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How to improve the management of a patient with heparin-induced thrombocytopenia?

To the Editor,

Heparin-induced thrombocytopenia (HIT) is an adverse drug reaction caused by immunoglobulin G platelet-activating antibodies against platelet factor 4 (PF4)/heparin complexes, leading to venous and arterial thromboembolism (1). I read with keen interest the case report describing a fatal case of probable HIT in a young man who experienced pulmonary embolism (PE) and concomitant deep vein thrombosis (2). The case of this patient with intracardiac thrombus formation and severe ischemic stroke highlights the high risk of thromboembolic events in patients with HIT despite anticoagulant treatment with fondaparinux (5 mg/d), which was ineffective in case of the patient even when the platelet count increased to 150.000/uL. However, without any description of the patient's weight, it remains unclear whether the dosage of fondaparinux was appropriate. The use of vitamin K antagonist (VKA) after normalization of the platelet count, along with the administration of low-molecular-weight heparins or non-VKA oral anticoagulants immediately after the diagnosis of PE in a hemodynamically stable patient could lower the risk of HIT development and significantly improve the prognosis (1, 3). The rationale for choosing unfractionated heparin (UFH), the most common cause of HIT, in the patient was not presented.

In 2018, we had reported our experience with the diagnosis and management of patients suspected of having HIT (4). We have also observed a male patient with PE who was heterozygous for factor V Leiden, as in the present case; was receiving UFH; and was found to have intracardiac thrombi at diagnosis; however, fondaparinux (7.5 mg/d) was effective in that patient (Undas unpublished data). A major limitation of this report is the lack of laboratory confirmation of HIT. On the basis of our experience, we consider that the most commonly used anti-PF4/heparin antibody enzyme immunoassays can frequently detect clinically irrelevant antibodies, with a risk of overdiagnosis. However, a high OD value above 2 well correlates with the positive results of specific assays, e.g., a platelet serotonin-release assay, and such assays can be used in low-income countries such as Poland and Turkey (1, 4). Considering other strong prothrombotic factors, authors did not present conclusive evidence for the absence of occult cancer, which might contribute to the resistance to the anticoagulant used (5). Autopsy could clarify such uncertainties. This interesting report supports not using UFH as a first-line therapy in most PE patients and treating PE vigourously to prevent life-threatening thrombotc events, including HIT, especially in young patients without serious comorbidities.

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Author`s Reply

To the Editor,

We would like to thank the authors for their interest in our article titled "Mitral valve and right ventricular thrombi possibly caused by heparin-induced thrombocytopenia" and for taking