CORRESPONDENCE

Chlamydia pneumoniae and Atherosclerotic Plaque

To the Editor—According to Campbell et al. [1] in their recent study of Chlamydia pneumoniae in coronary tissue, staining with a Chlamydia-specific monoclonal antibody detected chlamydiae in 45% of the atherectomy specimens from patients with atherosclerotic plaque. However, staining appeared only in macrophages; smooth muscle cells did not stain, a result that appears to contradict previous findings by some of the same researchers [2]. This apparent difference is not explained in the recent publication [1].

The discrepancy might be due to the different activities of the lesion types used in the two studies. Patients in the most recent study had angina or acute myocardial ischemia (or both). According to Saikku et al. [3], such patients show reactivation of chronic C. pneumoniae infection and an increase of inflammatory activity in atherosclerotic plaque. In the inflammatory processes, the production of interferon-γ by T lymphocytes increases, thus inhibiting the proliferation of the smooth muscle cells and activating apoptosis in these cells [4]. The speed of the apoptosis causes considerable cell loss, yet few cells are visualized in the destruction process [5]. The intense loss of smooth muscle cells in the active atherosclerotic plaque may also cause destruction of C. pneumoniae–infected smooth muscle cells. Perhaps, by eliminating infected cells, accelerated apoptosis acts as a defense mechanism in chronic C. pneumoniae infection.

The discrepancy between the two atherosclerosis studies [1, 2] might also be explained by other studies that more solidly associate the inflammatory process in atherosclerotic plaque with infectious agents. Saikku et al. [3] and Linnanmäki et al. [6] attribute 70%-80% of all atheromatous disease to chronic C. pneumoniae infection.

Perhaps, the benefits of pre- [7, 8] or postdiagnosis and treatment with erythromycin [3] of chronically C. pneumoniae–infected patients with coronary disease could be investigated in double-blind studies. Such studies might also determine if these patients would benefit from repeated treatments with erythromycin for the control of C. pneumoniae infections.

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References


Reply

To the Editor—We appreciate the comments of Casas-Ciria and Rodriguez-Iglesias [1] regarding the differences found in antigen localization of Chlamydia pneumoniae in atherectomy and aorta tissue from patients in two previous studies [2, 3]. C. pneumoniae was found in macrophages in both studies; however, it was found in smooth muscle cells in tissue from aortic atheroma in only one study [3]. In evaluating this difference, it is important to note the different nature of the specimens examined, which may partly explain the failure to find C. pneumoniae antigen in smooth muscle cells in atherectomy tissue.

Atherectomy specimens were from persons with symptomatic coronary artery disease. Thus, one can surmise that these lesions came from unstable plaque and may have included more inflammatory tissue. In addition, the nature of directional atherectomy tissue resection is such that arterial tissue layers that lie closest to the lumen are removed. Usually, this results in collection of tissue that contains more fibrous material, extracellular matrix, and inflammatory cells. In contrast, the aorta specimens were cross-sections including all layers of the artery, which may be dominated by smooth muscle cells. In the study by Kuo et al. [3], C. pneumoniae–laden macrophages were subendothelial, while the smooth muscle cells were in the deeper tissue layer, as described in [3].

With respect to the speculation on apoptosis, the thoughts of Casas-Ciria and Rodriguez-Iglesias are intriguing but require more data.