

November 29, 2005

DELAYED HIT AND THEIR ANTIBODIES

Recent publications (1,2) demonstrated that HIT antibodies can sometimes cause platelet activation in a Serotonin Release Assay and possibly in the related Heparin Induced Platelet Aggregation assay in the absence of added heparin. This phenomenon is not related to the presence of heparin in the serum sample. Therefore the HIT antibodies can interact with platelets when there is no heparin “in vivo”. This may lend significant importance to the finding of Delayed HIT in patients who have received Heparin weeks to months earlier. There also seems to be a significant increase in the titer of antibody in the PF4/Heparin Elisa assays for patients that have Delayed HIT thrombosis, which may represent a clinical edge to establish the diagnosis since Delayed HIT is not always associated with thrombocytopenia.

Although the paper shows that no heparin could be detected in these samples by either Anti-Xa assays or by using heparinase-1 to digest any heparin, some previous reports have shown that some positive platelet aggregation assays (in the absence of added heparin) can be reversed by heparinase treatment. It was also noted that the antibody (IgG) responsible for the positive platelet aggregation was heterogeneous in that some gave strong positive SRA reactions but not PF4/Heparin Elisa's and vice versa.

1. PRECHEL, M.M., McDONALD, M.K., JESKE, W.P., MESSMORE, H.L., AND WALENGA, J.M.. ACTIVATION OF PLATELETS BY HEPARIN-INDUCED THROMBOCYTOPENIA ANTIBODIES IN THE SEROTONIN RELEASE ASSAY IS NOT DEPENDENT ON THE PRESENCE OF HEPARIN. J. THROMBOSIS AND HAEMOSTASIS 3,2168-2175, 2005.

2. ASTER, R.H.. HEPARIN-INDEPENDENT ACTIVATION OF PLATELETS BY HIT ANTIBODIES: A CLUE TO THE ETIOLOGY OF DELAYED THROMBOCYTOPENIA/THROMBOSIS IN PATIENTS GIVEN HEPARIN? J. THROMBOSIS AND HAEMOSTASIS 3,2166-2167, 2005.