EComment: Plasmapheresis for near-fatal heparin-induced thrombocytopenia

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We appreciate reading the interesting case report by Dr Kramer and coworkers regarding the use of plasmapheresis in near-fatal heparin-induced thrombocytopenia [1]. Interestingly, the patient developed thrombocytopenia within the first 24 h of unfractioned porcine-derived heparin following myocardial infarction and pump failure necessitating intra-aortic balloon pump (IABP), coronary artery bypass grafting (CABG), and later extracorporeal membrane oxygenator (ECMO) and HeartMate support. ELISA testing seeking for antibodies to H-PF4 were negative, however thrombocytopenia was evident.

In plastic reconstructive surgery, heparin-induced thrombocytopenia (HIT) is an issue, too [2]. We encountered a 26-year-old male patient who developed toxic epidermal necrolysis (TEN) with painful blister formation affecting 70% of the total body surface area following treatment with carbamazepin for bipolar disorder. Further progression of TEN was observed the following day with symptoms, including severe involvement of the gastrointestinal tract, respiratory tract and eyes. As a result, the patient was intubated and systemic anticoagulation with unfractionated heparin was initiated with subsequent transfer to our burn center. On day 13, the patient complained about pain in his right leg. Physical examination revealed a cold right leg, and absent popliteal and foot pulses with Doppler ultrasound. Systemic heparin treatment was immediately discontinued and systemic anticoagulation with agatroban started. The screening test for HIT II antibodies was positive and further confirmed by a positive heparin-induced platelet aggregation test (HIPA test). CT angiography was performed, demonstrating a right-sided occlusion of the iliacal and popliteal artery. An emergency arterial thrombectomy under agatroban medication was performed and perfusion restored. The following night, perfusion in the right leg ceased again and a second thrombectomy of the popliteal artery with prophylactic fasciotomy of the right leg was performed. At this time, the patient had to be ventilated with catecholamine administration in increasing doses. Due to renal failure, dialysis was started the next day. A transthoracic echocardiogram revealed diminished left ventricular ejection fractions and signs of acute right-sided heart failure, consistent with an acute massive pulmonary embolism. The young patient deteriorated dramatically, resulting in immediate irreversible cardiac failure and an unsuccessful cardiopulmonary resuscitation.

Given the aforementioned observation a plasmapheresis should be at least considered in patients suffering severe near-fatal HIT.

References
