

Emostasi, infiammazione e immunità

HEPARIN-INDUCED THROMBOCYTOPENIA IN A PATIENT WITH GUILLAIN-BARRÉ SYNDROME: A POSSIBLE LINK WITH IMMUNE DYSREGULATION

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Background. Heparin-induced thrombocytopenia (HIT) is a potentially life-threatening immune-mediated adverse reaction caused by antibodies against platelet factor 4 (PF4)-heparin complexes, leading to platelet activation and an increased risk of thrombosis. Conditions associated with systemic immune activation may facilitate antibody formation through immune priming. A retrospective study (2018) reported that patients with HIT were significantly more likely to have a concomitant chronic autoimmune disease compared with those without HIT, suggesting a potential association between immune dysregulation and the development of HIT. Guillain-Barré syndrome (GBS) is an acute immune-mediated neuropathy, typically triggered by infections and characterized by dysregulated immune responses. Although rare, it represents a neurological emergency as a potentially fatal condition.

Case Report. We report an 87-year-old female with post-infectious GBS, occurring 10–14 days after gastroenteritis, with anti-GM1 IgG and anti-GD1a antibody positivity, treated with plasma exchange with good neurological response. According to the Padua Prediction Score, pharmacological thromboprophylaxis was indicated. Low-molecular-weight heparin (enoxaparin 4,000 IU subcutaneously once daily) was administered for five days and discontinued due to an isolated haemoglobin decrease to 8 g/dL (−2 g/dL from baseline), without thrombocytopenia. Anaemia was attributed to ongoing plasma exchange and resolved after completion of treatment and transfusion support. Following treatment completion, a femoral peripherally inserted central catheter (PICC)-related deep vein thrombosis (DVT) was diagnosed and therapeutic-dose enoxaparin was initiated. Platelet counts remained stable (>150×10^9/L), but within 24 hours of therapeutic heparin re-exposure, platelet count dropped to 85×10^9/L (>50% reduction), reaching a nadir of 30×10^9/L over subsequent days. The 4T score indicated high probability for HIT (score 6), prompting immediate discontinuation of heparin and initiation of fondaparinux at therapeutic dose. Anti-PF4/heparin IgG antibodies by ELISA were strongly positive, and a functional platelet activation assay confirmed heparin-dependent platelet activation, consistent with definite HIT. Anticoagulation with fondaparinux was continued. During the acute phase, a further catheter-related upper limb DVT (midline, left arm) occurred. Concomitantly, neurological worsening with suspected cortical hypodensity on brain CT was observed, although its thrombotic nature remained uncertain in the context of GBS.

Conclusions. This case highlights a temporal association between GBS-related immune activation and HIT onset, supporting the hypothesis that immune dysregulation, including acute conditions, may lower the threshold for anti-PF4 antibody development. Plasma exchange may represent a potential confounding factor in platelet count interpretation and, in this case, did not prevent the occurrence of high-titre antibodies, possibly due to ongoing immune activation. These findings highlight the need for increased clinical vigilance and suggest that acute autoimmune states may represent an underrecognized risk context for HIT. Further studies and additional case reports are needed to better define this potential association.

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