Acute Q Fever with Atrioventricular Block, Israel

Karawan Badarni, Miry Blich, Yafit Atiya-Nasagi, Nesrin Ghanem-zoabi

Cardiac involvement in acute Q fever is rare. We report 2 cases of an advanced atrioventricular block in young adult patients in Israel who sought care for acute Q fever without evidence of myocarditis. Q fever should be suspected in unexplained conduction abnormalities, especially in febrile young patients residing in disease-endemic areas.

Q fever is a zoonosis caused by *Coxiella burnetii* bacteria; the main route of infection is through inhalation of infected aerosols (*1*). Acute Q fever is mainly a self-limited influenza-like illness but may manifest as pneumonia or hepatitis. Less common manifestations involve different organs of the nervous, cardiovascular, skin, gastrointestinal, and hematopoietic systems (*2*). Cardiac involvement in Q fever is usually observed in the chronic form and manifests as endocarditis, aortitis, and vascular aneurysm infection. Less common cardiovascular manifestations in acute Q fever include myocarditis, pericarditis, and acute endocarditis (*2*). We report 2 patients in Israel who had acute Q fever and advanced atrioventricular block as the cardiac manifestation.

Patient 1 was a 48-year-old man was admitted to an intensive cardiac care unit (ICCU) for dizziness and electrocardiogram (EKG) abnormalities. He had type 2 diabetes mellitus and hypertension. Ten days before admission, he had fever and myalgia; his symptoms lasted for 1 week and resolved spontaneously. Three days later, he experienced dizziness; he sought care, and an EKG showed a new atrioventricular block.

At admission, the patient's vital signs were within reference ranges except for bradycardia (37 bpm); results of a physical examination was unremarkable. Complete blood count showed decreased hemoglobin (12.5 g/dL) and elevated C-reactive protein. Liver enzyme, cardiac troponin, and coagulation test results were all within reference ranges, and blood cultures were negative. Serology testing for brucellosis was negative. Chest radiograph showed no pathology. His EKG showed a complete atrioventricular block with a ventricular escape rate of 35 bpm and a QRS wave duration of 140 ms (Figure 1). A cardiac evaluation including resting EKG with standard and high leads, 12-lead Holter EKG, and echocardiogram all excluded a channelopathy or cardiomyopathy; cardiac stress testing excluded ischemia. Fluorodeoxyglucose positron emission computed tomography (FDG/PET-CT) showed no pathological uptake in the myocardium or elsewhere in the body. Because of his symptomatic atrioventricular block, a permanent pacemaker was implanted.

The patient had no familial history of cardiac conduction defects or cardiomyopathy. We suspected infectious etiology because of his history of a febrile illness preceding the atrioventricular block occurrence. This patient lived in northern Israel, where Q fever is endemic, and worked in a slaughterhouse. Serology testing for *C. burnetii* demonstrated positive phase II IgM, phase II IgG with a titer of 1:100, and negative phase I IgG. PCR testing for *C. burnetii* (targeting insertion sequence 1111) in serum returned negative results. Repeated serologic testing 4 weeks later demonstrated titers of phase II IgG had increased to 3,200. We used an in-house indirect immunofluorescence assay with a cutoff of 1:100 to diagnose definitive acute Q fever and treated the patient with doxycycline (100 mg 2×/d for 2 weeks). Pacemaker testing 5 months and 12 months later showed no restoration of normal conduction. Repeated serologic testing showed no evidence for progression to chronic disease, and repeated echocardiography and FDG/PET-CT showed no focal infection.

Patient 2 was an otherwise healthy 23-year-old man referred to the ICCU for recurrent episodes of

Author affiliations: Rambam Health Care Campus, Haifa, Israel (K. Badarni, M. Blich, N. Ghanem-zoabi); The Ruth and Bruce Rappaport Faculty of Medicine of Technion, Haifa (M. Blich, N. Ghanem-zoabi); Israel Institute for Biological Research, Ness-Ziona, Israel (Y. Atiya-Nasagi)

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