	–	×	(X)	(X)
Imaging myocarditis	TTE	X ^b		X ^c	X ^c
	CMR	X ^b		X ^c	X ^c

Závěry

- Fulminantní myokarditida je onemocnění s vysokou krátko- i dlouhodobou mortalitou a morbiditou.
- Invazivní diagnostický přístup a komplexní farmakologická i nefarmakologická léčba může pomoci překlenout akutní fázi onemocnění.
- V post-akutní fázi je třeba kardiologický follow-up zaměřený na stratifikaci rizika SCD a včasné detekce progrese onemocnění vyžadující LVAD / HTx.
- Nezbytná je koncentrace závažných forem MC do terciálních center s dostupností komplexní diagnostiky a přístrojové léčby, ale také se zkušeností s IS / IM léčbou a s návazností na LVAD/HTx programy.



Děkuji za pozornost!