Serial cardiac function tests in myocarditis

Candace L. Miklozek, Elizabeth M. Kingsley, Clyde S. Crumpaker, John F. Modlin, Henry D. Royal, Patricia C. Come, Roger Mark and Walter H. Abelmann

Charles A. Dana Research Institute and the Harvard-Thorndike Laboratory of Beth Israel Hospital, and the Department of Medicine (Cardiovascular Division), Beth Israel Hospital and Harvard Medical School, Boston, Massachusetts, Department of Medicine, The Memorial Hospital and Brown University, Pawtucket, Rhode Island, USA

Summary: Sixteen patients (mean age 27 years, range 16 to 39 years) with the diagnosis of myopericarditis established by strict clinical criteria were evaluated following recovery 0.7 to 4.0 years (mean 2.7 years) later. Evidence of an acute viral infection was present in 44%. During the acute illness, the major clinical manifestations consisted of pericarditis in 10 patients, acute myocardial infarction in 5, right ventricular dysfunction in 5, bundle branch or hemiblock in 4, ventricular arrhythmias in 3, congestive heart failure in 3 and cardiogenic shock and inappropriate sinus tachycardia in one patient each. Holter monitoring, echophonocardiography and radionuclide ventriculography results were abnormal in 57, 67 and 64% of patients respectively. At follow-up, these tests were abnormal in 67, 7 and 73%. Focal wall motion abnormalities were present in five. Seventy-five percent of patients had one or more abnormal tests at last follow-up. Although 8 patients had improved by a scoring system, 5 patients had an increase in the number of abnormal tests, including one who died. These findings indicate that persistent abnormalities following recovery from myopericarditis are not rare and support the hypothesis that the syndrome of dilated cardiomyopathy may be a sequel of myopericarditis.

Introduction

The syndrome of dilated cardiomyopathy has been suggested by many to be a late complication of acute viral myocarditis (Smith, 1970; Sainani et al., 1975; Bengtsson & Lamberger, 1966). Advances in the non-invasive technology used to evaluate cardiac function have made study of this postulate feasible.

In 1979, we undertook a 5 year clinical prospective study based on this hypothesis. Our goal was to determine if cardiac function deteriorated over time following clinical recovery from myopericarditis. We identified 16 patients using strict criteria for the diagnosis of myopericarditis and studied them at periodic follow-up intervals with a wide array of non-invasive tests of cardiac function. We have already presented earlier follow-up results from these patients (Miklozek et al., 1984). We now present the latest results.

Materials and methods

Criteria for the diagnosis of myopericarditis

Myopericarditis was diagnosed if the patient de-

Correspondence: C.L. Miklozek, M.D., The Memorial Hospital, Pawtucket, RI 02860, USA.

© The Fellowship of Postgraduate Medicine, 1986
described by Tavel (1979); a PEP/LVET ratio greater
than 0.420 was considered abnormal. Radionuclide
ventriculograms were performed in most patients by
intravenous injection of pertechnetate 99m labelled
red blood cells; left ventricular ejection fraction was
calculated by the count method of Parker et al. (1977).

Viral studies

Urine, stool, and throat specimens from each patient
were inoculated into continuous cell line cultures
maintained in the Virology Laboratory at Beth Israel
Hospital. Sera were collected during the acute illness
and at least 2 weeks later for determination of
microneutralization antibodies. In some patients, sera
were sent to the State Board of Health and to Dr John
V. Sullivan at the University of Massachusetts for
measurement of antibodies to cytomegalovirus and
Epstein-Barr virus, respectively.

Follow-up studies

Patients were seen by either CLM or WHA or the
primary care physician at various intervals in time
following clinical recovery. Whenever possible, elec-
trocardiograms, M-mode echophonocardiograms, radionuclide ventriculograms, 24 hour ambulatory
Holter monitoring and symptom limited exercise tests
were performed. Exercise tests were performed in
conjunction with radionuclide ventriculography using
a bicycle ergometer in most patients. Endpoints for the
exercise test consisted of (1) onset of cardiac symp-
toms, (2) significant depression of the S-T segments
and/or inversion of the T waves, (3) dysrhythmias, (4)
attainment of 85% of the age predicted maximal heart
rate, or (5) hypotension.

Results

Sixteen patients with the clinical diagnosis of acute
myopericarditis and with available follow-up studies
were enrolled from 1979 to 1981. The duration of
follow-up ranged from 0.7 to 4.0 years with a mean of
2.7 years. Twelve were males. The mean age was 27
years and ranged from 16 to 39 years. All patients
survived the acute illness. One patient died 8.3 months
later. There were no other deaths.

Acute illness

Seven patients (44%) had evidence of an acute viral
infection by culture or antibody titre. Echovirus 11
was isolated from 5 patients. Another, although
culture negative, had stable but elevated titres (1:40)
to echovirus 11. In one of these patients, Coxsack-
ievirus B-3 was also isolated during a subsequent
episode of myopericarditis. A seventh patient had
both a fourfold rise in titre to cytomegalovirus and
reactivation of an Epstein-Barr virus infection.

At the time of diagnosis, the following major
cardiac abnormalities were present with overlap in the
individual patient of myocardial and pericardial man-
ifestations. Ten patients had pericarditis based on
either history, electrocardiogram, presence of a
pericardial rub, or effusion by echocardiography. Five
presented with evidence of an acute myocardial infar-
cion. Five had clinical right ventricular dysfunction
with elevated venous pressure and a right ventricular
heave. A bundle branch or hemiblock was present in
four patients, while ventricular arrhythmias occurred
in three patients. One patient had an inappropriate
sinus tachycardia. Congestive heart failure developed
in three patients, one of whom had cardiogenic shock.

Abnormalities were commonly detected by all
methods used. Cardiac symptoms were elicited from
88% (14/16), and the examination was abnormal in
94% (15/16). Electrocardiograms, Holter monitoring,
echophonocardiograms, and radionuclide ventri-
culograms were abnormal in 88% (14/16), 57% (8/
14), 67% (10/15) and 64% (9/14), respectively. A
broader definition of abnormalities by Holter monitor
was used in this report and explains the larger number
of abnormals when compared to our initial report
(Miklozek et al., 1984). Focal wall motion abnor-
malities by radionuclide ventriculograms were present
in 3 patients.

Follow-up

At follow-up, 44% (7/16) still had cardiac symptoms,
while the physical examination was abnormal in 63%
(10/16). Electrocardiograms, Holter monitoring,
echophonocardiograms, radionuclide ventriculo-
grams, and exercise tests were abnormal in 60% (9/15),
67% (6/9), 7% (1/14), 73% (11/15), and 8% (1/13),
respectively. Regional wall motion abnormalities were
present in five by radionuclide ventriculography. The
ejection fraction by echocardiography had increased
from 58 ± 2.7% (s.e.m.) to 69 ± 1.8% (s.e.m.)
(P < 0.005), while mean Vcf increased from 1.18 ± 0.9
circ/s (s.e.m.) to 1.31 ± 0.5 circ/s (s.e.m.) (not sig-
nificant) in 13 of the group. The PEP/LVET ratio
calculated from the phonocardiogram decreased from
0.400 ± 0.2 to 0.310 ± 0.05 (s.e.m.) (not significant)
in 12 of the group. Ejection fraction by radionuclide
ventriculography decreased from 62 ± 3.8% (s.e.m.)
to 55 ± 3.9% (s.e.m.) (not significant) in 13 of the
group. Only 10 of these were also included in the
echocardiographic group above.

Since all tests could not be performed in all patients,
we attempted to compare the patients by the following
method. A scoring system was devised in which each
patient received one point for each abnormal category.
The number of points was then summed and divided by the total number of categories and expressed as a percentage. The categories consisted of (1) symptoms of myocardial dysfunction, (2) ventricular enlargement by physical examination, and (3) conduction disturbances or dysrhythmias by electrocardiogram or (4) by Holter monitoring, (5) echophonocardiogram and (6) resting radionuclide ventriculograms. The maximum score possible was 6 points per visit, or 100%. By this system, 8 patients improved, 3 patients showed no change, and 5 patients deteriorated.

Discussion

We have followed 16 patients with myopericarditis for a mean period of 2.7 years following clinical recovery using a wide variety of non-invasive tests of cardiac function. The diagnosis of myopericarditis was based on strict clinical criteria. Patients were less than 55 years of age, did not have known prior cardiac disease or alcoholism, and had new cardiac abnormalities during symptoms of an acute viral infection. Evidence of a viral infection was found in 44%. The large prevalence of cardiac symptoms and abnormalities by examination and by electrocardiograms is explained by the strict criteria used in identifying patients with myopericarditis. Holter monitoring, echophonocardiograms, and radionuclide ventriculograms identified abnormalities in 57, 67, and 64%, respectively.

During this stage, global and regional dysfunction of either or both ventricles was not unusual. Following apparent recovery, cardiac symptoms and abnormalities by examination and non-invasive testing continued to be common. Seventy-five percent of patients had one or more abnormal tests of cardiac function. In most, these findings were subclinical although one patient died. By our scoring system, 8 patients had improved, but 5 patients showed an increase in the number of abnormal tests.

Our findings indicate that persistent cardiac dysfunction after recovery from acute myopericarditis is not unusual. These findings are consistent with the observations of others. Bengtsson & Lamberger (1966) noted persistent electrocardiographic abnormalities following recovery from presumed myocarditis, as did Bergstrom et al. (1970). Residual electrocardiographic findings were observed in four and chronic heart failure in three of 19 patients up to 10 weeks after acute heart disease due to Coxsackievirus B by Sainani et al. (1975); five of those with abnormal tracings had cardiomegaly. Smith (1970) described 6 patients with abnormal electrocardiograms for 6 months to 6 years and 3 with residual cardiomegaly by chest X-ray following acute Coxsackievirus B heart disease. The demonstration of persistent abnormalities by non-invasive testing of cardiac function following clinical recovery supports the postulate that one cause of the syndrome of dilated cardiomyopathy may be acute viral myopericarditis.

References


Serial cardiac function tests in myocarditis.


doi: 10.1136/pgmj.62.728.577

Updated information and services can be found at:
http://pmj.bmj.com/content/62/728/577

These include:

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/