

Acute Coronary Syndromes

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Following rupture of a vulnerable plaque, its contents are exposed to the passing blood stream. Vulnerable plaques are lipid laden and rich in tissue factor, resulting in activation of the coagulation cascade, ultimately resulting in the deposition of fibrin strands. In addition, platelets are activated and aggregate. Thrombin that is generated as a consequence of activation of the coagulation cascade is a pivotal molecule not only for the formation of fibrin strands but also for activation of platelets. Therefore, there is considerable rationale for ancillary therapy to inhibit both the coagulation cascade and platelet function in patients with acute coronary syndromes.

The clinical use of antithrombotic agents must take into account:

- a) The prescribed dose and dosing interval
- b) The actually administered regimen
- c) The drug concentration at the site of action
- d) The intensity and duration of the pharmacologic effect.

Although unfractionated heparin (UFH) has been used for many decades, it has several limitations. This has inspired an ongoing search for a replacement for UFH. Drug classes evaluated to date include the low molecular weight heparins, direct thrombin inhibitors, and factor Xa inhibitors.

Aspirin is the foundation antiplatelet agent used in the management of ACS. Other classes of antiplatelet agents evaluated thus far include GP IIb/IIIa inhibitors and P2Y₁₂ antagonists. Novel antiplatelet agents being studied include thrombin receptor antagonists.