HEPARIN INDUCED THROMBOCYTOPENIA

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Heparin-induced thrombocytopenia (HIT) is a rare but severe prothrombotic adverse effect of heparin treatment, and remains the most frequent immune mediated adverse drug reaction involving blood cells.

The underlying cause is formation of highly immunogenic complexes between negatively charged heparin and positively charged platelet factor 4 (PF4). Resulting antibodies against PF4/heparin complexes can activate platelets via the platelet Fcylla receptor, leading to thrombin generation and thus to the paradox of a prothrombotic state despite thrombocytopenia and application of heparin.

The most important clinical feature of HIT is a decrease in platelet counts by more than 50% combined with a procoagulatory status that may cause venous and – less frequently – arterial thrombosis.

Prompt diagnosis of HIT is important in order to change treatment to prevent severe thromboembolic complications. However, this is often difficult, especially in intensive care patients in whom thrombocytopenia is frequent. The laboratory tests for HIT-antibodies have a high negative predictive value but only a poor positive predictive value. This leads to overdiagnosis and overtreatment of HIT, which also bear the risk for adverse outcomes.

There are two fundamentally different forms of thrombocytopenia induced by heparin: non-immune-mediated heparin-associated thrombocytopenia (HIT) Type I, which is of minor clinical relevance, and immune-mediated HIT, or HIT Type II. Treatment principles of HIT include: discontinuation of heparin; initiation of an alternative non-heparin anticoagulant; performing imaging studies for lower-limb deep vein thrombosis; postponing/avoiding vitamin K antagonists: minimizing platelet transfusions; and avoiding inferior vena cava filters.

Two different approaches to alternative anticoagulation are available: indirect antithrombin (AT)-dependent inhibitors of factor Xa (danaparoid, fondaparinux); and direct thrombin inhibitors (r-hirudin, argatroban, bivalirudin).