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Autoimmune heparin-induced thrombocytopenia: A case report

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Thrombocytopenia affects 30-50% critically ill patients. Heparin-induced thrombocytopenia (HIT) is rare (incidence 0.3-0.5%), but crucial to distinguish to treat the paradoxical prothrombotic state. Autoimmune HIT may follow an atypical clinical course. An 86 year old female sustained a tibia/fibula fracture. Enoxaparin 40 mg/day was commenced following intramedullary nailing. On D4 she developed pneumonia, requiring intravenous antibiotics and renal replacement therapy with intravenous unfractionated heparin (UFH) 20000 units per day. The platelet count fell to 25×109/l (D15) and platelet transfusion was required for vascath insertion. The clinical probability of HIT was intermediate ("4 Ts score" 5/8) and heparin stopped. Rapid particle gel immunoassay (PF4/H-PaGIA) and ELISA (result 1.7 OD; 80% correction with high-dose heparin) were positive for HIT and danaparoid commenced. Subsequently, the "4 Ts" was revised to 3/8 when platelet count nadired (17×109/l) and vancomycin-induced thrombocytopenia considered. There were no bleeding or thrombotic complications. Thrombocytopenia persisted despite cessation of heparin and vancomycin. On D24 intravenous immunoglobulin (IVIG) was administered with rapid recovery of platelet count. Later, the heparin-induced platelet aggregation (HIPA) functional assay confirmed HIT. The patient serum activated donor platelets with low dose heparin; but also activated platelets in the absence of heparin, indicating autoimmune HIT. In HIT, heparin/PF4/IgG complexes recognize platelet Fc receptors and activate platelets. In autoimmune HIT, PF4/poly anion complexes bind PF4/chondroitin sulfate complexes on platelets with high affinity, clustering PF4 molecules and activating platelets by a heparin-independent mechanism. Therefore, the timing of thrombocytopenia in relation to heparin may be atypical. Other rare cases have responded rapidly to IVIG.

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Quality in laboratory testing for hemophilia patients

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The hemostasis laboratory plays an integral role in the care of hemophilia patients. From initial diagnosis, to monitoring therapeutic efficacy, to detecting the development of inhibitors during the course of therapy, the laboratory is involved. Therefore it is essential that the results generated by the hemostasis laboratory are reliable. While technical skill and expertise are paramount in performing hemostasis testing, the cornerstone of ensuring test accuracy and precision is having a comprehensive quality program. The important elements of the quality program will be described with demonstrations of how these elements impact the care of hemophilia patients. The pre-analytical phase of testing which includes sample collection, handling, transportation and preparation is a major source of error in the testing process. How these pre-analytical factors can impact test results as well as strategies for identifying and eliminating pre-analytical errors will be discussed using a case-based approach. Important in ensuring the quality of the testing that is performed once the sample reaches the hemostasis laboratory are internal quality control (IQC) and external quality assessment (EQA), to ensure that results are safe to use for patient management and provide information on the accuracy of results that are provided by the laboratory. The use of IQC and EQA in the hemostasis laboratory will be described and the value of their incorporation will be demonstrated through a case-based approach. Finally, measures for improving the overall quality of testing in the hemostasis laboratory will be discussed.

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