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Autor(en) des Beitrags:
May, AE; Redecke, V; Grüner, S; Schmidt, R; Massberg, S; Miethke, T; Ryba, B; Prazeres da Costa, C; Schömig, A; Neumann, FJ

Titel des Beitrags:
Recruitment of Chlamydia pneumoniae-infected macrophages to the carotid artery wall in noninfected, nonatherosclerotic mice.

Abstract:
OBJECTIVE: Monocyte recruitment into the subendothelium is a crucial step in atherogenesis. Chlamydia pneumoniae resides in circulating monocytes and in the atherosclerotic vascular wall. However, the role of C pneumoniae for monocyte recruitment is unknown. The aim of this study was to examine the impact of C pneumoniae on monocyte adhesion and migration. METHODS AND RESULTS: C pneumoniae-infected, fluorescence-labeled mouse macrophages (ANA-1) were injected intravenously into noninfected, healthy mice. In vivo videomicroscopy showed increased rolling and firm adhesion to the carotid artery compared with noninfected macrophages. In vitro, C pneumoniae infection (yielding 25% to 35% infected monocytes) increased adhesion of human monocytes or MonoMac6 cells to human umbilical vein endothelial cells and improved cell migration through endothelial-like ECV604 cells. Cell adhesion was inhibited by antibody blockade of very late antigen-4, lymphocyte function-associated antigen-1, macrophage antigen-1, or urokinase receptor, which were found upregulated or activated on C pneumoniae infection (flow cytometry). In contrast, C trachomatis did not induce monocyte adhesion at comparable infection rates (25% to 35%), indicating a unique activation pathway for C pneumoniae. Polymyxin
B did not affect C pneumoniae-induced adhesion, excluding a relevant role of lipopolysaccharide in this process. CONCLUSIONS: These data indicate that C pneumoniae can direct monocytes to predilection sites of nonatherosclerotic vessel walls in vivo by activation of the integrin adhesion receptor system.

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