Clinical Images in Nephrology and Dialysis

Case Description

A 62-year-old man with a past medical history of diabetes mellitus presented to the hospital with shortness of breath and nonproductive cough. His vital signs revealed the following: BP, 83/52 mm Hg; heart rate, 119 beats per minute; respiratory rate, 28 breaths/min; oxygen saturation, 60% on room air; temperature, 103.3°F. Cardiopulmonary examination demonstrated sinus tachycardia and bilateral crackles. Initial basic laboratory values were as follows: white blood cells, 13.6 K/mm³ with neutrophil predominance; hemoglobin, 11.8 g/dl; platelets, 264 K/mm³; sodium, 135 mmol/L; potassium, 5.5 mmol/L; chloride, 97 mmol/L; bicarbonate, 21 mmol/L; BUN, 44 mg/dl; serum creatinine, 3.47 mg/dl; calcium, 7.4 mg/dl; albumin, 2.4 g/dl; normal liver function tests; lactate, 2.7 mmol/L; C-reactive protein, >300 mg/dl; D-dimer, 13,900 ng/ml; fibrinogen, 598 mg/dl; ferritin, 392 ng/ml; partial thromboplastin time, 73 seconds. Urinalysis was negative for hematuria or proteinuria. Urine sediment demonstrated many granular casts. Computed tomography scan of the chest without intravenous contrast showed bilateral posterior upper lobe consolidations (Figure 1A).

The patient was intubated and started on volume control ventilation using low tidal volumes according to the acute respiratory distress syndrome protocol (fraction of inspired oxygen, 100%; positive end-expiratory pressure, 12 cm H₂O) and required hemodynamic support with NE infusion. Testing confirmed infection with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). The patient developed anuria and a temporary nontunneled dialysis catheter was placed for continuous KRT (CKRT). Chest x-ray confirmed the tip of the dialysis catheter at the cavoatrial junction and CKRT was initiated with blood flow rate of 250 cc/min. Remarkably, within 2 minutes of dialysis initiation, a high venous pressure alarm was noted, and a large clot was extracted from the CKRT circuit tubing. Dialysis was discontinued, and the clot was aspirated (Figure 1B). The patient was started on systemic unfractionated heparin and CKRT was resumed uneventfully without any catheter manipulation and with blood flow rate of 250 cc/min.

Figure 1. | A multisystem disorder: Hypercoagulability in COVID-19. (A) Computed tomography scan of the chest without intravenous contrast demonstrates bilateral posterior upper lobe consolidations. (B) Blood clot extracted from the CKRT circuit tubing. (C) Computed tomography scan of the head without intravenous contrast reveals a left frontal infarct.
The hospital course was complicated by gastrointestinal bleeding, for which heparin was discontinued. The CKRT circuit clotted again and therefore the patient was started on prefiler citrate infusion at a rate of 100 cc/h, titrated to achieve a postfilter ionized calcium level of <0.4 mmol/L. Calcium gluconate was infused through a separate central line to replace calcium. No further clotting was noted in the CKRT circuit. However, a new neurologic deficit was observed and a computed tomography scan of his head without intravenous contrast revealed a left frontal (Figure 1C) and a small right occipital infarct. The patient transitioned to comfort care.

Thromboembolic events and disseminated intravascular coagulation are common in patients with coronavirus disease 2019 (COVID-19) (1). Hypercoagulability and thromboembolic events are associated with higher mortality (2,3). Empirical systemic anticoagulation is not unreasonable in severe cases of COVID-19 with elevated D-dimer levels (4). Frequent clotting of the CKRT filters and dialysis catheters was also observed (5). These complications can lead to reduced dialysis time, blood loss in the malfunctioning circuits, and waste of many dialysis filters. Although consensus guidelines for empirical anticoagulation in patients with COVID-19 who require CKRT have not been established, our center started using systemic unfractionated heparin or prefiler citrate in all of the patients infected with SARS-CoV-2. This intervention also helped in reducing the burden of filter clotting in patients on CKRT; however, we have found that some patients therapeutic on systemic heparin continue to experience clotting both systemically and within the CKRT circuit. More research is needed to explore the causes for and management of hypercoagulability in patients with COVID-19.

Teaching Points

- Thromboembolic events, hypercoagulability, and clotting of the CKRT filters and dialysis catheters are common in patients with COVID-19.
- Thromboembolic events are associated with higher mortality in patients with COVID-19.
- Using systemic unfractionated heparin or prefiler citrate reduces the burden of filter clotting during CKRT in patients infected with SARS-CoV-2.

Author Contributions

C. Cervantes wrote the original draft, and M. Hanouneh and S. Menez reviewed and edited the manuscript.

Disclosures

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References


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