

Does the number of platelets affect outcome in patients with acute myocardial infarction?

Coronary artery atherosclerotic disease (CAD) is the most prevalent disease in industrialized countries. Acute ST-elevation myocardial infarction (STEMI) results from sudden obstruction of a coronary artery causing abrupt interruption of blood flow to the myocardium. Nowadays, urgent primary percutaneous coronary angioplasty (PCI) is the treatment of choice in acute myocardial infarction (STEMI) in order to resume coronary blood flow and myocardial perfusion and thus rescue the still viable myocardium.

How does acute myocardial infarction develop?

Acute myocardial infarction (STEMI) develops as a result of rupture or erosion of the internal layer of the coronary artery covering an atherosclerotic plaque (intima) with superimposed thrombosis.

What is the role of platelets in acute myocardial infarction?

Endothelial erosion or plaque rupture expose platelets in the blood stream to thrombogenic surface and thus a chain of platelet activities begins with adhesion, activation, aggregation, secretion of platelet material and thrombus formation. Thus, Platelets play a very important role in the development of acute myocardial infarction and thrombotic coronary events. Therefore, due to the role of platelets in the development of coronary thrombosis, dual antiplatelet therapy in addition to anticoagulation is a must in patients with acute myocardial infarction.

How to improve antiplatelet treatment?

Because of the role of platelets in acute coronary thrombosis, more powerful antiplatelet drugs have been developed. However, platelet count in the blood is important. Previously, when coronary reperfusion in acute myocardial infarction was achieved by the administration of thrombolytic agent, high platelet count was associated with worse outcome. Therefore the aim of the present study was to test the hypothesis, that in patients with acute STEMI treated by primary coronary angioplasty, platelet count affects coronary artery flow, myocardial perfusion and left ventricular systolic function at admission.

The study included, 174 patients presenting with acute anterior STEMI and treated with primary coronary angioplasty. Patients were divided into subgroups of admission platelet blood count of <200K, 200-300K, 300-400K and >400K. Angiographic evaluation of coronary artery flow and myocardial perfusion (blush) grades were performed according to the TIMI criteria. Myocardial perfusion was also assessed by electrocardiographic ST-elevation resolution after the procedure. Doppler echocardiographic evaluation of left anterior descending coronary artery (LAD) velocities early and late after primary PCI and assessment of left ventricular systolic function were performed. Myocardial biomarker blood levels were also measured.

How did platelet count affect outcome?

After coronary angioplasty, angiographic TIMI, myocardial blush and ST-elevation resolution were similar in all groups. Patients with lower platelet count (<200K) had higher coronary flow as indicated by higher peak diastolic LAD velocity both early and late after angioplasty, and also higher prevalence of LAD velocity deceleration time (DDT) exceeding 600ms, (45.5% vs. 40%, p<0.05) indicating better function of

the coronary microcirculation. Patients with platelets count>400k had larger myocardial injury as indicated by higher maximal biomarker blood levels and had worse left ventricular systolic function.

Thus, patients with anterior STEMI treated by primary PCI with lower admission platelet count had higher LAD diastolic velocities, better myocardial perfusion with more patients having LAD-DDT >600ms. Patients with higher platelet counts had lower left ventricular systolic function both at admission and before discharge. Studies to examine if antiplatelet treatment should be tailored according to platelet blood counts are needed.

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