Effective Use of Continuous Arteriovenous Hemodialysis in a Critically Ill Human Immunodeficiency Virus-Positive Patient with Acute Kidney Failure

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To the editor,

Continuous arteriovenous hemodialysis (CAVHD) is a highly effective modality for the replacement of kidney function in patients with acute kidney failure. In patients with acute kidney failure and compromised circulation, hemodialysis may lead to serious hypotension or cardiac arrhythmia.[1] This fact may limit possible treatment; restriction of the patient's fluid intake may be attempted, usually with a concomitant restriction of nutrition. With continuous hemofiltration, however, almost any quantity of fluid can easily be removed over a 24-hour period. We had a patient on CAVHD who showed a good control of uremia and hypervolemia and stable hemodynamics while on CAVHD.

An 80-year-old man, a known case of diabetes mellitus, hypertension, ischemic heart disease, and right hemiparesis presented with high-grade fever, worsening breathlessness, generalized malaise of 7days' duration, and decreased urine output since 1 day earlier. There was no evidence of diabetic retinopathy or kidney dysfunction in the past. He was anuric at the time of admission and severely breathless at rest with a blood pressure of 90/60 mm Hg. He had bilateral pitting edema of the legs, and sinus tachycardia was present (110 beats/min with an S3 gallop). Auscultation of the lungs disclosed pulmonary congestion.

Laboratory data were suggestive of sepsis-induced acute kidney failure. Serum creatinine level was 5.63 mg/dL; blood urea nitrogen, 80 mg/dL; hemoglobin, 9.9 g/dL; leukocyte count, 21×10^9/L; platelet count, 280×10^9/L; C-reactive protein, 312 mg/dL; serum sodium, 136 mEq/L; serum potassium, 5.4 mEq/L; serum bicarbonate, 11 mEq/L; total bilirubin, 2.4 mg/dL; direct bilirubin, 1.6 mg/dL; aspartate aminotransferase, 629 U/L; alanine aminotransferase, 206 U/L; and alkaline phosphatise, 120 U/L. His serum was reactive for human immunodeficiency virus and negative for malarial parasite, leptospira, dengue, hepatitis B virus, and hepatitis C virus. His abdominal ultrasonography was unremarkable, and echocardiography revealed an ejection fraction of 35% with mild mitral regurgitation. His chest radiography confirmed pulmonary edema with infective consolidation in the left lung base. In view of tachypnea, fluid overload, and metabolic acidosis, he was put on ventilatory assistance and ionotropic support was initiated following profound hypotension. His blood pressure was stabilized at 86/54 mm Hg with dopamine, noradrenaline, and adrenaline infusion. He was started on diuretic therapy, bicarbonate infusion, and broad-spectrum intravenous antibiotics.

He continued to be anuric and his arterial blood gas analysis revealed severe metabolic acidosis with a pH of 7.096; pCO_2, 21.3 mm Hg; pO_2, 66 mm Hg; and HCO_3, 7 mEq/L. A decision to initiate renal replacement therapy was made. In view of his profound hypotension, hemodialysis and sustained low-efficiency dialysis were deferred. With a high-risk informed consent, he was started on CAVHD. Vascular access was obtained by right-sided femoral arterial and venous cannulae. We used a 0.5-m^2 hollow fibre dialyzer, and 1.5% dextrose peritoneal dialysis fluid was infused into the dialysate compartment by a constant infusion pump at 1 L/h. Anticoagulation was achieved by heparinization administrated through the arterial port. No blood pumps were used. The ultrafiltrate was collected into a container and was measured hourly, and the calculated amount of replacement fluid was infused to the patient through the venous port. The fluid loss was replaced with isotonic saline and 5% dextrose with an addition of sodabicarb and calcium gluconate on regular basis. The volume to be replaced in 1 hour was calculated as follows U/F + U - I – D, where U/F stands for previous hours ultrafiltrate; U, for previous hours urine

output; I, for previous hours infusion fluid; and D, for deficit volume. The rate of ultrafiltration was adjusted to maintain the extracellular volume status by adjusting the height of the dialysate bag. The effective ultrafiltration achieved was initially 50 mL/h and gradually increased to 100 mL/h. The patient's blood gases, serum electrolytes, blood urea nitrogen, serum creatinine, serum calcium, and serum magnesium levels were monitored on regular basis. His arterial blood gas improved substantially over a period of 72 hours (Table). After 72 hours of therapy, his blood urea nitrogen dropped to 30 mg/dL and serum creatinine, to 3.4 mg/dL, and his urine output started improving. His ionotropic requirement gradually came down, and at the end of the 3rd day, he was off ionotropes as well as CAVHD. There were no complications related to the extracorporeal circuit, the filter, anticoagulant therapy, electrolyte status, or changes in patients' hemodynamic state.

Continuous arteriovenous hemodialysis and continuous venovenous hemofiltration are the two commonly used modalities of continuous hemofiltration. The main advantages of CAVHD and continuous venovenous hemofiltration are effective control of azotemia as well as satisfactory regulation of electrolyte and acid-base balance in hemodynamically compromised patients. Additional advantages include highly effective fluid removal, better hemodynamic tolerance, easier administration of parenteral nutrition and intravenous medications, and the fact that it is a technically simple procedure. Patients in whom CAVHD is a preferred mode of treatment are those with acute kidney failure following a major surgery, hemodynamically unstable patients, those with a recent myocardial infarction, and patients with severe septicaemia, patients having cardiac failure with oliguria, acute pulmonary edema with azotemia, severe electrolyte, and acid-base disturbances, and those with profound hypotension and fluid overload.[1] There are no specific contraindications for CAVHD. Active bleeding should be considered a relative contraindication. The complications are mainly related to the vascular access, namely catheter-related infection and thrombosis of the catheter as well as thrombosis of the tubing and dialyzer, local or systemic bleeding, and complications related to arterial cannulation. Bellomo and coworkers obtained an excellent control of azotemia in 12 critically ill patients with acute kidney failure on CAVHD.[2]

In our experience, there has been a substantial improvement in azotemia, acid-base balance, and hemodynamic stability with the use of CAVHD. In developing countries where there is a lack of dedicated continuous renal replacement therapy machines for treating patients with human immunodeficiency virus, CAVHD offers a similar control of uremia. Continuous arteriovenous hemodialysis does not cure renal failure, but it is a safe and efficient way of replacing kidney function while the kidneys recover from the disease or injury.

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Arterial Blood Gas Parameters

Parameter	Time					
	Baseline	6 Hours	18 Hours	24 Hours	48 Hours	72 Hours
pH	7.09	7.31	7.38	7.42	7.48	7.57
pCO_2, mm Hg	21.3	23.5	25.6	28.0	34.9	35.0
pO_2, mm Hg	66	119	125	136	146	188
HCO_3, mEq/L	7.0	10.0	13.7	18.0	25.8	31.4