

## Heparin Induced Thrombocytopenia leading to cardiorespiratory arrest during haemodialysis - lessons from an uncommon occurrence and a review of the literature

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### Introduction:

Heparin Induced thrombocytopenia (HIT) is a life-threatening disorder occurring in 1-5% of patients exposed to heparin. It is classified into two types. Type 2 HIT is antibody mediated and occurs 5-10 days post heparin initiation. It causes a significant reduction in platelets causing thromboembolic and haemorrhagic sequelae. Cessation of heparin is required. Type 1 HIT causes mild reduction in platelets and cessation of heparin is not needed. Heparin is the most commonly used anticoagulant in Haemodialysis (HD) patients to prevent extracorporeal circuit (EC) clotting and as catheter locks. Although various complications are well recognised, cardio respiratory arrest during HD is an extremely rare complication of type 2 HIT, with a few cases in medical literature. Here in, we discuss such a case with a review of reported cases, outline a mechanism and highlight important lessons.

**Case:** A 88-year-old gentleman was switched from peritoneal dialysis to HD due to recurrent peritonitis. He had a background of hypertension, ischaemic heart disease and an implanted pacemaker for complete heart block. He commenced HD on an internal jugular tunnelled catheter. He started receiving regular Dalteparin 2500 Units on his 3rd session, after uneventful first two sessions. His fourth session was complicated by an arterial pressure of more than 200. On his 5th session, he went into cardio respiratory arrest 5 minutes after starting HD and receiving Dalteparin. After 5 minutes of resuscitation he regained cardiac output and had normal observations. His blood investigations revealed a platelet count of  $37 \times 10^9/L$  which was  $166 \times 10^9/L$ , 10 days ago. His 6th session was complicated by EC clotting despite saline flushes. As the platelet count had improved to  $74 \times 10^9/L$ , Dalteparin 5000 Units was given. Within a few minutes he had a cardio respiratory arrest, followed by spontaneous recovery on stopping HD. Platelet count checked the next day was  $34 \times 10^9/L$ . A HIT screen and mast cell tryptase were sent; the former came back strongly positive. He was taken off heparin followed by a rise in his platelet count.

### Discussion:

HIT being a pro thrombotic condition, can cause vascular access dysfunction and EC clotting which was evident in our case. Transient occlusion of blood vessels in the pulmonary and coronary vasculature is also a possibility leading to cardio respiratory compromise and even arrest. Table 1 details all past reported cases, the type of heparin implicated and adverse clinical event. Generally, past cases, describe HIT occurring 8 – 21 days after heparin exposure. Our patient presented a week after heparin exposure. Timely recognition by maintaining a high degree of suspicion and checking the platelet count in new HD patients with access dysfunction or EC clotting is pivotal. The most common response in these scenarios is to increase heparin dose, which only worsens the prothrombotic state as evident in our case. The treatment is to stop heparin and switch to non-heparin-based anti coagulation. The key differential is anaphylaxis, which is differentiated with a normal mast cell tryptase level and lack of suggestive clinical features