Viral Endocarditis or Simple Viral Disseminated Infection?

To the Editor—We read with interest the article by Blumental et al, “Enteroviral Infection of a Cardiac Prosthetic Device” [1]. The authors described a case of recurrent endocarditis in a 4-month-old boy with a Gore-Tex atrioventricular patch. They grew coxsackievirus B2 from several specimens, including the removed patch, an intracardiac vegetation, and respiratory and stool samples, and claimed that the patient had viral endocarditis. This case report questions once more the role of viruses in endocarditis. To date, despite old experiments demonstrating that coxsackieviruses can cause endocardial infection in mice and monkeys [2, 3], the role of viruses in human endocarditis remains uncertain.

The main problem with emerging pathogens is to demonstrate their causative role. In endocarditis, at the minimum, the agent should be found in endocardial lesions. In Blumental et al’s article, there was no direct evidence of endocardial cell infection with coxsackieviruses, because neither were cytopathic effects shown by electron microscopy nor were viruses found in cells with use of immunohistochemistry. There is no doubt that the patient developed a coxsackievirus B2 viremia, because the virus was retrieved from various specimens obtained during the third cardiac surgery. However, we believe that blood-circulating viruses contaminated all tissues rather than infected specifically endothelial cells. This hypothesis is supported by the fact that the patient underwent a fourth valvular surgery for an intracardiac false aneurysm 10 weeks later but no virus could be detected.

In contrast, in their analysis, the authors ruled out a bacterial etiology despite a blood culture positive for viridans streptococcus during the second endocarditis episode. This is a critical issue, because viridans streptococci are the main agents of subacute endocarditis, the histological form of endocarditis diagnosed in this patient. The negative result of the culture of a blood sample obtained at admission during the first endocarditis episode may be explained by the oral amoxicillin administered to the patient before admission. However, although antibiotics may have sterilized blood cultures, the duration of the antibiotic regimens given at each of the first 2 endocarditis episodes (7–14 days) is too short for curing a streptococcal endocarditis, for which antibiotic regimens of 4–6 weeks are recommended [4]. Therefore, we assume that the relapsing endocarditis may have been caused by streptococci rather than viruses.

Finally, in a previous study, we investigated 759 patients with blood culture–negative endocarditis [5], including 250 patients with a valvular prosthesis (bioprosthesis, mechanical valve, or pacemaker), for the involvement of enteroviruses in their cardiac infection but could not find any. In consequence, although we cannot formally rule out the role of enteroviruses in endocarditis, this etiology should thoroughly be proven, notably by showing the presence of the cytopathogenic images and viruses in endocardial cells. In conclusion, the case report of Blumental et al [1] stimulates further investigation of enteroviruses as putative agents of endocarditis.

Note

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

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References


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