Antiplatelet therapy in athrothrombosis

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Today atherothrombosis is the most common cause of mortality in developed countries. Platelets play an important role in atherogenesis and the formation of occlusive thrombi. Following the rupture of atheromatous plaque, platelets first adhere to the subendothelial tissue through the interaction of two major platelet membrane glycoproteins, GPIb/V/IX complex and GPVI/FcR γ complex, with collagen.

Subsequently, platelet activation and aggregation occur leading to the occlusion of the diseased arteries with fibrin simultaneously formed, causing myocardial infarction and stroke. One of the important approaches to prevent the atherothrombosis is antiplatelet therapy. Meta-analysis of randomized trials of antiplatelet therapy for prevention of atherothrombosis has clearly shown the significant reduction of serious vascular events. (Antithrombotic Trialists' Collaboration, 2002, Br Med J)

Among various antiplatelet agents, aspirin is widely used with dosages of 75 – 150mg daily. Effectiveness of anti-platelet therapy has been demonstrated not only for secondary prevention of vascular events, but also for primary prevention of acute myocardial infarction and stroke in high risk patients. Compared with aspirin, ADP receptor antagonists, ticlopidine/ clopidogrel, reduced serious vascular events by 10% in patients with coronary heart disease or stroke. Novel ADP receptor antagonists are now being developed and in clinical trials. Among them Prasugrel has been shown its potency over Clopidogrel in high risk patients (TRITON TIMI 38, 2007, New Engl J Med)

Usefulness of antiplatelet therapy should be carefully evaluated based upon risk / benefit since no single antiplatelet agent could avoid bleeding adverse effects.
Future directions for antiplatelet therapy are, therefore, to develop new agents with

potent antithrombotic effects and less bleeding complications.