Myocardial infarction as a thrombotic complication of myeloproliferative disorders

Essential thrombocythemia (ET) and polycythemia vera (PV) are myeloproliferative disorders (MPDs), comprising a heterogeneous group of hematological diseases characterized by excessive production of cells belonging to the myeloid lineage in the bone marrow. They are related to, and may evolve into, myelodysplastic syndromes and acute myeloid leukemia, although MPDs as a whole have a much better prognosis than these conditions (1).

MPDs are typically complicated by thrombosis, with a rate of major events as high as 50% (2). Advanced age and a prior history of thrombosis are the two most important risk factors for vascular complications, although hypercholesterolemia, hypertension, smoking, and diabetes have been recognized as incremental factors of thrombosis in patients presenting with MPDs (3). More recently, leukocytosis has been reported as an independent risk factor for thrombosis in both ET and PV (4). Furthermore, the additional value of the JAK2 V617F mutation as a risk factor for thrombosis is currently being investigated (5).

The mechanisms behind the increased thrombotic tendency in MPDs are complex and remain to be elucidated. No correlation has been found between platelet values and the risk of thrombosis (6). Pathophysiological mechanisms possibly involve all cellular blood components, plasma, interaction with endothelial cells, as well as hemodynamic alterations. In MPDs, platelets exhibit the feature of spontaneous aggregation (7), and, in addition, they demonstrate abnormal expression of glycoprotein (8). Leukocytes also contribute to platelet activation in MPDs (9), and erythrocytes show an increased tendency to adhere to the endothelium (10). Interestingly, leukocyte count seems to be a stronger predictor for thrombosis than do platelet counts and hemoglobin/hematocrit levels (9). The hemodynamic changes include increased viscosity, with displacement of circulating platelets toward the endothelium (11). Increased levels of microparticles with procoagulant activity are also found in MPD patients (12). Increased cytokine expression, leading to a general inflammatory response, probably also contributes to the thrombotic tendency (13). An increased number of prothrombotic circulating endothelial cells are seen among these patients; notably, the JAK2 V617F mutation is found in these cells (14).

In these respects, the paper entitled "Myocardial infarction as a thrombotic complication of essential thrombocythemia and polycythaemica vera" by Pósfai et al. (15) published in the current issue of the Anatolian Journal of Cardiology is of potential interest. In this descriptive study, 263 patients diagnosed with either ET or PV were included. In a retrospective analysis, the clinical characteristics of 14 patients who suffered myocardial infarction (MI) during their hematological follow-up were compared to 162 MPD patients who did not exhibit any kind of thrombotic complication. A major result of the study is that most of the MI complications appeared within 1 year after the hematological diagnosis of ET or PV, with evident implications for the necessity of early detection and management in this group of patients. Furthermore, JAK2 V617F mutation was present in most of the cases, and cardiovascular risk factors appeared in the majority of patients. Other interesting results refer to the variables of mean peripheral platelet count, hemoglobin, hematocrit, and red blood cell count.

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