Antithrombotic Therapy to Reduce Ischemic Events in Acute Coronary Syndromes Patients Undergoing Percutaneous Coronary Intervention

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KEYWORDS

• Percutaneous coronary intervention • Acute coronary syndromes • Oral anticoagulation
• Parenteral anticoagulation • Oral antiplatelet therapy • Parenteral antiplatelet therapy

KEY POINTS

- Because coronary thrombosis plays a pivotal role in the pathogenesis in most patients with acute coronary syndromes (ACS), antithrombotic therapy is essential in the management of ACS in the short and long term.
- Antiplatelet therapy should be started almost immediately after diagnosis and continued for at least 1 year after invasive therapy.
- Parenteral anticoagulation is indicated during coronary intervention for ACS.
- Possibly at discharge, oral anticoagulation may have a role in secondary prevention.
- There are major trials running that are studying the long-term antithrombotic protection of patients who have undergone percutaneous coronary intervention for ACS.

Blood coagulation is pivotal in the pathogenesis of acute vascular disease as shown in a large number of clinical trials on the effectiveness of antiplatelet drugs in vascular disease.[1] Because recently formed thrombi are mainly composed of fibrin and aggregated platelets, vasoactive mediators such as thromboxane A_2 released from platelets may occlude arteries. It has therefore been suggested that antiplatelet drugs like aspirin may be active in the prevention of vascular disease. Indeed, in a retrospective study in patients treated with high-dose aspirin for rheumatoid arthritis, the drug seemed to reduce the incidence of myocardial infarction, angina pectoris, sudden death, and cerebral infarction.[2] In patients presenting with acute coronary syndromes (ACS), revascularization is recommended in most cases,[3–6] of which percutaneous coronary intervention (PCI) by far is the most common. The most frequent complication of PCI is myocardial infarction, which has prognostic consequences.[7] It can be effectively prevented by aspirin.[8] Further reductions can be achieved by the use of platelet P2Y12 blockers.[8,9] The combination of aspirin and platelet P2Y12 blockers, dual antiplatelet therapy (DAPT), has become crucial after stenting had been introduced in the PCI practice. Clopidogrel, prasugrel, and ticagrelor are the available P2Y12 blockers for PCI in ACS.[3–6] The strongest platelet inhibition can be applied by platelet glycoprotein (GP) IIb/IIIa receptor antagonists,[10–14] but their role seems to diminish since the introduction of DAPT.

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In addition to antiplatelet therapy parenteral anticoagulation is important in the fight against thrombotic complications during PCI (Fig. 1, Tables 1–4). Unfractionated heparin is the most often used agent. Less common are low-molecular-weight heparin[15] and the recombinant thrombin blocker bivalirudin.[16,17]

NON-ST ELEVATION ACUTE CORONARY SYNDROME

In non-ST-elevation ACS (NSTE-ACS), coronary plaque rupture followed by thrombotic phenomena usually is the pathophysiologic basis. Therefore, NSTE-ACS patients are at increased risk for ongoing thrombosis and ischemia. To prevent these complications, antiplatelet and anticoagulation therapies are recommended for all patients with NSTE-ACS.[5,6]

As said, the most common complication is myocardial infarction, which can now be better detected by the high-sensitive troponin assays.[7] Embolization of atheromatous and/or thrombotic material in the area of the dilated lesion(s) is thought to be responsible for the infarction. Currently, stenting of the dilated vessels is standard practice, which introduces a second thrombotic risk: acute, subacute, or late stent thrombosis.[18]

Preprocedural Antithrombotic Therapy

With regard to antiplatelet therapy, treatment usually is started at first medical contact irrespective of a decision for an invasive strategy. When the latter is adopted, current guidelines recommend aspirin 200 mg chewed followed by 80 to 100 mg daily and possibly ticagrelor with loading dose of 180 mg followed by 90 mg twice a day (Box 1). There is no consensus as to whether DAPT should be started before coronary angiography.[5,6] Only for high-risk patients, GP IIb/IIIa blockers may be added to this regimen (see Box 1). Diabetes mellitus and ongoing ischemia are features of a high risk. The experience of the combination ticagrelor with GP IIb/IIIa blockers, however, is limited.

In patients in whom coronary surgery is anticipated, P2Y12 blockers may be withheld until coronary angiography. After PCI, prasugrel can then be used instead of ticagrelor.[5,6] A loading dose of 60 mg of prasugrel is necessary followed by 10 mg daily in patients less than the age of

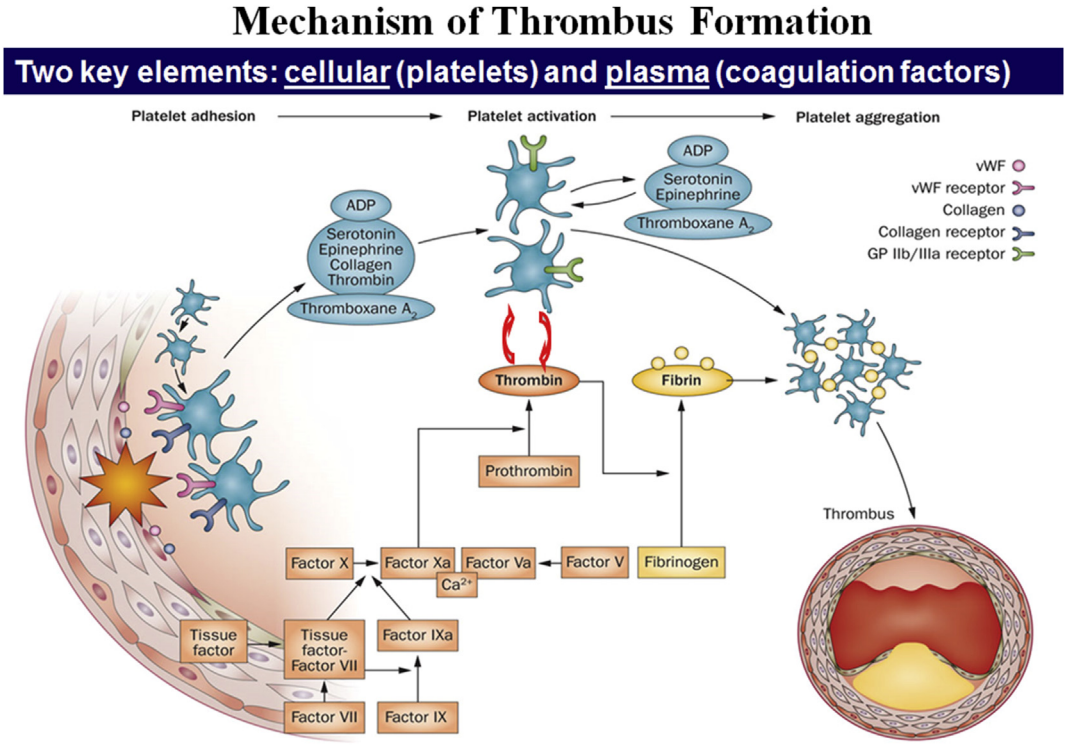

Fig. 1. Mechanisms of coronary thrombosis. ADP, adenosine 5′-diphosphate; vWF, von Willebrand factor. (*From* Franchi F, Angiolillo DJ. Novel antiplatelet agents in acute coronary syndrome. Nat Rev Cardiol 2015;12(1):30–47; with permission.)

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