Chronic Q Fever Detection in the Netherlands

To the Editor—The article on the current epidemic of Q fever in Holland, which has involved more than 4000 diagnoses, is very interesting because it makes it possible to make a preliminary point on the detection of chronic infection, the diagnosis of which is particularly difficult [1]. One of the elements of the diagnosis is serology, and there is an old consensus that the detection of immunoglobin G antibodies against phase I antigens is the best method for the diagnosis of Q fever. Recently, using data from our center, which has the most experience with the diagnosis of Q fever, we revaluated the predictive value of antibodies against phase I *Coxiella burnetii* [2]. Our results are comparable to those that are presented here. Antibodies against phase I antigens at a titer of 1/800 had a positive predictive value of only 37%, while antibodies at titers higher than 3200 had a positive predictive value of 75%. Between these 2 titers, the diagnosis should be confirmed by polymerase chain reaction (PCR) [3]. It is necessary to be careful when analyzing serum samples with very high antibody titers because, for an unknown reason, the PCR is frequently negative in these cases [3]. However, in the titer window between 1/800 and 1/6400, PCR plays a critical role in determining whether the disease is progressing along with the clinical conditions.

In all cases, it is necessary to be very careful when estimating the incidence of chronic Q fever because, as in a case reported in this work, a diagnosis of chronic Q fever can be made up to 10 years after the primary infection. In addition, we disagree with the interpretation of the echographic data cited here [4]. The valvulopathies predisposing patients to Q fever endocarditis are, first, prosthetic valves (which are present in 0.5% to 1% of the population in France) [5]; then, aortic bicuspidy [6] (0.5%–2% of the population); more rarely, mitral valve prolapse (0.6%–2.4% of the population) [7, 8]; and even more rarely, moderate aortic or mitral leaks (3% of the population) [9]. It is not true that more than 50% of controls or patients with acute Q fever have 1 or more of these valvular lesions. The tiny traces of valve insufficiency are physiological. However, aortic bicuspid valve and mitral valve prolapse can be clinically silent and must be detected to avoid the evolution towards chronicity. They account for 0.5%–3% of the patients. Among patients with endocarditis following acute Q fever, comparisons of prevalence of the different valvulopathies [10] with the corresponding prevalence reported in a population unexposed to acute Q fever infection shows the following gradient of risk: higher risk for aortic bicuspidy 19.06 (95% CI [4.29–84.63]), followed by 4.23 (2.08–8.58) for the mitral valve prolapse and 20.06 (10.09–39.87) for a moderate mitral insufficiency. Despite the heterogeneity of the unexposed populations used to compute these prevalence ratios, these results give an important insight about priorities in endocarditis prevention and clinical management of patients with valvulopathies.

In conclusion, given these data, it is premature to decide that echocardiography is useless in detecting factors that predispose patients to endocarditis. Based on our data, the failure to discover chronic endocarditis in a patient diagnosed with acute Q fever because that patient was not evaluated for underlying predisposing factors by echocardiography at the time of diagnosis may jeopardize the health of the patient and put the physician at risk for being sued.

Note

Potential conflicts of interest. All authors: No reported conflicts.

All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

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