The authors have no conflict of interest regarding this presentation to disclose.

Mild thrombocytopenia is an observable change seen in patients undergoing renal replacement therapy. One way we continue to work towards a life of hemodialysis without thrombocytopenia is to engineer biocompatible hemodialysis membranes. These membranes are described by an ability to elicit the least amount of inflammatory response, particularly through complement activation, and thus, decrease platelet destruction. Polysulfone membranes (F-160) used in Optiflux dialyzers are considered biocompatible, however here we will describe a rare case in which this particular membrane dialyzer induced severe acute thrombocytopenia that resolved after it was changed to a polyethersulfone membrane.

Our team reports a case of a 78-year-old female with a past medical history significant for diabetes mellitus type 2, hyperlipidemia, hypertension, and chronic kidney disease stage 4 with a glomerular filtration rate (GFR) of 16 and recent arteriovenous fistula placement for future dialysis who presented to the emergency department (ED) with complaints of worsening shortness of breath at rest that started 24 hours prior to arrival. She had no history of neuropathy, retinopathy, or bleeding disorders. She denied any family history of heart disease or bleeding disorders. She denied use of tobacco or illicit drug use in the past and drank alcohol occasionally. Home medications were sodium bicarbonate 650 mg three times daily (TID), simvastatin 20 mg daily, metoprolol succinate 25 mg twice daily, iron 150 mg daily, doxazosin 1mg daily, calcium carbonate 650 mg three times daily (TID), simvastatin 20 mg daily, metoprolol succinate 25 mg twice daily, iron 150 mg daily, doxazosin 1 mg daily, calcium carbonate 650 mg daily, aspirin 81 mg daily, and insulin regimen; lispro 3 units subcutaneously TID with meals and glargine 6 units subcutaneously at bedtime.

Initial vitals revealed a blood pressure of 212/86 mm Hg, heart rate of 88 beats per minute and regular, respiratory rate of 12 breaths per minute, temperature of 98.2°F, and oxygen saturation of 97% on 3 liters nasal cannula. Physical examination revealed mild jugular venous dilation with normal heart sounds and decreased lungs sounds at the bilateral bases. A one plus pitting bilateral pedal edema was also appreciated. There were no petechiae or purpura on skin examination. No lymphadenopathy, splenomegaly, or hepatomegaly was appreciated and no tenderness was elicited.

On admission, the patient’s platelet count was within a normal range at 156 x10^3/µL, BUN was 88 mg/dL and creatinine was 5.04 mg/dL. A chest x-ray showed evidence of congestive heart failure with an enlarged cardiac silhouette, bilateral pleural effusions, and unclear diaphragm margins. Initially, furosemide 60mg intravenous (IV) was given twice daily, and amlodipine 5mg was given twice daily for the initial regimen of what appeared to be primary acute heart failure secondary to acute renal failure and uncontrolled hypertension.

Hemodialysis was initiated on day three of hospitalization secondary to a progressive decline in renal function and no urine output. A significant fall in platelet count was observed following the first dialysis session reaching 80x10^3/µL. This was considered to be secondary to a progressive decline in renal function and no urine output. A significant fall in platelet count was observed following the first dialysis session reaching 80x10^3/µL. Ultimately, this reached a nadir of 45x10^3/µL on day eight. She was placed on sequential compression devices bilaterally for the prevention of DVT (deep venous thrombosis) so no HIT (heparin-induced thrombocytopenia) panel was ordered. Also, heparin was never used during any of her dialysis treatments. This aggressive decrease in platelets began to correct on day 10 to a peak of 210 x10^3/µL on day 18 following substitution with the Nipro ELISIO dialyzer which uses a polyethersulfone membrane and thus decreases direct interaction between the blood and polysulfone shown in Figure 1.

This report shows significant transient thrombocytopenia associated with a hemodialysis membrane recognized as being highly biocompatible (Fresenius Medical Care Optiflux polysulfone membrane F-160). It was reported that this particular hemodialysis membrane made of polysulfone lacks an alkyl polymer-grafted cellulose which would otherwise introduce a thin alkyl ether layer that covers the polysulfone membrane and thus reducing complement activation and therefore platelet consumption. Complement activity between blood products and dialysis membranes are a well known phenomenon causing thrombocytopenia due to C5a causing neutrophil activation, adherence, aggregation and release of platelet-activating factor, thus causing thrombosis and platelet consumption. Due to high cardiovascular mortality in hemodialysis patients related to inflammation and coagulation, membrane selection should be individualized, and novel biomarkers to identify membrane induced thrombocytopenia should be introduced.

References


Figure 1. Graphic representation of thrombocytopenia with improvement after change in dialyzer.