

Heparin Induced Thrombocytopenia

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Heparin is the most commonly administered parenteral medication during hospitalizations. Heparin-induced thrombocytopenia (HIT) is an immune mediated adverse drug reaction that occurs following exposure to unfractionated heparin (UFH) or low molecular weight heparin (LMWH) (1). Binding of antibodies to a complex of heparin and platelet factor 4 (PF4) produces platelet activation and aggregation, leading to thrombus formation in either the venous or arterial system (2,3). The spectrum of presentation ranges from no obvious clinical manifestations to life-threatening heparin induced thrombocytopenia with thrombosis (HITT), manifested by pulmonary emboli or arterial clots in the renal, cerebral, or coronary circulation (4).

HIT remains an important clinical problem with a high mortality rate, regardless of thrombosis development. Upon the diagnosis of HIT, there should be a rapid discontinuation of heparin in all forms (including low-molecular weight heparin and flushes). Followed by initiation of an agent that will not exacerbate thrombosis (direct thrombin inhibitors, fondaparinux), with a transition to extended duration anticoagulation with warfarin when indicated.

Prevention of HIT altogether should be our goal. Both the use of alternative anticoagulation and the failure to diagnose and treat HIT, pose their respective risks. It is imperative that clinicians have a sound understanding of the identification and management of HIT. Additionally, a patient's understanding of what HIT entails and proactive communication to other practitioners may avoid a potential catastrophe by re-administering a heparin product (5).

Standardization of care through the implementation of algorithms, guidelines and computerized alerts, can facilitate safe and effective care of patients with HIT. Proper documentation of HIT and education of patients and their caretakers are important steps to improve communication and achieve consistency of care.

References:

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