limbs has been described in association with abdominal aortic grafts (Dacron grafts). 22, 24

REFERENCES

8 Mendelowitz M. Clubbing and hypertrophic osteoarthropathy. Medicine 1942; 21:269-306
16 Fraser RG, Pare JAP, Pare PD, Fraser RS, Genereux GP. Diagnosis of disease of chest. 3rd ed. Philadelphia: WB Saunders; 1988; 1:404 (ch 3)

Spontaneous Regression of Cardiomyopathy in a Patient with the Acquired Immunodeficiency Syndrome*

Joseph F. Hakas, Jr., M.D.; Thomas Generalovich, M.D.;

Cardiac involvement is common in patients with the acquired immunodeficiency syndrome (AIDS) and, when symptomatic, it portends a poor prognosis. We present a case of marked spontaneous regression of cardiomyopathy in a patient with AIDS. To our knowledge, this is the first reported case of spontaneous recovery of ventricular function in an AIDS patient.

(Chest 1991; 99:770-72)

VEF = ventricular ejection fraction

Cardiovascular abnormalities are commonly found in patients with the acquired immunodeficiency syndrome (AIDS). We present a case of idiopathic cardiomyopathy in a patient with AIDS who experienced spontaneous resolution of congestive heart failure associated with improvement in ventricular function. To our knowledge, this is the first reported case of such a recovery.

CASE REPORT

A 32-year-old woman was found to have human immunodeficiency virus (HIV) infection (Walter-Reed stage 5, CDC group IV-B) in November 1987 when she developed Guillain-Barré syndrome. A history of intravenous cocaine and heroin abuse was obtained. Echocardiography was normal. Her clinical course was stable until June 1988, when she presented with onset of peripheral edema, abdominal fullness, and dyspnea. Her only medication was methadone.

Physical examination revealed a thin woman, afebrile, with normal vital signs. Marked jugular venous distention with prominent V waves was noted without pulsus paradoxus. The lungs were clear. Cardiac examination revealed a right ventricular heave, summation gallop, and a grade 2/6 pansystolic murmur over the xyphoid with inspiratory augmentation. Pulsatile hepatomegaly and 2+ bilateral edema were present.

Laboratory data at the time of hospital admission showed hemoglobin of 11.4 g/dl, WBC count of 4,800 cu/mm (normal differential), and normal creatinine. Room air arterial oxygen saturation was 92 percent. Chest roentgenography showed clear lung fields with a markedly increased cardiac silhouette (Fig 1). Electrocardiography demonstrated sinus tachycardia with left atrial abnormality, decreased voltage, and diffuse T-wave flattening.

Echocardiography showed biventricular dilatation with generalized hypokinesis, paradoxic septal motion, and small pericardial effusion. Doppler studies demonstrated pulmonic and tricuspid insufficiency. Gated radionuclide ventriculography revealed a left ventricular ejection fraction (VEF) of 36 percent; right VEF was 37 percent by first pass technique. Gallium and perfusion lung scan results were normal. All cultures, antibody titers, rheumatologic studies, and endocrine studies were normal.

*From the Division of Cardiology, Department of Medicine, The Mercy Hospital of Pittsburgh.
†Senior Medical Resident.
‡Director, Coronary Care.
Reprint requests: Dr. Generalovich, 1400 Locust, Pittsburgh 15219

770

Spontaneous Regression of Cardiomyopathy (Hakas, Generalovich)
Following routine treatment of congestive heart failure, right heart catheterization with endomyocardial biopsy was performed (Table 1). Findings included left ventricular dysfunction, pulmonary hypertension, and low cardiac output. Biopsy specimens of the right ventricle were subjected to the following: staining and culture for bacteria, fungi, and mycobacterium; HIV culture; and histologic study and electron microscopy. All were normal.

The patient was discharged from the hospital on a regimen of vasodilators, diuretics, and digitals. Nuclear ventriculography in October 1988 demonstrated further diminution in right and left VEF to 28 and 26 percent, respectively. Zidovudine (azidothymidine [AZT]) therapy was prescribed, but it was discontinued in December 1988 due to neutropenia. From January through March 1989, the patient was incarcerated in another state where treatment with all medication was discontinued.

In March 1989, the patient returned to our institution without complaints. Physical examination revealed resolution of the abnor-

<table>
<thead>
<tr>
<th>Table 1—Cardiac Catheterization Data*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td><strong>Pressures, mm Hg</strong></td>
</tr>
<tr>
<td>RA</td>
</tr>
<tr>
<td>12</td>
</tr>
<tr>
<td>RV</td>
</tr>
<tr>
<td>50/18</td>
</tr>
<tr>
<td>PA</td>
</tr>
<tr>
<td>50/30, mean:37</td>
</tr>
<tr>
<td>PAWP</td>
</tr>
<tr>
<td>6</td>
</tr>
<tr>
<td>LV</td>
</tr>
<tr>
<td>140/0</td>
</tr>
<tr>
<td>LVEDP</td>
</tr>
<tr>
<td>8</td>
</tr>
<tr>
<td>Aorta</td>
</tr>
<tr>
<td>140/95, mean:105</td>
</tr>
<tr>
<td>Cardiac output, L/min</td>
</tr>
<tr>
<td>3.1</td>
</tr>
<tr>
<td>Cardiac index, L/min/m²</td>
</tr>
<tr>
<td>2.0</td>
</tr>
<tr>
<td>Systemic vascular resistance, dyn/cm(-5)</td>
</tr>
<tr>
<td>Pulmonary vascular resistance, dyn/cm(-5)</td>
</tr>
<tr>
<td>Arterial O₂ saturation, %</td>
</tr>
<tr>
<td>87</td>
</tr>
<tr>
<td>Mixed venous O₂ saturation, %</td>
</tr>
<tr>
<td>42</td>
</tr>
<tr>
<td>A-V O₂ difference, vol %</td>
</tr>
<tr>
<td>6.0</td>
</tr>
</tbody>
</table>

*RA = right atrium; RV = right ventricle; PA = pulmonary artery; PAWP = pulmonary artery wedge pressure; LV = left ventricle; LVEDP = left ventricular end-diastolic pressure.

mal cardiopulmonary findings and roentgenographic cardiomegaly. Echocardiography demonstrated improvement in biventricular wall motion with resolution of the valvular insufficiencies. Right and left VEF by radionuclide ventriculography were now 33 and 45 percent, respectively. Repeated cardiac catheterization (Table 1) showed normal coronary arteries and only mild inferior hypokinesis. Cardiac output and pulmonary and systemic hypertension had markedly improved. The patient has continued to do well clinically with no medications.

**DISCUSSION**

Since 1984, a number of postmortem studies have documented cardiovascular abnormalities in 24 to 75 percent of the AIDS population. Myocarditis has been found postmortem in more than 50 percent of all AIDS patients, and ventricular dysfunction occurs in up to 42 percent of those in premortem studies. With symptomatic cardiac involvement, there is a significant reduction in four-month survival.

Excluding the opportunistic myocarditides, the etiology of ventricular dysfunction in AIDS is unknown. In one series, all patients with biventricular enlargement had histologic myocarditis at necropsy. Some have speculated that the HIV might directly induce myocarditis, and, in one patient, HIV has been cultured from an endomyocardial biopsy specimen. Other theories implicate vitamin deficiencies and cardiotoxins, both environmental and pharmacologic.

Despite extensive study, the cause of our patient's cardiomyopathy remains unknown. The myocarditis demonstrated postmortem in similar patients has been mild and patchy and, therefore, easily missed by endomyocardial biopsy. There was no evidence for nutritional deficiencies, and, with the exception of cocaine, she had no history of exposure to known cardiotoxins. Cocaine has been implicated in ventricular dysfunction, but the majority of those patients have clinical evidence of ischemia. Improvement in ventricular function has been reported with AZT therapy; however, our patient's recovery occurred after discontinuation of AZT therapy.
This case emphasizes potentially dramatic recovery from severe HIV-associated cardiomyopathy. We believe that, once treatable opportunistic infections have been ruled out, aggressive medical support is indicated in these patients.

REFERENCES
4 Kinney EL. Cardiac complications in AIDS: which are significant, which to treat. J Crit Illness 1989; 4:49-57

Fatal Adult Respiratory Distress Syndrome Following Successful Treatment of Pulmonary Strongyloidiasis*
John Randall Thompson, M.D.,† and Rolando Berger, M.D., F.C.C.P.‡

Hyperinfection with Strongyloides stercoralis occurs mostly in immunocompromised patients, including those treated with systemic steroids. A case of Strongyloides-induced adult respiratory distress syndrome was recently reported, and we now report a case in which fatal ARDS appeared to result from the successful therapy of massive parasitic infection.

Although extraintestinal hyperinfection with Strongyloides stercoralis can occur in normal individuals, the vast majority of cases occur in immunocompromised hosts. A case of Strongyloides-induced ARDS was recently reported, but we believe our patient constitutes the first reported case of ARDS developing after successful therapy of the parasitic infection and coinciding with the rapid taper of the steroid dose.

*From the Division of Pulmonary and Critical Care Medicine, Veterans Administration and University of Kentucky Medical Centers, Lexington.
†Pulmonary fellow.
‡Associate Professor of Medicine.
Reprint requests: Dr. Berger, VA Medical Center, 111-H, Lexington, KY 40511

CASE REPORT

A 65-year-old man from eastern Kentucky was admitted to the hospital because of progressive shortness of breath. Two months earlier, he had been diagnosed by transbronchial lung biopsy as having idiopathic interstitial pulmonary fibrosis. At that time, a white blood cell differential count revealed 9,000 cells/μm, with 7 percent eosinophils. The patient was started on oral prednisone (60 mg/day), and because of chronic complaints of vague abdominal discomfort, cimetidine was also prescribed. Excellent clinical response was objectively documented at the three week follow-up evaluation.

Prednisone therapy was continued, and on the eighth week, the patient returned with renewed dyspnea and new onset of cough productive of yellow-white sputum. Physical examination revealed central cyanosis, a respiratory rate of 32 breaths per minute, blood pressure 140/80 mm Hg, pulse rate, 126 beats per minute, and rectal temperature 37.5°C (99.6°F). Examination of the chest disclosed diffuse bilateral crackles. The abdomen was unremarkable, with normal bowel sounds.

Arterial blood gas analysis (FiO2, 0.21) showed a PaO2 of 32 mm Hg, a PaCO2 of 29 mm Hg, and a pH of 7.51. The WBC count was 13,700 cells/μm, with zero percent eosinophils. A chest roentgenogram (Fig 1) revealed bilateral interstitial/alveolar pulmonary infiltrates which had worsened only slightly since the film from eight weeks earlier.

The patient was admitted to the intensive care unit and given 100 percent oxygen by tight-fitting face mask. A repeat blood gas showed a PaO2 of 91 mm Hg, a PaCO2 of 34 mm Hg, and a pH of 7.49. The Gram-stain of the sputum revealed abundant polymorphonuclear cells and few bacterial organisms. Smears for AFB and fungus were negative. Bronchoscopy was not attempted because of severe hypoxia, and the patient refused intubation and/or open lung biopsy. Because of the original GI complaints and mild eosinophilia, wet preparations of sputum were examined for parasites and numerous larvae of S stercoralis were readily identified (Fig 2).

Stool samples were also positive for larvae. Therapy was started with oral thiabendazole, 1.5 g every 12 hours. The prednisone dose was decreased from 60 to 40 mg on day 1 of therapy, to 20 mg on day 2, and to 10 mg on day 3. The patient initially showed clinical improvement, and serial sputum and stool samples revealed a decreasing number of larvae, with complete disappearance of the