INTRODUCTION

Myocarditis is an inflammatory disease of the heart muscle that can have a toxic, autoimmune, infectious, or most frequently, idiopathic etiology. Infectious myocarditis often passes unnoticed when it occurs in the context of a systemic febrile condition, which is why its true incidence is unknown.1-4

*Coxiella burnetii* myocarditis is a rare but severe clinical form of acute Q fever. We report the case of a 40-year-old man hospitalized for acute febrile syndrome. Forty-eight hours later, he presented dyspnea, orthopnea, and paroxysmal nocturnal dyspnea; cardiac auscultation revealed a third sound and echocardiography showed a diffusely hypokinetic and dilated left ventricle (30% ejection fraction). Serological studies showed antibodies against phase-II *C. burnetii* antigens (IgG titer 1:320 and IgM 1:50). The patient was treated with losartan, furosemide, and clarithromycin, resulting in rapid improvement. Six months after admission, the echocardiographic changes had completely disappeared.

**Key words:** Myocarditis. Cardiomyopathy. Q fever. *Coxiella burnetii.*

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CLINICAL CASE

A 40-year-old man consulted for a 4-day fever. The patient was a smoker, 20 years/pack, and had no other toxic habits or other cardiovascular risk factors. In spi-
te of residing in an urban area, he kept parakeets and a rabbit. He had been hospitalized 15 years earlier for chest pain, which was diagnosed as acute idiopathic pericarditis. He did not have a history of effort dyspnea, orthopnea, nocturnal paroxysmal dyspnea, or peripheral edema.

The patient consulted for a high fever of 4 days duration that exhibited no focality. The physical examination and chest radiograph at admission indicated no abnormality. Forty-eight hours later he began to experience dyspnea, nocturnal orthopnea, and paroxysmal dyspnea. In the physical examination, the intense deterioration of the patient’s general condition was notable, with a temperature of 39 °C, heart rate 110 beats/min, respiratory rate 32 breaths/min, cyanosis, blood pressure 100/60 mm Hg, ventricular gallop, and inspiratory crepitations in both lung bases. There was no jugular inculigation, liver enlargement, or edema. The laboratory findings were 8000 leukocytes/µL (83% polymorphonuclear), hemoglobin 16 g/dL, platelets 140 000/µL, glucose 100 mg/dL, creatinine 0.5 mg/dL, AST 57 U/L, ALT 58 U/L, GGT 62 U/L, AlP 156 U/L, LDH 850 U/L, CPK 55 U/L, ESR 12 mm Hg, PCR 11 mg/dL. Arterial gasometry with FiO₂ 0.41 revealed pH 7.48, P O₂ 57 mm Hg, and P CO₂ 21% disclosed pH 7.48, P O₂ 57 mm Hg, and P CO₂ 30 mm Hg. The ECG revealed sinus rhythm 114 beats/min with non-specific repolarization disorders. The clinical evolution was favorable, with amelioration of the patient’s general condition, no jugular inculigation, liver enlargement, or edema. The echocardiographic study revealed a dilated left ventricle with diffuse hypokinesia and an ejection fraction of 30%. Antibiotic treatment was begun with intravenous ceftriaxone and clarithromycin (for 14 days) as well as furosemide and losartan. The clinical evolution was favorable, with abatement of the fever in 48 h and amelioration of the symptoms of heart failure.

Blood cultures were negative. Serological study by indirect immunofluorescence of a single sample revealed antibodies against phase II antigens at IgG titers of 1:200 or more and/or IgM 1:50 or more in a single sample. The diagnosis of acute Q-fever is specific and there are no cross-reactions with rickettsia or other microorganisms. The diagnosis of certainty can be made by cell cultures, but these are complex techniques that entail a risk of infection for the personnel that perform them. In most centers, when we knew the result of serology, our patient was totally asymptomatic and had received 2 weeks of treatment with clarithromycin, which is effective against C. burnetii, which is why doxycycline, the drug of choice, was not administered. We do not know what influence antibiotic treatment administered in the acute phase of infection could have on the evolution of myocarditis.

The available information on myocarditis due to C. burnetii is scant. We found only two series of cases, and in one of them the authors did not describe the clinical characteristics of the form of presentation. Fournier et al reported 8 cases of myocarditis in a series of 1276 patients with acute Q fever. Seven presented dilated cardiomyopathy, of which 6 evolved favorably and recovered cardiac function in the following months. In Spain, 3 cases have been described that were manifested by chest pain, ventricular fibrillation, and heart failure. All had a favorable outcome.

The pathogenesis of myocarditis in humans is unknown, although there are animal models in which immunological mechanisms have been implicated. In the specific case of Q fever, information is even more scant, although the presence of C. burnetii in myocar-
dium has been demonstrated. 

In conclusion, *C. burnetii* is a microorganism that must be investigated in patients with febrile syndrome and myocarditis. Patients who develop heart failure have a high mortality, but they can recover completely.

REFERENCES