Thrombocytopenia and Platelet Function Defects

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The major physiologic function of platelets is their roles in primary hemostasis. In addition, platelets also activate and enhance the coagulation cascade. Physiologic abnormalities regarding platelets involve their count and function.

Thrombocytopenia has diverse etiologies. According to its cause and severity, thrombocytopenia is associated with various degrees of bleeding tendency.

Spontaneous, life-threatening bleeding may occur in patients with platelet counts less than 10-20×10⁹/liter. Aggressive management is generally needed in such cases of profound thrombocytopenia. In contrast, most cases with mild to moderate thrombocytopenia in the absence of active bleeding do not require management but efforts should be made to find possible etiologies. Other than thrombocytopenia resulting from insufficient bone marrow production, immune thrombocytopenia (ITP) is the most important cause of thrombocytopenia. The management of ITP has been significantly changed after introduction of thrombopoietin receptor agonists in an era of evidence-based medicine. Steroids and immunoglobulin remain the standard

frontline treatment. Treatment options after steroid failure include thrombopoietin receptor agonists, rituximab, cyclosporine, splenectomy, etc. The optimal treatment should probably be individualized at the discretion of physicians and patients. Both congenital and acquired disorders may affect platelet functions. The classical congenital platelet function disorders are Glanzmann's thrombasthenia and Bernard-Soulier syndrome. Many acquired diseases, such as renal failure, hepatic dysfunction are associated with platelet function impairment. The most common cause of platelet dysfunction, however, is iatrogenic. Antiplatelet drugs were used extensively in treatment of cardiovascular disease and stroke. Currently available antiplatelet agents include aspirin, clopidogrel, prasugrel and glycoprotein IIb/IIIa inhibitors (abciximab, tirofiban, eptifibatide). Bleeding tendency associated with these drugs is generally mild but severe hemorrhagic events may occur, especially in the presence of co-morbidity (e.g. peptic ulcers). In recent years, tremendous efforts have been made to monitor the antiplatelet efficacy with laboratory techniques. Hopefully, antiplatelet treatment can be tailor-made to reach the maximal benefit while minimizing treatment-associated bleeding risk.