

Heparin Induced Thrombotic Thrombocytopenia ± Thrombosis.

D. ROYSTON

Heparin-induced thrombocytopenia (HIT) is a serious, yet treatable, prothrombotic disease that develops in approximately 0.5% to 5% of heparin-treated patients and dramatically increases their risk of thrombosis (1). The antibodies that mediate HIT, i.e., heparin-platelet factor 4 antibodies, occur more frequently than the overt disease itself and, even in the absence of thrombocytopenia, are associated with increased morbidity and length of hospital stay after cardiac (2) and orthopaedic surgery (3).

The diagnosis of HIT should be suspected whenever the platelet count drops > 50% from baseline (or to < 150 × 10⁹/L) beginning 5 to 14 days after starting heparin (or sooner if there was prior heparin exposure) and/or new thrombosis occurs during, or soon after, heparin treatment, with other causes excluded (1). This is the basis of the 4T's system of diagnosis (4).

Laboratory tests for HIT are slow and relatively imprecise or inaccurate. Interventions in strongly suspected HIT should not wait for laboratory confirmation.

When HIT is strongly suspected, with or without complicating thrombosis, heparin should be discontinued and a non-heparin alternative anticoagulant initiated.

Selection of the appropriate non-heparin anticoagulant depends upon availability, clinician experience and preference, concomitant liver or

renal disease, and planned invasive procedures. Three drugs are approved for anticoagulation in HIT (listed in order of approval): danaparoid (heparinoid), lepirudin (bivalent direct thrombin inhibitor [DTI]), and argatroban (monovalent DTI). Fondaparinux and bivalirudin are other non-approved anticoagulants with minimal but favourable experience.

Two clinical scenarios will be presented and the use of the various available agents in these settings discussed.

References

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