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# Commentary: Getting HIT with HIT minus T (thrombosis without thrombocytopenia)

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**CENTRAL MESSAGE**

The name heparin-induced thrombocytopenia (HIT) suggests obligatory thrombocytopenia, yet HIT may still occur without thrombocytopenia, a diagnostic trap for the unwary clinician.

Heparin-induced thrombocytopenia (HIT) is primarily a prothrombotic disorder, the end result of heparin-platelet factor 4 antibody complexes causing platelet activation with the release of procoagulant platelet microparticles, increased thrombin generation, platelet consumption, and thrombocytopenia.[1,2] Paradoxically, thrombosis is a far more prevalent manifestation of HIT than bleeding.

Heparin-induced thrombocytopenia suggests the obligatory association of absolute or relative thrombocytopenia, yet, as shown by Catalano and coworkers,[3] HIT may still be present without thrombocytopenia. In 5 of 6 patients readmitted for thromboembolism after cardiopulmonary bypass, readmission platelet counts were not significantly different from the preoperative values. Because thrombocytopenia was not obvious on readmission, HIT was not suspected and these patients were inappropriately reexposed to heparin. After all, how do you convince anyone that a patient has HIT without the T? In the case of these patients, the term HIT appears to be a misnomer. Notably, thrombocytopenia occurs in ~95% of HIT patients sometime during the course of illness,[4] and other workers have reported cases in which heparin-induced thrombosis was confirmed in the absence of thrombocytopenia.[5-10]

HIT typically occurs within 5-10 days of heparin exposure,[11] a period that coincides with reactionary thrombocytosis in the postoperative period. This means that in a postoperative patient experiencing HIT, activation and depletion of preformed platelets leading to a prothrombotic state and a tendency toward thrombocytopenia could occur in synchrony with reactive postoperative thrombocytosis that masks the thrombocytopenia on laboratory testing. This allows dissociation between the onsets of thrombosis and thrombocytopenia so that thrombotic complications may be present without the hallmark thrombocytopenia for which HIT is (mis-) named. Warkentin and Kelton[6] reported that 12 patients with HIT that they investigated, thrombocytopenia began an average of 9.2 days (range, 5-19 days) after the last use of unfractionated heparin; 6 of these patients manifested thrombotic complications beginning at an average of 8.3 days (range, 5-14 days) after heparin was withdrawn.

Without the awareness that heparin-induced thrombosis may occur without thrombocytopenia, patients with "HIT minus T" are at risk for heparin reexposure, which can result in a 33% mortality rate.[12] In the 12 patients reported by Warkentin and Kelton,[6] 9 (including 3 without thrombocytopenia) were retreated with heparin, resulting in further decreases in platelet counts in all 9 patients and cardiac arrest in 1 patient.

The present study is retrospective and limited by a small sample size, but it emphasizes 2 very important points. The first point is that cardiac surgery patients may be discharged to home just at the time when HIT starts, and the platelet count may fail to reveal this. The second point is that the patient may be readmitted with thromboembolism, and the platelet count may fail to reveal this as well. The learning point is appreciating that the beguiling factor in the setting of postoperative

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Disclosures: The authors reported no conflicts of interest.

The *Journal* policy requires editors and reviewers to disclose conflicts of interest and to decline handling or reviewing manuscripts for which they may have a conflict of interest. The editors and reviewers of this article have no conflicts of interest.

Received for publication Aug 16, 2020; revisions received Aug 16, 2020; accepted for publication Aug 21, 2020; available ahead of print Sept 17, 2020.

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JTCVS Open 2020;4:43-4
2666-2736



https://doi.org/10.1016/j.xjon.2020.08.016

thromboembolism after cardiac surgery is the absence of thrombocytopenia. Undue reliance on the platelet count as the hallmark of HIT risks getting hit with "HIT minus T."

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