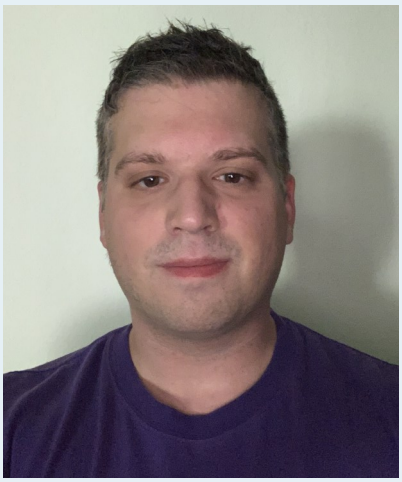
# SUSPECTED HEPARIN INDUCED THROMBOCYTOPENIA AND ECMO PATIENTS, CONSIDERATION OF CVVH FILTER

**Warren Anderson**

Milwaukee School of Engineering

Master's Program of Cardiovascular Perfusion

Milwaukee, WI



Platelet counts drop expectedly following cardiac surgery and cardiopulmonary bypass. More complex and critically ill patients may require extracorporeal membrane oxygenation (ECMO) from failure to wean from the cardiotomy or from some sort of respiratory condition unrelated to a postoperative period. The stress due to the patient's condition and comorbidities may also lead to the acute renal failure requiring use of continuous veno-venous hemofiltration (CVVH). Considering the patient is being supported by both an ECMO circuit and CVVH, platelet counts decrease. Such a large drop in platelet count can lead to suspicion of heparin induced thrombocytopenia, as the ECMO circuit requires some form of anticoagulation and the patient possibly is recovering from a procedure involving cardiopulmonary bypass using heparin [1–3].

Thrombocytopenia in the critically ill is common and can occur due to decreased production, consumption, or destruction of platelets. Naturally, a patient on ECMO checks many of the risk factors for thrombocytopenia including, sepsis, bleeding, various medications, hemodilution, the ECMO circuit, and possibly heparin induced thrombocytopenia [4].

Heparin induced thrombocytopenia (HIT) is a severe anti-body mediated reaction leading to thrombotic state and greatly increased morbidity and mortality. In addition it causes a longer ICU stay and failure to wean from ECMO [5]. While it is often suspected, it is rarely confirmed. Various retrospective studies of patients on ECMO showing significantly less confirmed cases in relation to suspected cases [5,6]. Heparin induced thrombocytopenia is believed to only affect approximately 0.5-3% of individuals receiving unfractioned heparin, however it is suspected far more often [7].

While it sounds simple in concept, a patient has a positive PF4 or serotonin release assay and the care team switches the patient over to a direct thrombin inhibitor and the patient is able to eventually recover, it can be more difficult. Serological tests for HIT are not resulted immediately. Thus, if a patient were to be suffering from HIT while still on heparin the consequences could be disastrous. A direct thrombin inhibitor is also a finite and expensive resource and waste results in an astronomical cost to hospitals. Argatroban waste can generate a cost of up to half a million in pure drug waste alone of unused medication, not including unnecessary use [8].

A large decrease in platelet count can be an indicator of heparin induced thrombocytopenia, requiring the immediate removal of heparin and replacement with a direct thrombin inhibitor such as argatroban [9]. These drugs are irreversible and more expensive, as well as often being instituted prior to detection of PF4 antibodies from laboratory tests. Argatroban, because of its clearance by the liver, makes it a great choice in a patient with compromised renal function [2]. Though it must be considered that argatroban is costly, and quite often a great amount of it is wasted placing extra cost on the patient and hospital [8].

Complicating this dilemma for physicians determining an anticoagulant to use on ECMO, is when a patient is on CVVH. Literature has shown that there could be a drop-in platelet count for a patient just undergoing a renal replacement therapy as high as 48%, which would immensely complicate the potential determination of a significant drop in platelet count for a patient on ECMO [10–12]. A patient on just CVVH may not be exposed to any

heparin at all and still express a large decrease in platelet count, which is why it would be beneficial to compare this drop-in platelet count to those that could be seen in a patient being treated with ECMO. The culprit has been found to likely be the CVVH filter, which causes a decrease in platelet count almost instantly and increases over time [12]. I plan to investigate this connection between the CVVH filter and the ECMO circuit through a retrospective study for my thesis project.

When looking at the ECMO circuit in itself, a 2016 study showed that the duration of ECMO had no discernable impact on platelet count whatsoever, and that drops in platelet count are merely associated with severity of illness and the platelet count at time of cannulation [13]. Another study from 2015 examining 119 patients on ECMO showed that suspected HIT occurred in 19% of the subjects with only one having a confirmed laboratory diagnosis [5]. HIT is rare, but the fear of it occurs often, resulting in alternative treatments being used that may not be necessary.

Overall, it is worth noting that critically ill patients being treated with ECMO may have other devices that can substantially impact anticoagulation from a perfusionist perspective. It could be worth it to increase awareness of other forms of extracorporeal devices. It is important to be cognizant of the entire patient's plan of care and recognize that a significant drop in platelet count is worth investigating, but to consider all potential causes.

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