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What is this chocolate milk in my circuit? A cause of acute clotting of a continuous renal replacement circuit: Answers

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Answers

1. Factors influencing the patency of continuous renal replacement therapy (CRRT) circuits include catheter size and location, choice of anticoagulant, blood flow and replacement fluid flow rates, ultrafiltration rates (filtration fraction), hematocrit, platelet count, blood product transfusion as well as levels of sedation and paralysis of patients in the intensive care unit.
2. Early clotting of CRRT circuits leads to blood loss, decreased efficacy, and increased costs.
3. First, it is important to assess the course of events. What was happening when the circuit went down? Was the patient adequately sedated, or was he moving? The catheter placement and flow should be checked to ensure that nothing was blocked or kinked. The dialysis prescription including blood flow and replacement fluids should be reviewed to ensure adequacy for the patient's body surface area. The transmembrane pressure (TMP), filter pressures, return line pressures, and ultrafiltration rate of the circuit should be reviewed, as should the activated clotting time (ACT) and heparin dose, or ionized calcium levels and citrate dose, depending on the anticoagulant used. It is also important to look at the circuit. Close examination of the "clot" in the tubing of this circuit revealed a milky appearance, which was unusual and prompted further investigation to examine possible sources of lipemic or chylous build-up. Blood work, including

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hematocrit and platelet count, should be reviewed. In this patient, in retrospect, triglyceride levels were extremely elevated.

4. Physicians should be mindful of hypertriglyceridemia as a potential risk factor in critically ill patients receiving parenteral nutrition who have recurrent clotting of CRRT circuits. At our center, we currently monitor triglyceride levels once a week in patients on total parenteral nutrition (TPN). Closer monitoring of triglyceride levels is required in patients with elevated levels and the dose of fat emulsion administered should be adjusted accordingly.

Discussion

This case describes a child who required CRRT for management of acute kidney injury (AKI) with electrolyte imbalance and fluid overload. He was noted to have elevated triglyceride levels and had acute clotting of the CRRT circuit with lipemic blood noted in the circuit tubing. A literature search revealed three case reports of lipemic blood causing recurrent clotting and reduced CRRT circuit survival time in adult patients, but none reported in children [1–3].

The results of blood work done in our patient on day 7 and day 8 of admission are summarized in Table 1. The brownish-milky fluid was initially presumed to be the TPN/lipids being drawn into the dialysis catheter. On X-ray, left internal jugular central line and right external jugular dialysis catheter were approximately 4.4 cm apart. The lipid infusion was stopped. Blood was then drawn from the dialysis catheter, which separated in the syringe with a thick layer of brownish-milky viscous fluid. At the same time, an arterial line sample was drawn with similar separation noted after 20 min. Review of blood work from day 3 of admission showed a triglyceride level of 157 mg/dl (normal range, 27–125 mg/dl) that increased to 988 mg/dl 10 h after starting TPN/lipids (Fig. 1). Blood from the clotted CRRT circuit tubing had a triglyceride level of 1099 mg/dl. To ensure proper positioning, the dialysis catheter was pulled back 2 cm. Dialysis was reinitiated while keeping the patient off the lipid infusion. There was a film of lipemic fluid noted in the CRRT filter after initiation. The pre-filter flow rate was increased to help decrease the viscosity of blood flowing through the filter. The next morning, the triglyceride levels were 363 mg/dl. The third CRRT circuit lasted 63 h prior to being taken down routinely when the patient was transferred to another facility.

Advances in pediatric critical care, including management of sepsis, critical congenital heart disease, and organ transplantation depend on interventions that involve exposure to nephrotoxic agents. AKI is known to occur in 10–18 % of all patients admitted in the intensive care unit (ICU) [4]. CRRT is now the preferred modality for management of critically ill children with severe AKI, electrolyte abnormalities, and fluid overload. Compared to hemodialysis and peritoneal dialysis, CRRT provides gradual but constant removal of solutes and fluid while maintaining hemodynamic stability and the near elimination of the need for fluid restriction [4, 5].

One of the greatest challenges with CRRT is the early clotting of filters, leading to blood loss, decreased efficacy, and increased costs [6]. Factors influencing the patency of CRRT

circuits include catheter size and location, choice of anticoagulant, blood flow and replacement fluid flow rates, ultrafiltration rates (filtration fraction), hematocrit, platelet count, blood product transfusion, as well as levels of sedation and paralysis of patients in the ICU [6–8]. Clogging of the hollow fibers of the dialysis filters occurs due to deposition of protein and red cells on the membrane [9]. This is detected by increasing TMP, which was not noted in our patient.

Elevated triglyceride levels were noted in our patient shortly after initiation of TPN with fat emulsion. Fat emulsions consist of droplets that simulate endogenous chylomicrons, with a monolayer of phospholipids enveloping a triglyceride core [10]. Hemolysis may be associated with increased triglyceride synthesis and decreased catabolism [11]. The patient may have been predisposed to hypertriglyceridemia due to immunosuppression with tacrolimus [12], ongoing sepsis, as well as familial risk factors, although family history was unavailable.

Various factors contribute to the increased risk of circuit clotting in patients with elevated triglyceride levels. Hyperlipidemic patients have increased blood viscosities that correlate with serum triglyceride levels [13]. Further, hypertriglyceridemic patients are hypercoagulable due to elevated factor VIIc, factor VIIIc, plasminogen, and anti-thrombin-III levels [14]. Kazory et al. reported a shortened CRRT circuit life span after starting TPN with lipid emulsion [3]. The electron microscopic evaluation of the hemofilter fibers showed spherical lipid droplets, some of which were coalesced, surrounded by disorganized fibrin.

Early clotting of CRRT circuits is commonly encountered, but the cause is often unknown. Elevated triglycerides causing clotting of CRRT circuits is rare and has not been reported in the pediatric population. Physicians should be mindful of this potential risk in critically ill patients receiving parenteral nutrition who have recurrent clotting of CRRT circuits. Closer monitoring of triglyceride levels is required in patients with elevated levels and the dose of fat emulsion administered should be adjusted accordingly.

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Figure 1.

The patient's triglyceride levels increased after starting (total parenteral nutrition) TPN. He had a rise in serum creatinine and was started on continuous renal replacement therapy (CRRT). The second CRRT circuit clotted 18 h after starting.

Table 1

Table summarizing the pertinent laboratory results on the morning of day 7 and day 8 of admission

	Value (day 7)	Value (day 8)	Normal range	Units
Sodium	133	140	138–145	mEq/l
Potassium	3.5	3.5	4.1–5.4	mEq/l
Chloride	96	98	98–106	mEq/l
Carbon dioxide	23	20	20–26	mEq/l
Urea nitrogen	17	18	2–19	mg/dl
Creatinine	0.6	0.6	0.1–0.4	mg/dl
Calcium	9.5	9.8	8.7–9.8	mg/dl
Phosphorus	3.6	5.4	3.8–6.5	mg/dl
Albumin	3.9	4.4	3.5–4.6	g/dl
Hemoglobin	8.0	6.4	10–15	g/dl
Tacrolimus	-	4.9	5–20	ng/ml
Triglycerides	988	-	27–25	mg/dl