Correction of Respiratory Acidosis by Continuous Renal Replacement Therapy

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ABSTRACT

Background: Continuous renal replacement therapy is generally deployed for volume overload in a hemodynamically unstable patient with renal insufficiency. Management of acute respiratory disease syndrome and systemic inflammatory response syndrome are recent novel indications for continuous renal replacement therapy in the intensive care unit.

Methods: We present a case of a patient with worsening chronic obstructive pulmonary disease, volume overload and renal failure.

Results: In the face of acute renal failure due to hemodynamic causes, continuous renal replacement therapy was instituted to replace adequate bicarbonate to correct a combined metabolic and respiratory acidosis. At the same time, the patient's volume status was corrected by volume removal.

Conclusion: This case illustrates how continuous renal replacement therapy may be used to replace adequate bicarbonate when the kidney cannot either reclaim or generate bicarbonate to correct a combined metabolic and respiratory acidosis while simultaneously removing volume.

INTRODUCTION

Continuous renal replacement therapy (CRRT) is one of the more important advances in intensive care medicine over the past decade. Hemodynamic instability in the setting of acute renal failure and severe fluid overload are established main indications for CRRT.[1-3] However, it has also been utilized for non-renal indications, as it may be of benefit in the removal of inflammatory mediators and the elimination of other endogenous toxic solutes from the blood. With this in mind, CRRT has been used for treatment of systemic inflammatory response syndrome (SIRS), sepsis, acute respiratory distress

syndrome (ARDS), cardiopulmonary bypass (CPB), inborn errors of metabolism, lactic acidosis, crush injury, and tumor lysis syndrome.[4-9]

We recently used CRRT on a 76-year-old patient, to treat a pulmonary problem, who also had renal insufficiency. We report on a patient with a history of chronic obstructive pulmonary disease who presents with severe respiratory acidosis when acute renal failure (ARF) was precipitated by the aggressive treatment of congestive heart failure.

CASE REPORT

A 76-year-old African American female was admitted to the hospital for progressive shortness of breath, orthopnea, paroxysmal nocturnal dyspnea, and lower extremity edema. Her medical history included congestive heart failure, atrial fibrillation, hypertension, asthma, chronic obstructive pulmonary disease (COPD), diverticulosis, gastroesophageal reflux disease (GERD), and anxiety disorder. Cardiac catheterization one year prior to admission showed normal coronary arteries. She smoked a half pack of cigarettes a day for 40 years. Pulmonary function tests one year prior to admission showed the patient to have a FEV_1 = 0.9 L (53%), FVC = 1.1 L (51%), and FEV1/FVC = 81%. Her medications (which she had only been taking intermittently) were warfarin, diltiazem, albuterol/Azmacort MDI, oxybutinin, Celexa, theophylline, and furosemide.

On admission, her blood pressure was 133/109 mm Hg; pulse, 120 bpm; respirations, 32 breaths/minute; pulse oximeter, 89% on room air; and temperature 36.8˚C. Physical exam revealed an elderly female in respiratory distress, only able to speak short sentences. Her lungs had poor air movement with diffuse wheezes and bi-basilar rales. Her heart rate was tachycardic with frequent ectopic beats and a grade III/VI systolic ejection murmur. The abdomen was distended, but soft and nontender with normal bowel sounds. Her lower extremities had 2+ pitting edema.

Chest radiography showed cardiomegaly with a bibasilar infiltrate consistent with pulmonary edema. The EKG showed atrial fibrillation with a rapid ventricular response. Echocardiogram showed left ventricular hypertrophy with significant mitral stenosis and a normal ejection fraction. Representative laboratory results for each day are shown in Table 1.

Initially, the patient was diagnosed to have both a COPD exacerbation and congestive heart failure (CHF). She was given albuterol/atrovent nebulizers, solumedrol, and low dose oxygen by nasal cannular for COPD management. On admission, the patient stated she did not want to be intubated. Antibiotics were also given for suspected pneumonia. The patient was aggressively diuresed to treat CHF on day 1. She became oliguric despite diuretic regimen. Due to hypotension, diuretics were discontinued and dopamine drip was started at 4.5 mcg/kg/hour on day 2. Simultaneously, atrial fibrillation responded poorly to diltiazem and digoxin resulting in a diltiazem drip to control the heart rate of 150 bpm despite a systolic blood pressure in the 90's. An amiodarone drip at 0.5 mg/hr to restore normal sinus rhythm was unsuccessful in converting the heart rate to sinus. She was anticoagulated for future cardioversion. A pulmonary artery catheter was placed and the initial set of hemodynamics were pulmonary artery pressure of 74/42 mmHg and a postcapillary wedge pressure of 38 mmHg, consistent with both a left and right-sided heart failure (Table 2).

Renal failure was determined to be pre-renal due to a fractional excretion of sodium (FENA) of 0.6. On hospital day

Table 1. Serial Measurements of Arterial Blood Gases and Selected Serum Chemistries

Hospital Day	ABG ($pH/pCO_2/pO_2$)	O_2 Sat (%)	Na mmol/L	K mmol/L	Cl mmol/L	CO_2 mmol/L	BUN mmol/L	Creat µmol/L
1	7.27 / 64 / 71	92	140	3.6	96	30	6.4	106.1
2	7.28 / 68 / 49	81	133	4.3	91	27	13.2	229.8
3	7.22 / 68 / 67	90	137	4.4	89	28	20.0	344.8
4	7.19 /72/ 58	87	131	4.4	95	24	21.1	362.4
5	7.22 / 68 / 51	80	128	4.9	88	27	25.3	450.8
6	7.20 / 69 / 43	72	128	4.5	92	24	21.4	247.5
7	7.34 / 59 / 51	83	133	3.9	95	28	14.3	132.6
8	7.29 / 61 / 45	77	131	4.6	97	27	10.0	106.1
9	7.25 / 66 / 81	94	133	4.0	97	27	10.4	114.9
10	7.32 / 48 / 77	95	136	4.1	97	32	19.3	176.8
11	7.35 / 65 / 57	87	136	4.3	93	32	30.3	194.5
12	7.38 / 31 / 59	90	139	3.6	93	37	36.4	194.5
13	7.41 / 56 / 55	88	135	4.0	94	36	29.3	141.4

5, the patient was started on continuous venovenous hemodialysis (CVVHD), with ultrafiltration of –50 cc/hour using 1.5% Dextrose peritoneal fluid as dialysate. The patient did not have adequate renal response to normalize the pH, which fell due to a combination of respiratory acidosis and renal failure. She was thus given IV sodium bicarbonate as part of the replacement fluid via CVVHD as volume was removed on hospital days 6 to 8 to maintain a pH above 7.3. On hospital day 8, CVVHD was changed to continuous venovenous hemodiafiltration (CVVHDF) with lactated ringers at 100 cc/hour as replacement fluid. On hospital day 9, the patient's BUN and creatinine improved to 10.4 mmol/L and 114.9 µmol/L, respectively, and CRRT was stopped. On hospital day 11, the patient was still oliguric. BUN and creatinine worsened to 30.3 mmol/L and 194.5 µmol/L, respectively. Hemodialysis was thus performed, since she was hemodynamically stable. On hospital day 13, the patient spontaneously started to diurese with 1600 cc of urine output in 24 hours with creatinine dropping to 141.4 µmol/L.

DISCUSSION

This case illustrates the use of continuous renal replacement therapy to treat respiratory acidosis in a patient with concomitant CHF and acute renal failure. Because of the patient's renal insufficiency, the normal compensation of bicarbonate retention was precluded. We selected CRRT as the form of renal replacement therapy in order to treat the patient's volume overload and respiratory acidosis.

In the intensive care unit, continuous forms of renal replacement therapy are superseding conventional hemodialysis as the treatment of choice because of their ability to remove volume in a gentle fashion. There has not been a large-scale prospective trial that shows a survival advantage to using CRRT versus conventional intermittent hemodialysis (IHD). However, there are comparative reports between CRRT vs IHD.[10-13]

CRRT was first used in 1977 by Kramer at the University of Gottingen.[14] Since then there have been many technological advancements, including increased ultrafiltration rates, improvements in pumps and filters, and automation of the system.[15] As a result of these improvements, CRRT has become accessible to intensive care units in many medical centers. CRRT has had an

Table 2. Serial Swann Ganz Catheter Measurements*

Hospital Day	PAP mmHg	PCWP mmHg	CWP mmHg	CO L/min	CI L/min
3	74 / 42	38	15	6.6	3.6
4	61 / 21	27	16		3.4
5		17	9	6.1	3.4
6 . . 10					
11		11	6	4.4	2.4
12		14	7	3.9	2.2

*PAP indicates pulmonary artery pressure; PCWP, pulmonary capillary wedge pressure; CVP, central venous pressure; CO, cardiac output; and CI, cardiac index.

impact in the treatment of acute renal failure in the intensive care unit.[16-21] Traditional indications for CRRT are shown in Table 3.[22]

CRRT relies upon a slow dialysate flow rate, which is administered on a continual basis, 24 hours a day for as long as it is required. Commercially prepared dialysate specifically for CRRT is available. The dialysate contains sodium, chloride, potassium, calcium, lactate, and magnesium. In the absence of this solution, one may use solutions prepared for peritoneal dialysis. Hyperglycemia and increased pCO_2 production from dextrose metabolism may limit its use. Alternatively, the in-hospital pharmacy may customize dialysate solutions with the desired solutes for patients with specific needs. Dialysate containing bicarbonate is now commercially available.[23]

CRRT is "gentler" on the patient, avoiding drops in blood pressure that frequently occur with conventional intermittent hemodialysis. The advantages of CRRT are that it avoids rapid fluid and electrolyte shifts, provides more precise fluid and metabolic control, decreases hemodynamic instability, supplements solute and fluid removal, when used in conjunction with hemodialysis, permits virtually unlimited fluid administration, and may have enhanced ability to remove injurious cytokines.[7-8,15,24] The disadvantages are that it is costly, requires expensive commercially prepared dialysate, one-on-one nursing, and prolonged anticoagulation.[15,16]

In the context of respiratory acidosis, volume overload, and renal insufficiency, renal replacement therapy, both CRRT and IHD, can increase pH and correct solute abnormalities. However, when volume overload is present in a patient who is hemodynamically unstable, CRRT offers an advantage over IHD. In addition, CRRT offers the ability to "fine tune" a patient's pH. In the patient presented, the pCO_2 remained uniformly elevated. When the pCO_2 is set in this fashion, pH titration with CRRT and IHD are probably equivocal. However, there are other similar scenarios in which the pCO_2 is elevated and rapidly changing. Patients with acute respiratory distress syndrome (ARDS), who are commonly treated with low lung volume therapy, which often results in permissive hypercapnia, suffer from prolonged respiratory acidosis with the pCO_2 oscillating as the patient's lung compliance changes. Recently, the ARDS Network trial showed a mortality benefit with the use of low lung volume therapy.[25] Unlike previous trials, which were unable to show a mortality benefit, this large well-powered study showed a survival advantage. The authors commented that one of the major differences between this study and the other low lung volume approaches was the decision to defend pH to as close to normal range as possible. This was achieved

Table 3. Traditional Indications for CRRT

• Volume overload secondary to congestive heart failure or massive resuscitation states.
• Cardiopulmonary bypass.
• Systemic inflammatory response syndrome (SIRS) with multisystem organ failure.
• Adult Respiratory Distress System (ARDS).
• Fulminant or subfulminant liver failure as a bridge to liver transplantation.
• Rhabdomyolysis.
• Severe burns associated with acute renal failure.
• Cerebral edema secondary to trauma, vascular accident, or toxic overdose when associated with renal insufficiency.
• Severe episodes of tumor lysis syndrome.

by respiratory rate increases and bicarbonate infusions to maintain a minimum pH of 7.30. Patients with multi-system organ dysfunction (MSOD) often develop concurrent ARDS and ARF. If the decision is made to use the low lung volume approach, permissive hypercapnia is likely to ensue. Bicarbonate infusion in a patient with renal insufficiency could eventually cause volume overload and worsening hypoxemia. Therefore, CRRT would be an advantageous choice to treat renal insufficiency and to titrate the pH in conjunction with the patient's varying pCO_2.

The ARDS Network study has recently come under criticism because of the tidal volumes used in the control arm.[26] Nonetheless, it is possible that if the use of permissive hypercapnia becomes accepted and widespread, CRRT might be useful as an adjunct to this therapy.

We recommend the early use of continuous renal replacement therapy for hypercapnic patients with renal insufficiency when base replacement would cause unacceptable volume overload. The two most common scenarios are patients with COPD and renal insufficiency exacerbated by CHF, and those patients with ARDS and renal insufficiency who are treated with low lung tidal volume strategies.

This case illustrates another indication for CRRT in the critically ill patient with renal insufficiency and volume overload, namely respiratory acidosis.

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