Neutrophil Count and Complex Lesions in Patients With Coronary Artery Disease

To the Editor:

In a recent article, Coller1 reviews the role of leukocytosis in vascular disease morbidity and mortality. Despite the exhaustive nature of the review, we were concerned that the author failed to address an issue of major importance, namely the relationship between neutrophil count and the presence of vulnerable atheromatous plaques in patients with coronary artery disease.

Acute coronary syndromes result mainly from occlusive coronary artery thrombosis at sites of plaque fissure, rupture, or superficial erosion. At angiography, disrupted or ulcerated plaques often appear as “complex” stenoses with rough contours or “filling defects” suggestive of intracoronary thrombosis. Complex stenoses are at a higher risk of rapid progression than smooth lesions,2 and we have recently shown that markers of both inflammation and macrophage activation predict rapid coronary artery disease progression in patients with chronic stable angina.3

In angiographic studies in patients with chronic stable angina, we have shown that neutrophil count is an independent predictor of the presence of multiple complex stenoses irrespective of coronary artery disease extent.4 Moreover, in patients with acute coronary syndromes, neutrophil count is also associated with coronary artery disease complexity.5 Furthermore, neutrophil infiltration of culprit lesions with release of elastase and myeloperoxidase has been implicated in the pathogenesis of atherosclerosis.6 These findings are important, as it has been shown that the presence of multiple complex coronary artery plaques is associated with adverse prognoses in patients with coronary artery disease. Neutrophil count may have also a predictive role in CSA patients in the clinical setting. New techniques such as intravascular ultrasound (IVUS) and MRI, which allow the assessment of the arterial wall and the composition of atheromatous plaques, will be instrumental in the evaluation of total atherosclerosis burden. They will represent a significant step forward in the understanding of plaque vulnerability and should help us move beyond the type of “lumenographic” analysis provided by conventional coronary angiography.

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In response:


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