
TO THE EDITOR: The recent report by Zweifel et al.1 is an important paper. The findings of such nontuberculous mycobacteria in lung granulomas of a patient with choroidal lesions so similar to those seen in active multifocal choroiditis (MFC) buttresses previous speculation that such agents are a cause of MFC, particularly in areas where histoplasmosis is not endemic.2 In fact, given the recognized unreliability of skin test evaluation and the ubiquitous environmental presence of nontuberculous mycobacteria, they could also be causative in histoplasmosis endemic areas.3,4

In this study, nontuberculous mycobacteria were found in lung granulomas of the one patient who came to autopsy but not in the choroidal lesions. Nor was choroidal tissue positive by 16S rRNA or polymerase chain reaction analysis. The authors suggest that this could be owing to several factors, such as postmortem time, antibiotic therapy, and low polymerase chain reaction sensitivity.

I cannot tell from the manuscript whether the retinal pigment epithelium (RPE) was subjected to the same analysis for M. chimerae as the choroid but, if not, another explanation can be found in the report of Rao et al.5 In their histologic analysis of a patient clinically diagnosed with panuveitis, mycobacterial DNA were found by polymerase chain reaction analysis in the RPE but not in the choroid. In fact, although there was granulomatous inflammation involving the choroid, acid-fast bacteria were found in the RPE only. These investigators suggest M. tuberculosis is preferentially sheltered in such macrophage-like tissue as the RPE. Could this explain the almost universal finding of this and previous clinical pathologic confirmation of MFC, that choroidal lesions so prominent clinically have been devoid of bacilli in histologic studies?

This report supports the hypothesis that M. chimerae is one of the causes of MFC and its chorioretinal scars which later become symptomatic if neovascularization occurs.

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Financial Disclosures: The authors have no proprietary or commercial interest in any materials discussed in this article.

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References