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_.

Full English text available at: www.revespcardiol.org

---

ACUTE MYOCARDIAL FAILURE IN A YOUNG MAN: Q-FEVER MYOCARDITIS

José Murcia, Sergio Reus, Vicente Climent, María I. Manso, Íñigo López and Antonio Tello

*Servicios de Medicina Interna y *Cardiología. Hospital General Universitario de Alicante.

We report the case of a previously healthy young man who presented reversible acute cardiac failure in the context of systemic infection by _C. burnetii_.

We reviewed the literature in Spanish and English found in a Medline search using the key words «myocarditis» and «Q-fever». Since 1980, 20 cases of myocarditis due to _Q-fever_ have been reported.

---

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 focolay. 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 ingurgitation, 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 156 U/L, ALT 58 U/L, GGT 850 U/L, CPK 55 U/L, ESR 12 mm Hg, PCR 11 mg/dL. Arterial gasometry with FiO 2 0.6 revealed a pH 7.48, P O 2 57 mm Hg, and P CO 2 30 mm Hg. The ECG revealed sinus rhythm 114 beats/min with non-specific repolarization disorders. Cardiomegaly with bilateral interstitial edema was evident in the chest radiograph, with bilateral Kerley B lines and pleural effusion. Echocardiography revealed a dilated left ventricle with diffuse hypokinesia and an ejection fraction of 30%. Antibiotic treatment was begun with intravenous ceftriaxone and clarithromycin. The clinical evolution was favorable, with abatement of the fever usually made by serology, either by demonstrating seroconversion, or, as in our patient, by the demonstration of antibodies against phase II C. burnetii 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 who perform them and they are not performed 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 al12 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,13 ventricular fibrillation,14 and heart failure.15 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.7 In the specific case of Q fever, information is even more scant, although the presence of C. burnetii in myocar-
diurn has been demonstrated.16

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

15. Lozano de León F, Gómez-Mateos JM, Grilo A. Miocarditis por fiebre Q. Rev Esp Cardiol (Barc) 1987;89:886.