Original article

Myocarditis – clinical course and 2-year outcome in 32 patients

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Abstract

Background: Acute myocarditis is one of the most challenging diagnoses in cardiology. It is a disease with variable clinical presentation, progression and outcome.

Aim: To assess clinical characteristics and outcome of patients hospitalised with diagnosis of acute myocarditis from year 2006 to 2008.

Methods: We analysed hospital files of consecutive 32 patients admitted to our hospital due to myocarditis. All demographic, clinical and laboratory data were analysed and compared between patients with acute or subacute myocarditis. After discharge the patients were followed for 8-24 months.

Results: The majority of patients were males (84%) in a mean age of 33 years. Clinical and echocardiographic parameters improved in 25 (78%) of patients during hospital stay. During follow-up decreased left ventricular ejection fraction (LVEF) was observed more often in patients with subacute than acute myocarditis (mean LVEF values of 49 vs. 61%, respectively). Patients with a subacute form of the disease more frequently required chronic pharmacological therapy and more often retired from occupational activities.

Conclusions: Diagnosis of myocarditis is still challenging. Careful history taking, serial laboratory, ECG and echocardiographic examinations are helpful in therapeutic decisions making and assessing prognosis. Patient with subacute myocarditis are more symptomatic than patients with acute myocarditis.

Key words: myocarditis, clinical course, prognosis

Introduction

Myocarditis is a disease of varying aetiology with inflammation comprising cardiomyocytes, intracellular tissue, vessels or pericardium which may finally lead to post-inflammatory cardiomyopathy and heart failure [1]. The infectious factor is related to geographic location and viruses are the most common cause on our continent. According to O'Connell up to 70% of the population is at risk of contact with cardiotropic viruses, however despite that myocarditis occurs sporadically [2]. According to the latest reports, biopsy specimens from patients with myocarditis most often disclose enteroviruses (mainly *Coxsackie*), adenoviruses, influenza viruses, parvoviruses and herpesviruses, and less likely hepatoviruses [3]. Clinical manifestation of viral myocarditis is highly heterogenic and is a consequence of interaction between a virus and the human organism. Symptoms may be scarce, with asymptomatic clinical course in some patients. The disease may be characterised by severe general and/or local symptoms, imitate myocardial infarction or lead to arrhythmias, acute heart failure and even cardiogenic shock [4].

The aim of the study was to assess the clinical course and outcomes in patients with myocarditis hospitalised between 2006 and 2008.

Methods

The study included all patients hospitalised for myocarditis between 1 January 2006 and 1 May 2008. There are no firm diagnostic criteria for myocarditis in the clinical setting; therefore patients were diagnosed with myocarditis based on the history of upper respiratory or gastrointestinal tract infection with the following symptoms present on admission: marked limitation of exercise tolerance, chest pain, arrhythmias or new onset symptoms of heart failure with elevated activity of myocardial necrosis markers in plasma: creatine kinase (CK), myocardial fraction of creatine

kinase (CK-MB) and/or increased concentration of troponin T (TnT), increased parameters of inflammation such as concentration of high-sensitivity C-reactive protein (hsCRP), erythrocyte sedimentation rate (ESD), leucocytosis with absence of other identifiable causes of the clinical symptoms or abnormal laboratory findings [1, 3, 5]. Each patient underwent serial electrocardiographic recordings (ECG), 24-hour ambulatory Holter monitoring focused on the assessment of coexisting arrhythmias and conduction disorders, and echocardiography for the assessment of left ventricular (LV) systolic function.

Patients with diagnosed myocarditis were divided into two groups with different history and clinical course. Rapid onset of the disease with directly preceding respiratory or gastrointestinal tract infection, chest pain, pericardial effusion, elevated concentration of inflammatory parameters or myocardial necrosis markers was classified as the acute myocarditis group. The subacute myocarditis group included patients with undetermined onset of the disease or with persistent symptoms (dyspnoea, fatigue, tiredness, arrhythmias, unspecific chest pain) associated with infection up to 4 weeks prior to hospitalisation, with LV systolic dysfunction on echocardiography, elevated concentration of inflammatory and/or myocardial necrosis markers and slowly diminishing or persistent clinical symptoms. A follow-up visit was performed between 8 and 24 months with clinical, electrocardiographic and echocardiographic assessment.

Results

There were 6590 patients admitted to the department between 1 January 2006 and 1 May 2008, of whom 32 were diagnosed with myocarditis (0.53% of all patients). Mean age of patients was 33 years and 25 subjects were below 40 years of age (Table I). The majority of the patients were male. Seasonal morbidity (autumn and winter) was observed mostly in patients with subacute myocarditis. Symptoms of upper respiratory or gastrointestinal tract infection before admission were present in 75% of patients with myocarditis and in all patients with acute type of the disease. Other symptoms reported by patients included pericardial chest pain [retrosternal resting pain with varying character related to the body position in 21 (65%) patients mostly with acute myocarditis] or anginal chest pain, palpitations, decreased exercise tolerance or joint pain. General symptoms such as fever or subfebrile state, fatigue or sweating were present in one third of patients. Symptoms of heart failure with fluid retention in the pulmonary and peripheral circulation, arrhythmias and dyspnoea were disclosed in 28% of patients (almost all patients with subacute myocarditis). One patient with acute myocarditis suffered from recurrent paroxysmal convulsions, transient dextral hemiparesis, with magnetic resonance imaging results suggesting presence of inflammatory changes in the deep brain structures (in the hippocampus).

Resting electrocardiogram (ECG) performed on admission was normal only in 2 out of 32 patients. The most common findings included concave ST-segment elevation in most leads (22 patients, 69%) (Figure 1), negative T-waves in precordial leads (5 patients, 15%) (Figure 2), decreased amplitude of QRS complexes, ventricular and supraventricular arrhythmias as well as atrioventricular or intraventricular conduction disorders.

Table I. Clinical characteristics of patients with myocarditis

Parameter	All patients (n = 32)	Acute myocarditis (n = 21)	Subacute myocarditis (n = 11)
Age < 40 years	25 (78%)	21 (100%)	4 (36%)
Male sex	27 (84%)	18 (85%)	9 (81%)
Infection prior to admission	24 (75%)	21 (100%)	3 (27%)
History of prior infection (> 4 weeks)	10 (31%)	3 (14%)	7 (63%)
Symptoms of heart failure during hospitalisation	9 (28%)	2 (9%)	7 (63%)
hsCRP > 5 mg/l	24 (75%)	17 (80%)	7 (63%)
Increased TnT concentration/CK-MB activity	31 (97%)	21 (100%)	10 (90%)
LVEF < 50%	26 (81%)	15 (71%)	11 (100%)
Systolic function improvement, ↑ LVEF > 10% during hospitalisation (refers to patients with depressed LVEF)	19/26 (73%)	14/15 (93%)	5/11 (45%)
Epicardial-pericardial separation on echocardiography	28 (87%)	21 (100%)	7 (63%)
Ventricular tachycardia (nsVT, sVT)	7 (22%)	2 (9%)	5 (45%)
AV or intraventricular conduction disorder	6 (19%)	1 (5%)	6 (54%)
Supraventricular tachycardia, atrial fibrillation	9 (28%)	4 (19%)	5 (45%)
Significant ↑ of antibody titres against *Coxsackie B* viruses	8 (25%)	4 (19%)	4 (36%)

Abbreviations: hsCRP – high-sensitivity C-reactive protein, TnT – troponin T, CK-MB – heart type of creatine kinase, LVEF – left ventricular ejection fraction assessed with echocardiography, nsVT – non-sustained ventricular tachycardia, sVT – sustained ventricular tachycardia, AV – atrioventricular

Laboratory examinations showed increased ESR, leucocytosis, increased hs-CRP and TnT concentrations and elevated CK-MB activity. Coexisting elevation of all myocardial necrosis biomarkers – TnT, CPK, CK-MB – was found in 23 patients, while isolated increase of TnT concentration was observed in 8 (25%) patients. Patients with acute myocarditis had significantly higher troponin T concentration (above 1.0 ng/ml; reference range: 0.01-0.03 ng/ml) and CK-MB activity above 50 U/l (reference range: < 30 U/l), while patients with subacute myocarditis most often had troponin concentration between 1.0 and 0.03 ng/ml and CK-MB activity below 50 U/l.

Serological blood analysis of antibody titres against *Coxsackie B* viruses was performed in 18 patients. A positive result was found in 4 patients with acute and 4 patients with subacute myocarditis. Coexisting *Coxsackie B* and *Epstein-Barr* virus infection or *Coxsackie* and *Herpes* (HHV_6) infection was present in 2 patients. In two patients the aetiology of myocarditis was determined as non-infectious (due to collagenosis and reactive arthritis) and in one patient as bacterial.
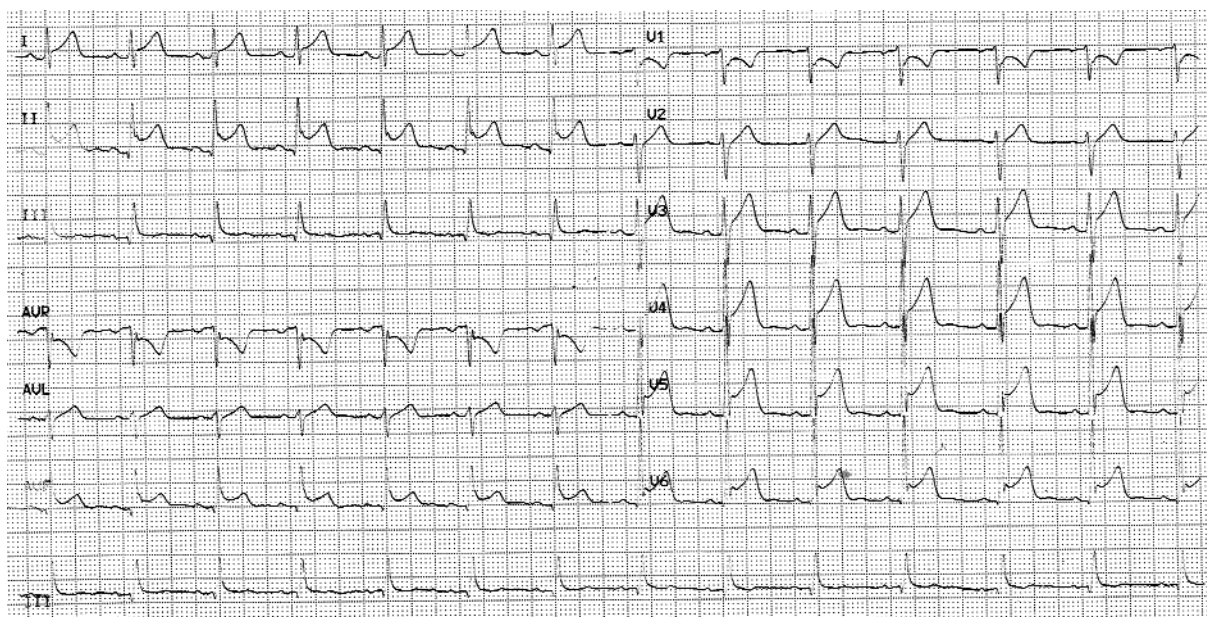

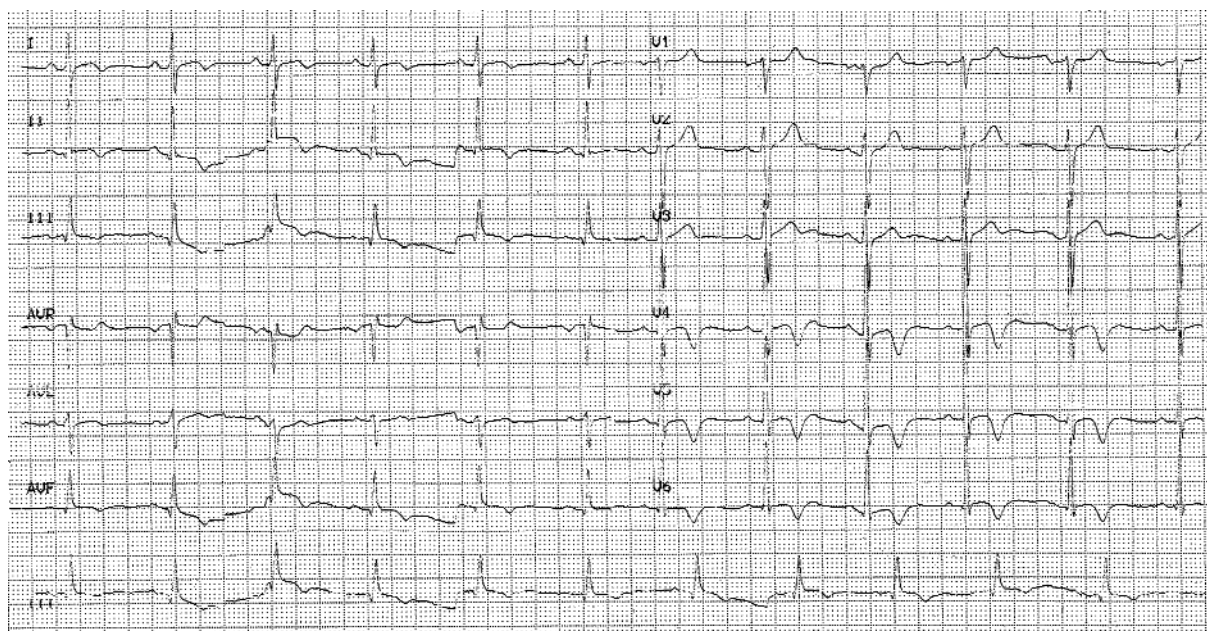**Figure 1.** Concave ST-segment elevation in leads I, II, avF, V_3-V_6

Figure 2. Mild ST-segment elevation with biphasic T-waves in leads I, II, III, avL, avF, V_3, negative T-waves in precordial leads V_4-V_6

Transthoracic echocardiography (TTE) performed on admission disclosed LV systolic dysfunction in 26 patients, with regional systolic dysfunction present in 9 of them. Epicardial-pericardial separation (2-15 mm) with increased echogenicity was found in 28 patients (Table I).

Analysis of 24-hour ambulatory Holter ECG monitoring showed presence of supraventricular tachyarrhythmias in 9 patients, severe ventricular arrhythmias (ventricular tachycardia, extrasystoles, salvos) in 7 patients and conductance disorders in 6 patients (Table I). Of note, arrhythmias and conduction disorders had a transient character and responded to treatment. They were more often present in patients with subacute myocarditis. Patients with subacute myocarditis were more likely to present with symptoms of heart failure; all of them had global systolic dysfunction on echocardiography and decreased LV ejection fraction (EF) (< 50%). Improvement of systolic function defined as increase of LVEF > 10% in comparison to baseline was observed only in 45% of patients with subacute myocarditis and in 93% with acute myocarditis. Patients with subacute myocarditis more often had hyperlipidaemia, were smokers, three of them suffered from hypertension and one from transient renal failure, secondary to heart failure.

Coronary angiography, demonstrating absence of significant stenosis in coronary arteries, was performed in 8 patients (4 patients with subacute and 4 patients with acute myocarditis) because of chest pain, elevated myocardial necrosis markers, dynamic ECG changes and regional systolic dysfunction on echocardiography.

Pharmacological treatment in most patients included angiotensin-converting enzyme inhibitors, aldosterone antagonists, loop diuretics, digoxin, and low molecular weight heparin in therapeutic doses in selected patients with significantly depressed LV systolic function and symptoms of heart failure. Patients with persistent tachycardia and preserved systolic function received beta-blockers. Two patients were treated with non-steroidal anti-inflammatory drugs. Administration of catecholamines was necessary in 5 patients.

Mean time of hospitalisation was 12 days with slower improvement in patients with subacute myocarditis, which accounted for around 7 days longer stay in comparison to those with acute myocarditis. Remission of the disease or significant improvement defined as regression of clinical symptoms, normalisation of inflammatory parameters and myocardial necrosis markers with regression of systolic dysfunction or improvement of LVEF was achieved in 25 patients (78%) on discharge (including 5 patients with subacute myocarditis with improvement of LV systolic function at long-term follow-up in the next 2 patients).

Follow-up echocardiography performed between 8 and 24 months from the onset of the disease in 27 patients demonstrated preserved LV systolic function in patients with acute myocarditis (mean LVEF = 61%) and depressed LVEF in those with subacute myocarditis (mean LVEF = 49%). At the time of the follow-up visit symptoms of heart failure (NYHA class II) were present in 6 patients with subacute myocarditis and in 2 patients with acute myocarditis. Those patients were receiving pension benefits (5 patients) or remained under the one-year rehabilitation pension (3 patients). Chronic pharmacological treatment in those patients includes angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, aldosterone antagonists, loop diuretics, beta-blockers, and digoxin. Other patients returned to work or continued education while remaining under close observation without the need of pharmacological treatment.

Discussion

In the presented group of patients myocarditis was mostly related to viral infection either directly before onset of the disease or in the near past. Varying onset of the disease as well as clinical course and outcomes allowed the distinction of two groups of patients – with acute and subacute myocarditis.

A classification of primary viral myocarditis into fulminant, acute, chronic active and chronic persistent was proposed in 1991. This classification is based on the time of symptoms onset, left ventricular systolic function on admission, results of endomyocardial biopsy performed on admission, and clinical and histological results of treatment [6, 7]. We would like to point out that our classification into acute and subacute myocarditis does not directly correspond to that histopathological-clinical classification because of the lack of endomyocardial biopsy, but is to some extent associated with it in respect of clinical symptoms and disease course. We are aware that classification of subacute myocarditis without evidence of a direct time relation with viral infection, less spectacular or no clinical improvement and existence of risk factors of coronary artery disease in those patients may be questionable. The diagnosis of subacute myocarditis (instead of for example dilated cardiomyopathy) in the studied group of patients was based on the presence of: parameters of active inflammatory process and/or markers of myocardial necrosis, pericardial effusion or epicardial-pericardial separation, elevation of antibody titres against *Coxsackie* in some patients, and observed improvement of left ventricular systolic function during hospitalisation and at long-term follow-up. In the majority of patients there was evidence of recent respiratory or gastrointestinal tract infection and other potential causes of myocardial damage were excluded (none of the patients had a history of alcohol abuse or familial cardiomyopathy, hypertension present in 3 patients was only mild and results of the coronary angiography were normal).

There was no suspicion of giant cell myocarditis in any of the patients during hospitalisation. Patients did not

present symptoms of rapidly progressive heart failure resistant to initiated conventional treatment. There were no severe, resistant to treatment forms of ventricular tachyarrhythmias or conduction disorders constituting an indication for endomyocardial biopsy [7, 8]. At present, due to similar treatment strategy in most of the patients and frequent controversies in interpretation of the results (false positive or false negative), the role of endomyocardial biopsy in the diagnosis of myocarditis is not crucial [9]. However, confirmation of viral aetiology by endomyocardial biopsy may be an argument for initiation of treatment with interferon, pleconaril, acyclovir or high doses of immunoglobulin.

The low rate of positive virological examinations in our group might have been caused by the lack of testing for parvovirus B_{19} (PVB$_{19}$), which according to reports is a common aetiological factor of myocarditis [3, 5].

Myocarditis may lead to dilated cardiomyopathy and heart failure [10, 11]. Chronic significant myocardial damage (LVEF < 50%) after 8-24 months of follow-up may be diagnosed in 5 patients from the presented group. Establishing prognosis is a very important element, especially in the young. It seems that patients with acute myocarditis have a good prognosis while remission may be less certain in those with subacute myocarditis.

We would also like to comment on the administration of low molecular weight heparin in patients with subacute myocarditis, myocardial damage and symptoms of heart failure. Adding this form of treatment to conventional therapy of heart failure was based on the work of other authors, especially Wojnicz et al. [16-18], who reported encouraging results of treatment in similar groups of patients with evidence of pleiotropic effects of enoxaparin: anti-inflammatory, immunomodulation, improvement of endothelial function and microcirculation (with preserved safety of enoxaparin administration). Prevention of thromboembolism was also considered as an aim of enoxaparin treatment.

Recent reports confirmed the utility of echocardiography in the differential diagnosis and assessment of prognosis in fulminant and acute myocarditis documented with endomyocardial biopsy. Felker et al. [12] assessed end-diastolic LV diameter, myocardial thickness, regional LV systolic dysfunction and ejection fraction by means of transthoracic echocardiography performed on admission and after 6 months. Patients with fulminant myocarditis had a better prognosis than those with acute myocarditis. In 2006 Mahrholdt et al. [3] showed that not only echocardiography but also magnetic resonance imaging (MRI) and virological examination are important tools in the assessment of prognosis in patients with viral myocarditis. Mahrholdt et al. [3] also found that LV end-diastolic volume in the acute phase, the presence of late gadolinium enhancement of the interventricular septum and the extent of LV mass injury are all independent risk factors of deterioration of systolic function and LV enlargement. In that report systolic LV dysfunction was associated with coexisting PVB$_{19}$ and HHV$_6$ infection [3]. The PVB$_{19}$ and HHV$_6$ were the most common pathogens of viral myocarditis in that study. PVB$_{19}$ infection is characterised by a clinical course similar to myocardial infarction as the virus damages the microvascular endothelial cells which in consequence leads to ischaemic myocardial injury. Myocardial injury is only regional and LVEF is not significantly depressed [2]. That would explain the regional systolic dysfunction on echocardiography in our patients. Recently a similar course of myopericarditis was presented by Kukla et al. [15]. This type of clinical manifestation is a cause of more or less justified indication for coronary angiography. Another type of myocardial injury with subacute onset of symptoms of heart failure and more pronounced general state is caused by HHV$_6$ or coexisting PVB$_{19}$ and HHV$_6$ infection and less often leads to complete regression of symptoms. Clinical characteristics of PVB$_{19}$ infection may correspond to acute myocarditis in our group, while HHV$_6$ or coexisting PVB$_{19}$ and HHV$_6$ infection may indicate subacute myocarditis. Dedicated virological examinations could confirm those assumptions.

Conclusions

Diagnosis of myocarditis is still challenging. Careful history taking, serial laboratory, ECG and echocardiographic examinations are helpful in therapeutic decisions making and assessing prognosis. Patient with subacute myocarditis are more symptomatic than patients with acute myocarditis.

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Zapalenie mięśnia sercowego – przebieg kliniczny i rokowanie w grupie 32 chorych hospitalizowanych z rozpoznaniem zapalenia mięśnia sercowego, obserwacja 2-letnia

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Streszczenie

Wstęp: Zapalenie mięśnia sercowego (ZMS) jest procesem o etiologii infekcyjnej bądź nieinfekcyjnej obejmującym komórki mięśnia serca, tkankę śródmiąższową i naczynia. Choroba ma różnorodną manifestację kliniczną i różne rokowanie. Nie ma zdefiniowanych kryteriów diagnostycznych ZMS.

Cel: Charakterystyka chorych hospitalizowanych w latach 2006–2008 z rozpoznaniem ZMS oraz ocena przebiegu klinicznego choroby.

Metody: Zapalenie mięśnia sercowego rozpoznano na podstawie charakterystycznego wywiadu i objawów klinicznych oraz wyników badań laboratoryjnych u 32 chorych.

Wyniki: Średni wiek chorych wynosił 33 lata, a 84% badanej populacji stanowili mężczyźni. U wszystkich chorych wykonano badanie echokardiograficzne oraz badanie EKG holterowskie, a w 8 przypadkach także koronarografię. Ze względu na odmienny przebieg kliniczny chorych podzielono na dwie grupy: „ostrego" i „podostrego" ZMS, odpowiednio 21 (66%) chorych i 11 (34%) chorych. Chorzy w grupie „ostrego" ZMS uskarżali się najczęściej na bóle w klatce piersiowej, osłabienie, męczliwość, w tej grupie infekcja wirusowa poprzedzała hospitalizację. W grupie „podostrego" ZMS przeważały objawy niewydolności serca w postaci duszności, retencji płynów, pogorszenia wydolności fizycznej. Zmiany w EKG obserwowano niemal u wszystkich chorych w obu grupach, zaburzenia rytmu i przewodzenia występowały znamiennie częściej w grupie „podostrego" ZMS. W badaniach laboratoryjnych obserwowano podwyższone parametry procesu zapalnego oraz wzrost stężenia markerów martwicy kardiomiocytów: TnT, wzrost aktywności CPK, CK-MB. Badania krwi w kierunku miana przeciwciał przeciwko wirusom kardiotropowym wykonano u 18 (56%) osób. Wynik był dodatni w 8 przypadkach. U 26 (81%) chorych wystąpiło istotne zmniejszenie frakcji wyrzutowej lewej komory (LVEF) < 50%. Poprawę kurczliwości, zdefiniowaną jako zwiększenie LVEF > 10% w stosunku do wartości wyjściowej, obserwowano jedynie u 45% chorych z grupy „podostrego" ZMS i aż u 93% chorych z grupy „ostrego" ZMS. Średni czas hospitalizacji wyniósł 12 dni.

Wnioski: Badanie przedmiotowe, EKG, echokardiograficzne, metodą rezonansu magnetycznego z oceną parametrów procesu zapalnego, markerów martwicy mięśnia serca i rozszerzoną diagnostyką serologiczną pozwala na ustalenie rozpoznania ZMS, ocenę kliniczną oraz optymalizację leczenia.

Słowa kluczowe: zapalenie mięśnia serca, przebieg kliniczny, rokowanie

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