Journal of the American College of Cardiology
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Published by Elsevier Science Inc.

Vol. 36, No. 1, 2000
ISSN 0735-1097/00/$20.00
PII S0735-1097(00)00690-2

Echocardiographic Findings in Fulminant and Acute Myocarditis

G. Michael Felker, MD,* John P. Boehmer, MD, FACC,*‡ Ralph H. Hruban, MD,† Grover M. Hutchins, MD,† Edward K. Kasper, MD, FACC,* Kenneth L. Baughman, MD, FACC,* Joshua M. Hare, MD, FACC*

Baltimore, Maryland

OBJECTIVES We sought to use echocardiography to assess the presentation and potential for recovery of left ventricular (LV) function of patients with fulminant myocarditis compared with those with acute myocarditis.

BACKGROUND The clinical course of patients with myocarditis remains poorly defined. We have previously proposed a classification that provides prognostic information in myocarditis patients. Fulminant myocarditis causes a distinct onset of illness and severe hemodynamic compromise, whereas acute myocarditis has an indistinct presentation, less severe hemodynamic compromise and a greater likelihood of progression to dilated cardiomyopathy.

METHODS Echocardiography was performed at presentation and at six months to test the hypothesis that fulminant ($n = 11$) or acute ($n = 43$) myocarditis could be distinguished morphologically.

RESULTS Patients with both fulminant (fractional shortening 19 ± 4%) and acute myocarditis (17 ± 7%) had LV systolic dysfunction. Patients with fulminant myocarditis had near normal LV diastolic dimensions (5.3 ± 0.9 cm) but increased septal thickness (1.2 ± 0.2 cm) at presentation, while those with acute myocarditis had increased diastolic dimensions (6.1 ± 0.8 cm, $p < 0.01$ vs. fulminant) but normal septal thickness (1.0 ± 0.1 cm, $p = 0.01$ vs. fulminant). At six months, patients with fulminant myocarditis had dramatic improvement in fractional shortening (30 ± 8%) compared with no improvement in patients with acute myocarditis (19 ± 7%, $p < 0.01$ for interaction between time and type of myocarditis).

CONCLUSIONS Fulminant myocarditis is distinguishable from acute myocarditis by echocardiography. Patients with fulminant myocarditis exhibit a substantial improvement in ventricular function at six months compared with those with acute myocarditis. Echocardiography has value in classifying patients with myocarditis and may provide prognostic information. (J Am Coll Cardiol 2000;36:227–32) © 2000 by the American College of Cardiology

Myocarditis is an inflammatory disease of the myocardium of uncertain etiology and may be a precursor of dilated cardiomyopathy (DCM) (1,2). Although standardized histologic criteria, the Dallas Criteria, have been widely applied both clinically and in therapeutic trials (3), the optimal prognostic classification system remains controversial (4). Because both complete recovery of left ventricular (LV) function and progression to DCM have been described, the ability to assess prognosis at the time of presentation is clearly of great benefit in patients with myocarditis (1,5,6).

We have previously proposed a clinicopathologic classification utilizing both histologic and clinical features that may provide prognostic information in patients with myocarditis (7). Fulminant myocarditis is characterized by a distinct viral prodrome, the sudden onset of severe hemodynamic compromise and marked myocardial inflammation. In contrast, patients with acute myocarditis have an indistinct onset of symptoms, less severe hemodynamic embarrassment and a more variable degree of myocardial inflammation. Recently, we have shown that patients with fulminant myocarditis have improved long-term survival compared with those with the acute form of the condition (8). The purpose of this study was to use echocardiography to assess the presentation of patients with fulminant myocarditis as compared with those with acute myocarditis. A secondary aim of this study was to compare the potential for recovery of LV function in patients with fulminant and acute myocarditis.

METHODS

Study population. Seven hundred fifty patients underwent endomyocardial biopsy at our institution over a seven-year period as part of a comprehensive evaluation of unexplained cardiomyopathy. Seventy-two of 750 patients (9.6%) were diagnosed as having myocarditis or borderline myocarditis by the Dallas Criteria (3). This group did not include patients who had evidence of myocarditis in the setting of a systemic process such as human immunodeficiency virus infection or sarcoidosis. Patients were further classified as having acute, fulminant, chronic active or chronic persistent myocarditis, using our previously reported classification system (7). Using these criteria, 43 patients were classified as

From the *Division of Cardiology and †Department of Pathology, Johns Hopkins Hospital, Baltimore, Maryland; presently at the ‡Division of Cardiology, Hershey Medical Center, Hershey, Pennsylvania.

Manuscript received June 24, 1999; revised manuscript received January 21, 2000, accepted March 6, 2000.

JACC Vol. 36, No. 1, 2000
July 2000:227–32

Abbreviations and Acronyms

DCM = dilated cardiomyopathy
LV = left ventricle or ventricular
LVEDD = left ventricular diastolic dimension
%FS = percentage fractional shortening

having acute myocarditis and 11 as having fulminant myocarditis. These 54 patients were the subjects of this study.

Echocardiography. Standard two-dimensional echocardiography was performed at the time of presentation and after six months. Baseline echocardiograms for each patient were evaluated. Follow-up echocardiograms at six months for seven patients with fulminant myocarditis and 25 patients with acute myocarditis were evaluated. Of the patients with no follow-up echocardiograms available, two patients with fulminant (18%) and four patients with acute myocarditis (9%) died. Two patients with fulminant (18%) and 14 with acute myocarditis (32%) were lost to follow-up. For each echocardiogram, left ventricular diastolic dimension (LVEDD) and maximal septal thickness were measured by M-mode in the parasternal long axis view using the leading-edge to leading-edge method. Percentage fractional shortening (%FS) was calculated in a standardized manner (9).

Endomyocardial biopsy. All patients underwent endomyocardial biopsy using a right internal jugular vein approach and a Stanford-Caves or disposable bioptome. At least four specimens were taken from the right ventricular septum and immediately placed in either 10% formalin for light microscopy or 3% glutaraldehyde for electron microscopy. Each formalin-fixed specimen was stained with hematoxylin-eosin and examined at at least four levels by an experienced cardiac pathologist (R.H.H., G.M.H.). The presence of myocarditis or borderline myocarditis was determined according to the Dallas Criteria (3) and graded as mild, moderate, or severe blinded to patient classification. The diagnosis of myocarditis was confirmed by immunohistochemical staining for T-lymphocytes.

Hemodynamics. After endomyocardial biopsy, all patients underwent right heart catheterization using a balloon-tipped, flow-directed thermodilution catheter. Measurements of cardiac output were obtained in triplicate with <10% variability between measurements. Cardiac index, LV stroke work index, systemic vascular resistance index and pulmonary vascular resistance index were calculated using standard formulas.

Statistical analysis. All values are reported as mean ± standard deviation. Measurements between groups at baseline were compared using the Student *t* test. Two-way repeated measures analysis of variance was used for assessing the interaction between type of myocarditis and changes at baseline and six months, with time as the within patient variable and type of myocarditis as the between patient variable (Stata 6.0, College Station, Texas). A two-sided p value < 0.05 was considered statistically significant.

Table 1. Baseline Clinical Characteristics of Patients With Fulminant and Acute Myocarditis

	Fulminant (n = 11)	Acute (n = 43)
Flu-like illness within 4 weeks	100% (11)	21% (9)
Fever within 12 weeks	91% (10)	23% (10)
Acute onset of symptoms	100% (11)	56% (24)
NYHA Functional Class		
4	73% (8)	58% (25)
3	27% (3)	26% (11)
2	0% (0)	16% (7)
1	0% (0)	0% (0)

NYHA = New York Heart Association.

RESULTS

Patient characteristics. The initial clinical characteristics of the patients with fulminant and acute myocarditis are shown in Table 1. By definition, the baseline characteristics differed between the two groups. All patients with fulminant myocarditis reported a flu-like prodrome or fever in the four weeks proceeding the onset of cardiac symptoms, compared with 21% in the acute myocarditis group. Patients with fulminant myocarditis generally had the abrupt onset of symptoms within two weeks of presentation, whereas symptom onset in those with acute myocarditis was indistinct (dated to a period of weeks to months). All patients classified as fulminant myocarditis had New York Heart Association class III or IV heart failure symptoms, while functional limitation in those with acute myocarditis was more variable.

Echocardiography. Echocardiographic measurements at baseline and at six months are shown in Figures 1 and 2. Both groups had markedly decreased LV systolic function at baseline that was of similar magnitude (%FS = 17 ± 7% for acute vs. 19 ± 4% for fulminant, p = NS, normal 28% to 41%). Patients with fulminant myocarditis had significantly less ventricular dilation at baseline (LVEDD = 5.3 ± 0.9 cm vs. 6.1 ± 0.8 cm, $p < 0.01$, normal < 5.5 cm) but greater septal thickness (1.2 ± 0.2 cm vs. 1.0 ± 0.1 cm, p = 0.01) than patients with acute myocarditis.

At six months, the patients with fulminant myocarditis showed a marked improvement in the %FS (30 ± 8%, $p < 0.01$ vs. baseline), resulting in normal LV systolic function. In contrast, patients with acute myocarditis showed no significant improvement in fractional shortening at six months compared with baseline (%FS = 19 ± 7%, p = NS vs. baseline). The interaction between type of myocarditis and time for fractional shortening was highly significant ($p < 0.01$). Complete normalization of ventricular function was significantly more likely in patients with fulminant myocarditis (57%) than in those with acute myocarditis (20%, p = 0.05).

There was a trend towards greater resolution of septal thickening in the fulminant group (from 1.2 ± 0.2 cm at baseline to 1.0 ± 0.1 cm at six months) than the acute group (1.0 ± 0.1 cm at both baseline and six months), but this did

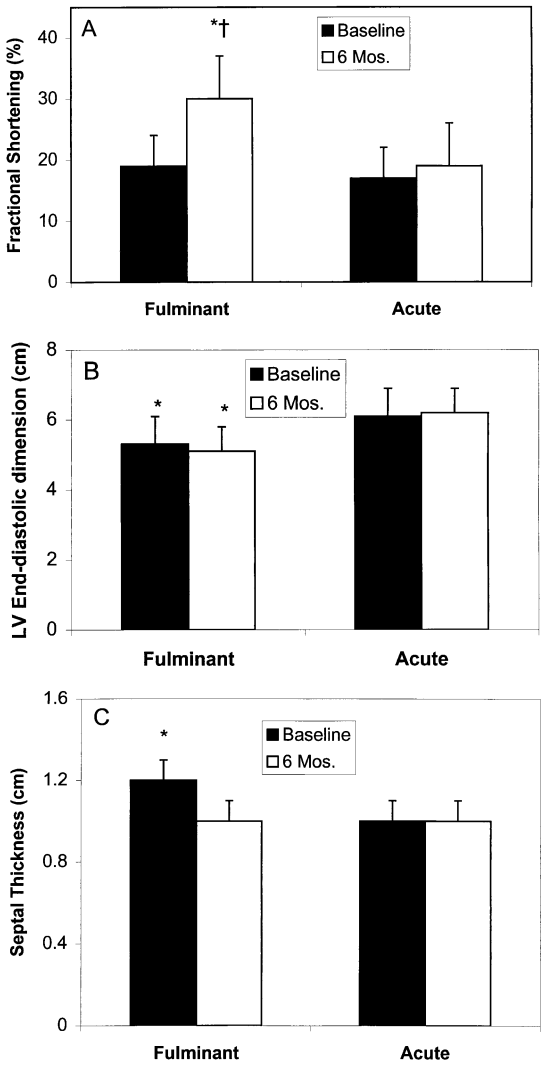

Figure 1. Echocardiographic findings in patients with fulminant and acute myocarditis at baseline and six months. **(A)** Fractional shortening, **(B)** left ventricular end-diastolic dimension, **(C)** septal thickness. *$p < 0.01$ vs. acute; †$p < 0.01$ for interaction between time and type of myocarditis.

not reach statistical significance ($p = 0.095$ for the type of myocarditis/time interaction). Left ventricular diastolic dimension at six months remained significantly smaller in the fulminant group than it did in the acute group (5.1 ± 0.6 cm vs. 6.2 ± 0.9 cm, $p < 0.01$). Left ventricular diastolic dimension did not significantly changed in either group between baseline and six months ($p = 0.63$ for type of myocarditis/time interaction).

Histology. The histologic characteristics of endomyocardial biopsy specimens for study patients are shown in Table 2. All patients in the study had either histologic myocarditis (81%) or borderline myocarditis (19%) on endomyocardial biopsy as criteria for study entry. All patients in the fulminant group had histologic myocarditis by the Dallas Criteria. In the acute group, myocarditis was diagnosed in 77% of patients, with the remainder having borderline myocarditis. By definition, the degree of inflammation seen on endomyocardial biopsy specimens was substantially greater in patients with fulminant myocarditis than those with acute myocarditis (Fig. 3).

Hemodynamics. Hemodynamic measurements at the time of endomyocardial biopsy for both groups are shown in Table 2. Patients with fulminant myocarditis had more severe hemodynamic compromise at presentation than those with acute myocarditis. Mean arterial pressure and systemic vascular resistance were both significantly decreased in the fulminant group when compared with those with acute myocarditis ($p < 0.01$ for each), despite substantially higher use of intravenous inotropic agents in patients with fulminant myocarditis (64% vs. 5%). Heart rate, right atrial pressure, pulmonary artery mean pressure and pulmonary capillary wedge pressure were all significantly higher in the fulminant myocarditis group than they were in the acute group ($p < 0.03$ for each).

DISCUSSION

This study reports the echocardiographic findings of patients with two different clinical syndromes of myocarditis, fulminant and acute. Patients with these two subtypes could be distinguished by echocardiography. Patients with fulminant myocarditis presented with nondilated, thickened and hypocontractile LVs, whereas those with acute myocarditis had marked LV dilation, normal LV thickness and decreased LV function. At six-month follow-up, significant improvement in %FS had occurred in the fulminant group, whereas those with acute myocarditis showed no improvement. Echocardiography may aid in classifying patients with myocarditis into clinically relevant subgroups with prognostic implications.

Prior studies of echocardiography in myocarditis. Previous studies of echocardiography in myocarditis have demonstrated a variety of echocardiographic findings. In addi-

Table 2. Hemodynamics and Histologic Characteristics of Patients With Fulminant or Acute Myocarditis

	Fulminant (n = 11)	Acute (n = 43)	p Value
Histology			
Myocarditis	100% (11)	77% (33)	NS
Borderline myocarditis	0% (0)	23% (10)	
Inflammation			
Severe	55% (6)	5% (2)	< 0.01
Moderate	45% (5)	14% (6)	
Mild	0% (0)	81% (35)	
Hemodynamics			
Right atrial pressure	11 ± 8	4 ± 3	< 0.01
Mean pulmonary artery pressure	28 ± 11	21 ± 9	0.03
Pulmonary artery wedge pressure	21 ± 11	14 ± 9	0.03
Mean blood pressure	79 ± 11	90 ± 12	< 0.01
Heart rate (beats/min)	109 ± 21	91 ± 21	< 0.01
Cardiac index (L/min/m^2)	2.8 ± 0.9	2.5 ± 0.6	NS
SVRI (dynes·sec·cm^{-5}·m^2)	2,072 ± 440	2,939 ± 752	< 0.01
PVRI (dynes·sec·cm^{-5}·m^2)	244 ± 242	218 ± 150	NS
LVSWI (g-m/m^2)	21 ± 11	31 ± 14	NS

All pressures in mm Hg; all values given as mean ± standard deviation.
NS = not significant; SVRI = systemic vascular resistance index; PVRI = pulmonary vascular resistance index; LVSWI = left ventricular stroke work index.

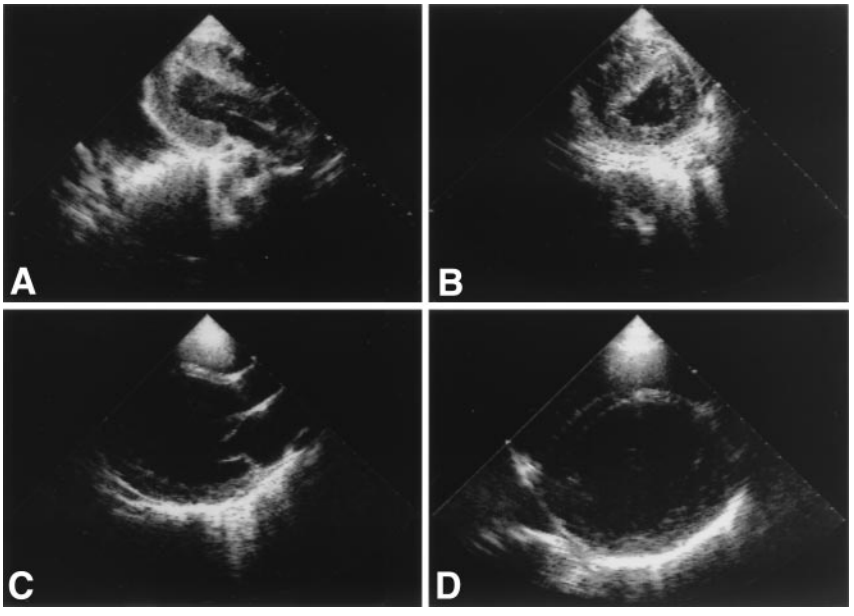

Figure 2. Two-dimensional echocardiograms from patients with fulminant and acute myocarditis at presentation. The **top panels** show the parasternal long axis **(A)** and short axis **(B)** views of a 20-year-old man with fulminant myocarditis who presented after five days of a viral syndrome followed by acute hemodynamic collapse. Note the severe ventricular thickening (septal thickness 2.1 cm) but small ventricular cavity size (LVEDD 2.5 cm). After hemodynamic support with intravenous inotropic agents and a left ventricular assist device, this patient recovered near normal left ventricular function. The **bottom panels** show parasternal long axis **(C)** and short axis **(D)** views from a 19-year-old man with acute myocarditis who presented with three weeks of fatigue, fevers and the gradual onset of dyspnea on exertion. Note the decreased ventricular thickness (septal thickness = 0.6 cm) and marked dilated left ventricular cavity (LVEDD = 6.9 cm). At six month follow-up, LVEDD had increased to 8.0 cm, and the patient was awaiting cardiac transplantation. LVEDD = left ventricular diastolic dimension.

tion to systolic dysfunction (10,11), regional wall motion abnormalities (10), diastolic dysfunction (12) and changes in echocardiographic image texture (13) have been reported. These studies have not attempted to distinguish clinical syndromes within the spectrum of patients with myocarditis. Additionally, some reports have utilized clinical rather than histologic criteria in making the diagnosis of myocarditis, potentially confounding their results by including patients who may not have had histologic evidence of myocarditis. We have shown the utility of echocardiography in classifying patients with histologically proven myocarditis based on LV morphology at presentation.

Data from several previous studies are consistent with our findings. Pinamonti and colleagues (10) described echocardiographic findings in 41 patients with histologically proven myocarditis and found LV dysfunction in 69%, primarily with little to no dilation of the LV cavity (10). They also noted four patients in their series with transient "LV hypertrophy" in the setting of myocarditis. In two of these patients, ventricular thickening was associated with dramatic improvement in LV function. Transient "LV hypertrophy" associated with myocarditis has also been reported by Hauser and coworkers (14) in a 28-year-old woman with sudden onset of severe heart failure (14). Spontaneous resolution of LV thickening in this patient was associated with normalization of systolic function and clinical recovery. It is likely that these cases represent examples of the fulminant type of myocarditis as described in this report. Although the degree of LV dilation is often related to the severity and duration of heart failure, our data demonstrate that severe systolic dysfunction may occur in the absence of significant ventricular dilation in patients with fulminant myocarditis. Left ventricular dysfunction without significant cavity dilation has also been reported as a variant of idiopathic DCM in patients without myocarditis, and both progression to DCM and normalization of systolic function have been reported (15).

Potential mechanisms. The increased LV thickness in fulminant myocarditis is likely due to the greater inflammatory response seen on endomyocardial biopsy in these patients. Interstitial edema may contribute to both thickened ventricular walls and decreased ventricular contractility in this disorder. The contrast between LV morphology seen on echocardiography in fulminant and acute myocarditis is demonstrated in Figure 2, showing dramatically differing echocardiographic findings between fulminant and acute myocarditis. The explanation for the differences in the presentation and natural history of these disorders is unknown. These differences may be influenced either by the virulence or inoculum of the causative viral agent or by host factors such as genetic predisposition (16–18). It is possible that a more vigorous immune response in patients with fulminant myocarditis is responsible for both the more severe initial presentation and the improved outcome at six months.

Outcomes in myocarditis. Widely variable outcomes in patients with myocarditis have been described. A long-term follow-up study by Quigley and colleagues (6) confirmed

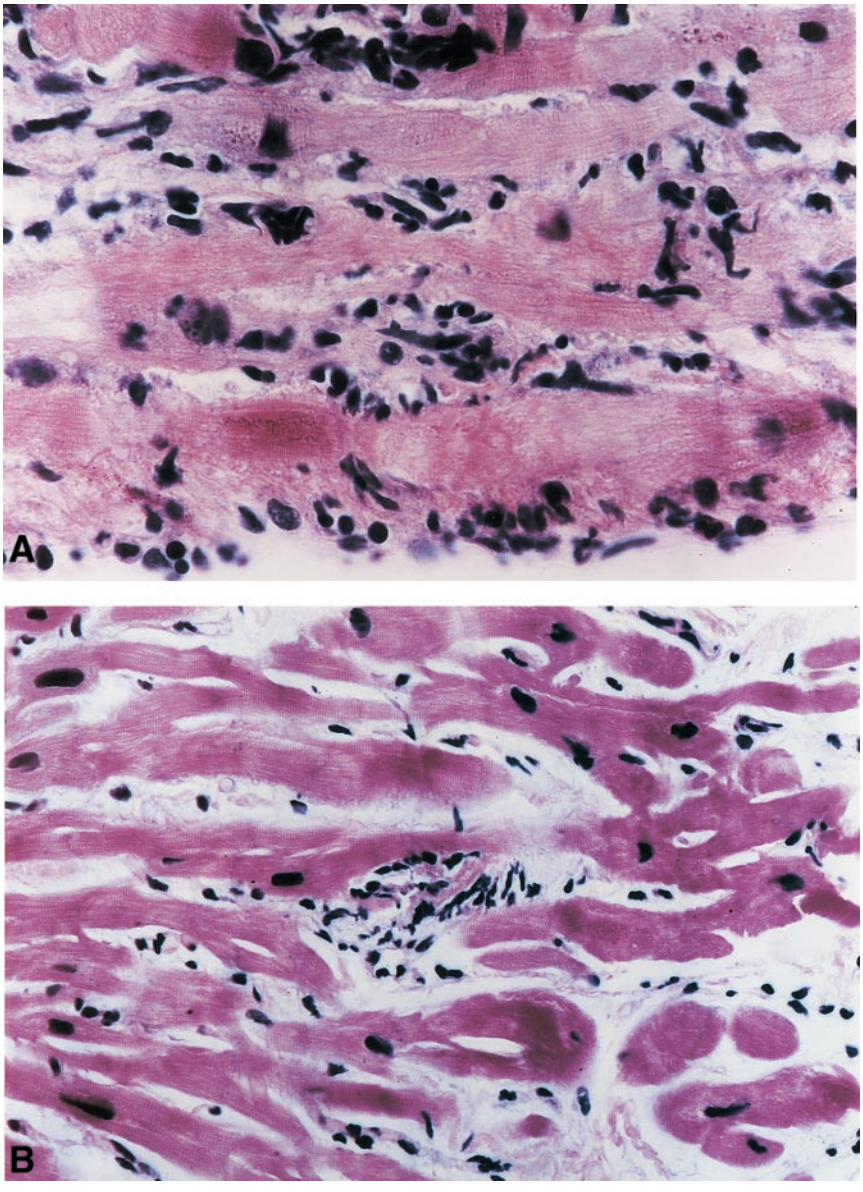

Figure 3. Endomyocardial biopsy specimens from patients with fulminant **(A)** and acute **(B)** myocarditis. Fulminant myocarditis was characterized by more extensive and diffuse lympocytic infiltration and myocyte necrosis than acute myocarditis (hematoxylin-eosin stain, original magnification × 400).

the variable prognosis in patients with this disorder, with some patients regaining normal ventricular function and others progressing to DCM. This study did not find a correlation between the degree of LV dysfunction at presentation and subsequent echocardiographic or clinical outcome. Similar findings were reported by Dec and coworkers (1), who described 18 patients with acute onset of cardiomyopathy and histologic myocarditis, of whom some made complete recovery while others progressed to DCM. Notably, the two patients in this study with endomyocardial biopsies demonstrating a "diffuse, dense inflammatory infiltrate" on histologic examination both recovered LV function, whereas patients with a focal pattern of myocarditis had a prognosis similar to patients with idiopathic DCM. These data are in agreement with our findings, in which patients with a more severe inflammatory infiltrate (i.e., those with fulminant myocarditis) had a much greater propensity for recovery of normal ventricular function. Echocardiographic criteria that predict outcome in patients with myocarditis have not been well-established. Mendes and colleagues (19) reported 23 patients with biopsy proven myocarditis in whom the presence of right ventricular dysfunction by echocardiography predicted worse clinical outcome. We have examined the differences in echocardiographic features between patients with differing clinical syndromes and myocarditis and found substantial morphologic differences at presentation that may predict subsequent recovery of ventricular function.

Study limitations. This study has several potential limitations. The number of patients was relatively small, particularly in the group with fulminant myocarditis. Echocardiographic follow-up was incomplete in both the acute and

fulminant myocarditis groups, reducing the power of our conclusions regarding the changes in morphology between baseline and six months. Still, this study represents the largest systematic study of echocardiographic findings in patients with biopsy proven myocarditis. Our study was retrospective, but the classification into categories of myocarditis was done prospectively. All diagnoses of myocarditis were based on accepted histologic criteria. Although inflammatory cell infiltrates have been demonstrated in some cases of idiopathic DCM (20), 81% of our cases showed frank myocarditis, and we used immunohistochemical staining for T-lymphocytes to confirm the diagnosis in all cases of borderline myocarditis.

The use of immunosuppression was not controlled in this study. Two of 11 patients with fulminant myocarditis and 30 of 43 patients with acute myocarditis received some type of immunosuppressive therapy, generally corticosteroids. While it is possible that the greater use of immunosuppressive therapy in the patients with acute myocarditis may have led to worse echocardiographic outcomes in this group, this does not seem to be a plausible explanation for our findings. Our previous experience has suggested that patients with fulminant myocarditis, despite their severe clinical presentation, improve without immunosuppression. The utility of immunosuppression in acute myocarditis is unclear, but a consistent benefit was not seen in the Myocarditis Treatment Trial (21). Given the observational methods used in this study, no conclusions can be drawn regarding the utility of immunosuppression in either group.

Treatment implications. Our data suggest that the presence of septal thickening and normal LV dimensions in a patient with a fulminant presentation and severe hemodynamic compromise portends a high likelihood of ventricular recovery. Maximal medical support, including intravenous inotropic agents and placement of a temporary LV assist device, should be considered for these patients in order to provide time for potential recovery of LV function, as has been described in case reports (22,23). Given the high likelihood of ventricular recovery, early consideration of cardiac transplantation may be avoided in patients with fulminant myocarditis.

Conclusions. This study suggests that patients with fulminant myocarditis have an echocardiographic presentation that is distinct from that of patients with acute myocarditis. Additionally, patients with fulminant myocarditis are significantly more likely to recover LV function than are those with acute myocarditis. Echocardiography provides important prognostic information and may aid in the clinically relevant classification of patients with histologically proven myocarditis.

Reprint requests and correspondence: Dr. Joshua M. Hare, Division of Cardiology, the Johns Hopkins Hospital, Carnegie 568, 600 North Wolfe Street, Baltimore, Maryland 21287. E-mail: jhare@mail.jhmi.edu.

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