INTRODUCTION

Heparin is one of the most frequently prescribed medications, with more than 12million patients receiving heparin annually in the United States for thromboprophylaxis or treatment in variety of clinical settings ***(Jang and Hursting, 2005).***

Heparin therapy can result in two types of thrombocytopenia. One is a mild, transient, non_immune disorder and generally has no clinical consequence. The other, known as heparin induced thrombocytopenia, is the potentially serious and immunoglobulin mediated type that carries a risk of thromboembolic events ***(Chong B.H, 1995).***

Because thrombocytopenia is common in hospitalized patients, occurring in up to 58% of critically ill patients, and can be caused by variety of factors. HIT unfortunately often remains unrecognized. However, consistent with standard clinical practice for life threatening condition, HIT should be suspected in a heparin treated patients who has thrombocytopenia with or without thrombosis. Increased awareness and a high suspicion for HIT are critical to ensure its prompt recognition, diagnosis, and treatment ***(Jang and Hurstig, 2005).***

Because of the drop in platelet count is a primary way of recognizing HIT, routine monitoring of platelet count is recommended for most patients receiving heparin therapy. A baseline platelet count before initiating heparin treatment is important to allow estimation of relative changes. In higher risk patients, such as individuals receiving unfractionated heparin at therapeutic doses, the platelet count should be

checked at least every other day until day 14 of therapy. In lower risk patients, monitoring should be at least every 2 or 3 days between days 4 and 14 while on heparin therapy ***(Warkentin and Greinacher 2004).***

Although the prevalence of HIT has decreased with the use of low molecular weight heparin in the past ten years; HIT remains a life threatening prothrombotic state, HIT can be complicated by thrombosis even after withdrawal of heparin, explaining why substituting heparin with an alternative anticoagulant (danaproid, lepirudin. argatroban) is always necessary. However, management by these alternative treatments is difficult ***(Emmanuel and Gruel, 2006)***.

More recently, a number of agents which can act to directly inhibit thrombin have been licensed for use in human, it is expected that the use of these compounds will eventually grow to replace the use of heparin and vitamin k antagonists within the next few years**.** ***(Walker and Royston, 2002).***