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Heparin-induced Thrombocytopenia

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ABSTRACT

Heparin products are commonly used in the hospitalized patient for prophylaxis or treatment of thromboembolic disease. One adverse effect of using heparin products with potential devastating consequences is heparin-induced thrombocytopenia (HIT). It is crucial the health care providers, including nurse practitioners (NPs), consider HIT in the differential diagnosis for this patient population when they present with thrombocytopenia. This article provides NPs with the latest evidence-based guidelines for diagnosing and managing the patient with HIT.

Keywords: diagnosis, heparin-induced thrombocytopenia, heparin products, management, thrombocytopenia

Heparin-induced thrombocytopenia (HIT) is a complication that can occur between 5 and 10 days after starting heparin, one of the most widely used therapeutic agents in the hospitalized patient.[1] The risk of life-threatening thrombotic events can occur in up to 75% of patients diagnosed with HIT, which makes prompt diagnosis and management of HIT critical.[2,3]

The purpose of this article is to provide nurse practitioners (NPs) with the latest evidence-based guidelines for diagnosing and managing the patient with HIT through discussion of a comprehensive review of the literature. The discussion includes a review of the pathogenesis of HIT, as well as the clinical and diagnostic tools used to confirm diagnosis and the pharmacologic treatment recommendations to manage the patient with HIT.

Once HIT is suspected, it is important to evaluate both clinical and laboratory data to confirm the diagnosis.[3] Stopping all sources of heparin and selecting and initiating an alternative anticoagulation treatment is the primary focus of managing the patient with HIT.[1,2,4,5]

THE CLINICAL PROBLEM

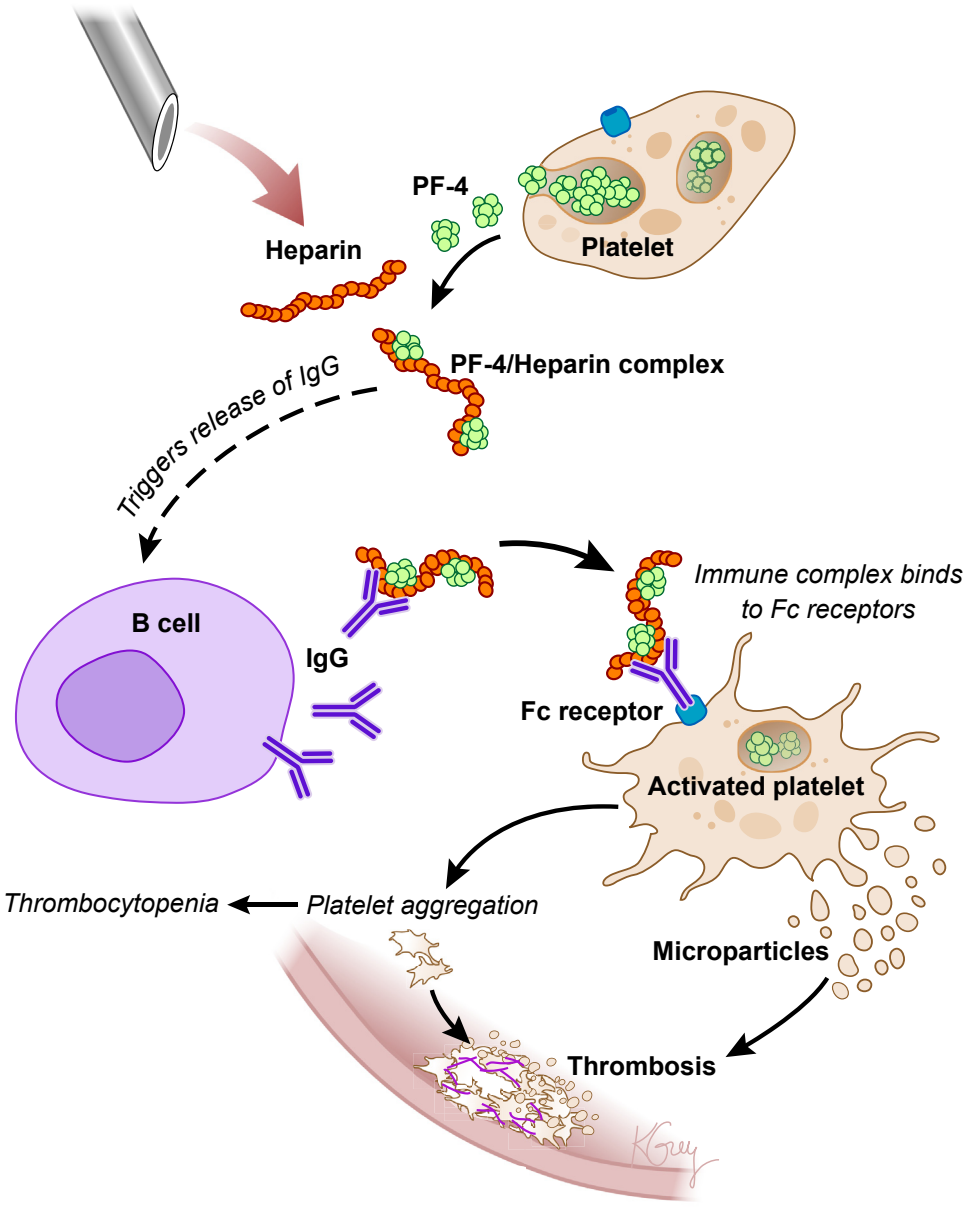
There are 2 types of HIT that can occur when the patient receiving heparin therapy is thrombocytopenic. HIT type I is characterized by transient thrombocytopenia, usually occurring within 2 days of heparin administration, that resolves spontaneously.[2,4] HIT type II is an immune process and should be suspected when platelets drop by 50% from baseline and/or a patient develops a new thrombosis while on heparin.[4] HIT type II, now known simply as HIT, is the clinical syndrome discussed in this article.

The reported risk of HIT for patients receiving heparin is 0.5% to 5%.[6] Any type of heparin exposure, including heparin flushes and heparin-bonded devices, can cause HIT, although the administration of unfractionated heparin carries a higher risk than low-molecular-weight heparin.[2,6,7] Women have a higher risk of developing HIT than men.[6,7] Researchers have described several genetic polymorphisms that might explain the sex difference, but larger studies are needed to define the clinical significance.[8] Cardiac and orthopedic surgical patients have the highest incidence of HIT, with the lowest incidence being with medical and obstetric patients.[2,7,8] Although the reasons for the risk differences among these patient populations are not fully known, differences in basal levels of platelet activation and circulating platelet factor 4 (PF4) levels may be the contributing cause.[8]

PATHOGENESIS OF HIT

HIT is a pathologic immune-mediated reaction to heparin therapy[1,2,4,5] as shown in the Figure.[1,2,5] PF4 is a heparin neutralizing protein present within platelets that bind to heparin. The release of

Figure. Pathogenesis of HIT. The PF4 protein released by platelets attaches to heparin, forming PF4/heparin complexes. This complex triggers the release of the IgG antibody from beta cells, which attaches to the PF4 /heparin complex. This immune complex activates platelets and the release of platelet microparticles that initiate thrombotic activity.



immunoglobulin (Ig) G antibodies is triggered by the formation of the PF4 /heparin complex. Subsequently, the binding of IgG to the PF4/ heparin complex activates platelets and the release of platelet fragments through the platelet Fc receptor. The platelet fragments shown as microparticles in the Figure are procoagulant rich and initiate the thrombotic activity that occurs in HIT. At the same time, thrombocytopenia develops because of platelet activation