Henry Ford Hospital Medical Journal

Volume 23 | Number 3 Article 6

9-1975

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Henry Ford Hosp. Med. Journal
Vol. 23, No. 3, 1975

Congestive Cardiomyopathy as an End Stage of Toxoplasma Myocarditis

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Congestive cardiomyopathy is a clinical entity which may be due to many different etiologies in addition to the primary type. The case presented defines the etiology in a 30-year-old patient as previous acute toxoplasma myocarditis. This diagnosis was corroborated by diagnostic rise of fluorescent antibody and Sabin-Feldman dye test titers and response to specific therapy. Toxoplasmosis has to be considered among the causes of congestive cardiomyopathy.

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Acquired toxoplasma myocarditis and pericarditis have been well described in literature, [1-10] but congestive cardiomyopathy as a consequence of a clinically well documented toxoplasma myocarditis has been rarely documented during life.[1]

We are presenting a case of cardiomyopathy felt to be due to toxoplasmosis where the titers of Sabin-Feldman dye tests and indirect fluorescent antibody tests rose during the acute illness and returned to normal with clinical improvement after specific therapy.

Case Report

When first seen in June 1971, R. P. was a 31-year-old white butcher, with a five-week history of cough producing yellowish sputum, right pleuritic chest pain, and progressive dyspnea on exertion. He also experienced increasing fatigability and had been having fever and chills for three weeks. Three years earlier the patient had had clearly documented hepatitis but he denied any contact with cats or dogs.

The patient appeared toxic and acutely ill with a temperature of 38.2°C. Eye examination was normal. There was no lymphadenopathy but there was some jugular venous distention. The pulse was 130/min, regular, with pulsus alternans, and blood pressure of 110/86 mm Hg. The maximum cardiac impulse was 13 cm from the midsternal line in the left intercostal space. The pulmonic closure sound was accentuated and a prominent S3 gallop was heard. A grade II/VI holosystolic murmur, maximal at the apex, radiated to the axilla. There was no pericardial rub. Bronchial breath sounds were heard over the left lower lobe and there were bilateral basal rales.

Liver and spleen were not palpable, although there was 3+ ankle edema.

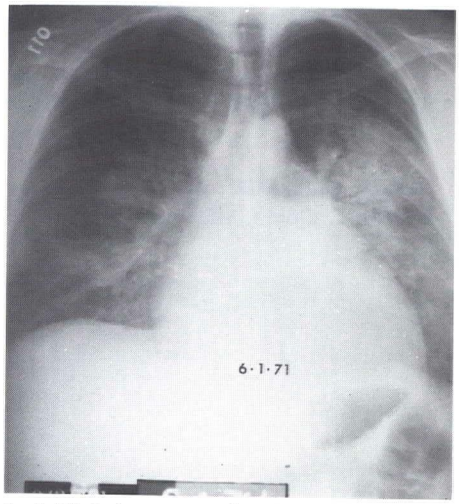
Laboratory studies disclosed a white blood cell count of 12,200/cu mm with normal differential, hemoglobin of 14.3 gm/dl and a Westergren sedimentation rate of 27 mm/hr. The following studies were normal: urinalysis, serum electrolytes, SGOT, LDH, CPK, fasting blood sugar, blood urea nitrogen, creatinine, T4 by column, ASO titer, VDRL, LE clot test, ANF, rheumatoid factor, and cold agglutinins. Multiple blood, urine, and sputum cultures showed no growth. Sputum for acid fast bacteria was negative repeatedly. The patient had negative complement fixation tests for trichinosis, Q fever, leptospirosis, psittacosis, histoplasmosis, and blastomycosis. The EKG was interpreted to show sinus tachycardia, left ventricular hypertrophy and nonspecific ST-T changes. The chest x-ray (Figure 1A) revealed cardiomegaly with infiltrates in the right upper lobe, left lower lobe, and right pleural effusion compatible with congestive heart failure and pneumonitis. Skin tests for acid fast bacteria, coccidioidomycosis, histoplasmosis and blastomycosis were negative. Serial viral neutralizing antibody titers against poliomyelitis, echovirus and coxsackie virus, A-9, B-1, B-2, B-3 and B-5 were all negative. A heterophile test was within normal limits. A cardiac blood pool scan suggested pericardial effusion. Pulmonary angiograms confirmed bilateral multiple pulmonary emboli and the presence of pericardial fluid. Intracardiac pressures are shown in Table 1. Eighty ml of serosanguineous pericardial fluid was aspirated. It was a characteristic exudate but cultures were negative for AFB, bacteria, or fungi. Cytologic examination was normal. Pleural fluid had features similar to those of the pericardial fluid. Deltoid muscle and skin biopsies were negative.

The patient continued to be febrile and toxic. Continuing the search for possible etiologies, it was learned that he frequently ate raw beef during his work as a meat cutter. Toxoplasmosis was suspected and Sabin-Feldman and indirect fluorescent antibody tests were ordered (Table 2).

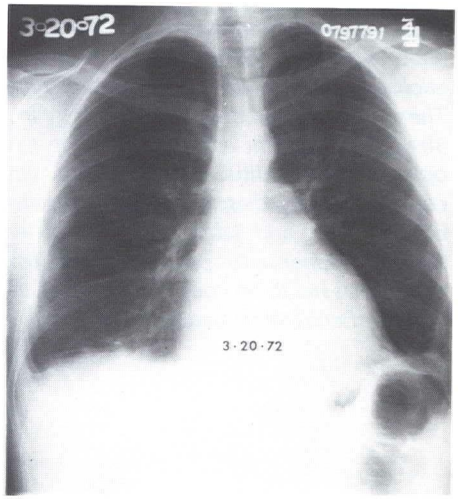
When a significant rise in titers for toxoplasmosis was reported on October 5, 1971, therapy was begun for a presumptive diagnosis of toxoplasma myopericarditis. This consisted of pyrimethamine 50 mg daily, sulfadiazine 4 gm daily and folinic acid 6 mg intramuscularly daily for one month.

Soon after initiation of this regimen, the toxicity subsided, the patient became afebrile and made a slow but steady recovery. Although he achieved a stable condition, he continued to have considerably reduced exercise tolerance

Figure 1



A. Initial chest x-ray showing cardiomegaly with bilateral infiltrates, pulmonary edema, and right pleural effusion.



B. Chest x-ray nine months later showing resolution of congestive and infiltrative changes with persistent right pleural scarring and cardiomegaly.

and evidence of congestive cardiomyopathy. Repeat chest x-rays disclosed persistent cardiomegaly with resolution of the pulmonary infiltrates (Figure 1B). An intermittent S3 gallop and a holosystolic apical murmur of mitral regurgitation persisted. Subsequent catheterization was compatible with the diagnosis of congestive cardiomyopathy, with moderate elevation of right-sided pressures, elevated pulmonary capillary

TABLE 1

CATHETERIZATION DATA

	6/16/71		10/1/71		10/27/72	
Pressures	**Syst/Diast**	**Mean**	**Syst/Diast**	**Mean**	**Syst/Diast**	**Mean**
SVC		8		7		4
IVC		7		10		2
RA		7		10		4
RV	58/3-14		54/0-23		40/0-4	
MPA	58/24	36	54/22	34	36/17	26
RPA	60/26	37	54/23	35	37/18	25
RIW		23		21		19
CA			108/74	84	124/80	100
LV			116/0-17-26		120/0-27	
CO			3.7 1/min.		4.5 1/min.	
CI			1.9 1/min.		2.5 1/min.	

Table I:

The initial catheterization data show elevated right ventricular, pulmonary arterial and pulmonary capillary wedge pressures. On 10/1, there is slightly less pulmonary hypertension, elevated left ventricular end diastolic pressure and low cardiac output and cardiac index. The last study one year later shows only minor improvement.

TABLE 2

TOXOPLASMA TITERS

Date	**S.F. Dye Test**	**Indirect Fluorescent Antibody Test**
7-10-71	1:256	1:128
7-20-71	1:256	1:128
10-5-71	1:512	1:512
10-15-71	1:512	1:512
11-12-71	1:32	1:256
8-23-72	Negative	1:256
10-30-72	1:64	1:128

wedge pressures, and left ventricular end-diastolic pressures (Table 1). Cardiac output and cardiac index remained low. Angiography revealed gross mitral insufficiency with poor ventricular contractions. The coronary arteries appeared to be within normal limits and surgical treatment for the mitral valve lesion was considered inappropriate.

Discussion

Our patient presented many features compatible with the diagnosis of congestive cardiomyopathy following acute toxoplasma myopericarditis. He had a history of many years of eating raw beef, one of the common sources of toxoplasma infection in man.[11] Cooking and freezing usually destroys the toxoplasma cyst. Desmonts et al[12] reported that 93% of Parisian mothers had elevated Sabin-Feldman titers because of their habit of eating raw or undercooked meat. In the same study, titer changes without clinical disease were also observed in a group of hospitalized children who were habitually fed with undercooked mutton.

Pneumonitis in the patient's right upper lobe and left lower lobe is compatible with toxoplasmosis. A rise in Sabin-Feldman dye test titers and indirect fluorescent antibody titers during clinical disease and the decrease in titers following specific therapy and clinical improvement are consistent with the diagnosis. Possible causes of myopericarditis such as viral infection, connective tissue disorder or alcoholism were ruled out clinically and by laboratory studies. A deltoid muscle biopsy to rule out trichinosis was negative.

Toxoplasma myocarditis responds variably to therapy. If there is associated heart failure, the response tends to be poor.[1] In our case, the response was slow with significant residual damage causing congestive cardiomyopathy. The pericardial involvement we encountered is not a frequent presentation of toxoplasmosis. Only a few cases have been reported.[1,4,5,6,7] Two cases of constrictive pericarditis have been attributed to toxoplasmosis.[6,7] Pulmonary embolism, a complication in our case, was also described in one of 11 cases reported by Arribada et al (1).

Arrhythmias were not a problem in our patient, although ventricular tachycardia,[9] Stokes-Adams attacks, [10] AV block,[1] supraventricular tachycardia,[8] atrial flutter and fibrillation,[1,2,4] bundle branch block,[1] PVC's and paroxysmal nodal rhythm have all been described. Arrhythmia in this disease may be caused by a reaction to ruptured intramyocardial toxoplasma cysts.

Toxoplasmosis can present with many different clinical manifestations.[13] Judging from the positive antibody reaction encountered in various populations,[11,14] the facts that almost any mammal or bird can be infected and that the parasite can be found in any tissue of the human body except erythrocytes, it is conceivable that many cases of subclinical toxoplasma cardiomyopathy escape detection.

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