Autoreactive CD4$^+$CD28$^-$ T Cells and Acute Coronary Syndromes

In Response:

The letter by Zal et al addresses an important issue. It is undisputed that CD4+CD28$^-$ T cells can be clonally expanded in the periphery and that T cells belonging to these clones infiltrate into atherosclerotic plaques.[1] How much of their activity that is antigen specific, however, is still a question of debate. The Weyand group has shown that CD4+CD28$^-$ T cells are able to lyse endothelial cells in vitro without the need for antigen recognition.[2] In addition, they have provided evidence that interleukin (IL)-12 enhances lesion recruitment of these cells.[3] Weyand and colleagues state "Thus, (CD4+)CD28$^-$ T cells functionally resemble NK cells, which have proinflammatory activity even in the unprimed state and respond to any IL-12–inducing host infection with a shift in tissue trafficking and accrual in inflammatory lesions".[3] At present, both antigen specific and unspecific activation of CD4+CD28$^-$ T cells are therefore possible scenarios, and more studies are needed to shed light on this issue.

Disclosures

None.

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