



Heparin Induced Thrombocytopenia Management in Burns



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Introduction

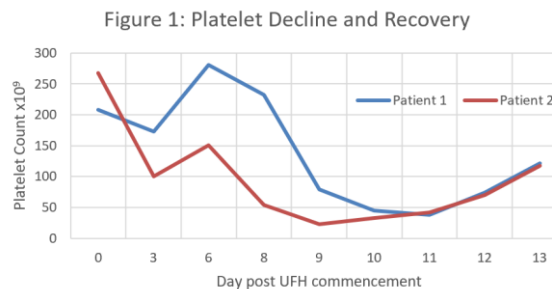
Heparin induced thrombocytopenia (HIT) is a severe complication of unfractionated heparin (UFH) and low molecular weight heparin (LMWH) therapy. An autoimmune disorder mediated by heparin-dependent IgG antibodies, it has the potential for widespread arterial or venous thrombotic complications. The aim of this study was to review cases of HITs in a severe burns centre highlighting outcomes and HITs management in the setting of burns.

Case Series

This is a case series of two patients who were managed for HITs whilst admitted with severe burns at Royal North Shore Hospital between 2008 and 2018.

Patient one was a 34 year old male who presented with 43% TBSA following a gas bottle explosion at home. His ICU admission was complicated by deep vein thrombosis (DVT) requiring UFH anticoagulation via infusion. On day 11 post heparin infusion it was noted the patient's platelet count had rapidly fallen (see Figure 1). UFH was ceased and the patient's anticoagulation changed to danaparoid then rivaroxaban. His platelet count improved and there were no bleeding, thrombosis, graft or vascular complications as a result of his thrombocytopenia.

The second patient is a 54 year old male who presented with 45% total burn surface area post gas bottle explosion. HITs was diagnosed via enzyme-linked immunosorbent assay (ELISA) day 8 during his admission whilst on UFH for DVT prophylaxis. DVT prophylaxis was changed to argatroban followed by apixaban for three months. The patient experienced nil complications as a result of HITs and the platelet count improved with cessation of the UFH (see Figure 1)



Discussion

Burns patients are at high risk of thrombosis [1], hence the need for VTE prophylaxis. HITs is a severe complication secondary to UFH and at a lesser rate LMWH mediated by platelet activated antibodies resulting in a hypercoagulable state, potential venous or arterial complications and thrombocytopenia [2].

Previous published studies on HITs in burns patients have reported an incidence of 0.6-1.6% in patients receiving both UFH and LMWH [3,4]. Fortunately, the presentation is rare but the diagnosis can be difficult in the burn patient. Thrombocytopenia in the acute phase post burn is not unexpected secondary to consumption by damaged tissue and dilution secondary to fluid resuscitation (as seen in Figure 1 over the first three days). Therefore, the treating team must be vigilant and utilise the appropriate investigations when HITs is suspected [2].

Patients suspected or proven HITs require cessation of UFH or LMWH and commencement on anticoagulation. Aprotinin is favoured for patients with acute thrombosis or the potential need for urgent intervention as it is a monitored infusion with a short half-life (39-51mins). Fondaparinux is a fixed dose anticoagulation better suited for stable patients. Patients should be anticoagulated for a minimum of 3 months [2].

Conclusion

HITs is difficult to diagnose and a potentially devastating complication of UFH and LMWH therapy [3]. The treating burns teams needs to monitor the patient's platelet count closely when utilising heparin. Specialist referral is recommended as there are a number of options for the management of acute HITs [2].

References

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