CORRESPONDENCE

White Blood Count Components as Risk Predictors for Coronary Heart Disease: The Role of Eosinophils

To the Editor:

We read with great interest the paper by Madjid and Fatemi[1] about the components of the complete blood count as indicators of inflammation and risk predictors of coronary heart disease. Although all subtypes of the white blood count have been associated with an increased risk of coronary heart disease, the role of eosinophils has been underestimated. The authors[1] correctly refer to genetic analysis of the inflammatory role of eosinophils, because a single nucleotide polymorphism at 12q24, in *SH2B3*, was found to have a significant association with myocardial infarction (MI).

Eosinophils play an important role in inflammation, and their existence denotes hypersensitivity inflammation. Hypersensitivity inflammation can induce acute MI, and the Kounis hypersensitivity acute coronary syndrome is well described.[2] In patients who had vasospastic angina pectoris, eosinophil counts were significantly increased and could predict the severity of the disease. After these patients underwent medical therapy and attained relief from chest symptoms, their eosinophil counts decreased significantly, to the same level as those of the control group.[3] Clinical reports have shown that the absolute number of eosinophils and the eosinophil-to-leukocyte ratio are significantly elevated in patients who have coronary artery disease.[4] Coronary vasospasm associated with eosinophilia responds poorly to conventional vasodilator therapy, and although the risk of recurrent coronary events is high, most patients respond to therapy that suppresses eosinophilia, such as corticosteroids.[5] Eosinophils are pleiotropic multifunctional leukocytes involved in the initiation and propagation of inflammatory responses, and they express H_4 histamine receptors on their surface. These receptors facilitate eosinophil chemotaxis toward mast cells, which are a source of inflammatory soluble mediators. These mediators can induce the coronary hypersensitivity associated with Kounis syndrome that manifests itself as vasospastic angina, acute MI, or both.[2] Soluble mediators secreted by mast cells and eosinophils also modulate reciprocal interactions between these 2 cells in the so-called "allergic effector unit." Major basic cationic protein released from eosinophils can activate mast cells.[6] This activation elicits exocytosis and new eicosanoid and cytokine production, both of which are prominent responses after FcεRI-dependent activation of mast cells.

Mediators similar to those found in Kounis syndrome are found in cases with nonallergic causes, which suggests that this is a more general problem. A common pathway between allergic and nonallergic coronary syndromes seems to exist.[2]

Eosinophil count has emerged as a novel biomarker of risk stratification in patients who have coronary artery disease. This reality is essential for elucidating the cause of inflammation.

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Discovery of Biomarkers for Chronic Graft-versus-Host Disease

To the Editor:

We read the article by Dogan and colleagues[1] regarding the cardiac effects of chronic graft-versus-host disease (cGVHD) in patients who had undergone allogeneic stem cell transplantation. Chronic GVHD is a severe complication after allogeneic bone marrow transplantation, which is an immunotherapeutic option in several hematologic diseases.

Although the pathogenesis of cGVHD remains unclear, it is solidly linked to graft-versus-leukemia (GVL) reactions; the balance between GVHD and the benefi-