Mechanism of Arterial Infection by Chlamydia pneumoniae

To the Editor:

The article by Gieffers et al1 in the January 23, 2001, issue of Circulation reiterates the general belief that lungs harbor Chlamydia pneumoniae organisms that are continuously released into the bloodstream. It is suggested that these organisms are carried by lung macrophages, which repeatedly infiltrate and infect atherosclerotic lesions.

There are certain pathological features of human atherosclerotic lesions that suggest that this mechanism may not be entirely correct. Chlamydia pneumoniae usually causes mild upper respiratory tract infections. If the organisms do indeed enter the lungs, then there is some difficulty in explaining how macrophages are dispersed into the bloodstream to infiltrate arteries. Lung macrophages drain to the lymphoid tissue of the lung and to the hilar glands, not into the circulation. It should also be borne in mind that blood monocytes, not macrophages,2,3 infiltrate atherosclerotic lesions. These blood monocytes are derived from spleen, lymphoid tissue, and bone marrow rather than from lung tissue. In fact, these cells cannot be derived from lung macrophages because lung macrophages contain anthracotic pigment, and no such pigment is noted in the cells that infiltrate the artery.

The pathology of the arterial lesion indicates that only 1 infective episode is necessary. The infection usually occurs in adolescence or earlier. The organisms possibly may enter arteries via the vaso-vasorum, inasmuch as early lesions are seen to occur in the lower intima. In this region, C pneumoniae infects intimal smooth muscle cells, causing rupture of the cells with dispersion of organisms and infection of adjacent intimal tissue. A florid monocyte and T-lymphocyte infiltrate occurs in relation to this damage. Monocytes enter the subendothelial space, are transformed into a phagocytic state,2,3 and are seen to ingest C pneumoniae organisms.4

With progression, necrosis and ulceration of the lesions occur. This is especially prominent in the aorta, where the surface is usually covered with open atherosclerotic ulcers. These open ulcerating lesions release necrotic tissue, together with cells and organisms, into the circulation. It is not necessary to look further than this for the source of infection or for the C pneumoniae–containing macrophages in the circulation.

There are some unclear aspects of the mechanism by which C pneumoniae reaches the arteries from the respiratory tract,1 but because the infection occurs in childhood, the benefit to be derived from treating this aspect of the disease in later years is questionable. In adults, the central focus of treatment should be directed at the lesions in the arterial vasculature.5

Allan Shor, MB, ChB
Department of Pathology
National Center for Health
University of Witwatersrand
Johannesburg, South Africa

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Allan Shor

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