Case Report

Sodium bicarbonate buffer for weaning from venovenous extracorporeal membrane oxygenation in patients with hypercapnic respiratory failure and acute renal failure

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Abstract:

Although the routine use of alkali buffer is not recommended in patients with respiratory acidosis, some patients may benefit from its administration. A 42-year-old man was treated with venovenous extracorporeal membrane oxygenation (VV-ECMO) and continuous venovenous hemodiafiltration (CVVHDF) due to necrotizing pneumonia and emphysematous cystitis with *Klebsiella pneumoniae*. Although the sweep gas flow rate of the VV-ECMO was gradually reduced, he failed to wean off VV-ECMO due to respiratory acidosis, followed by tachycardia and tachypnea on the 63rd day of VV-ECMO. Therefore, we mixed sodium bicarbonate in the replacement fluid of CVVHDF for 5 days to avoid an intolerable decrease in blood pH after discontinuing the VV-ECMO sweep gas. When the serum bicarbonate concentration was >30 mmol/L and pH was maintained at >7.30 with a PCO_2 of >60 mmHg, VV-ECMO was finally decannulated. Sodium bicarbonate buffer through the replacement of CVVHDF fluid facilitated VV-ECMO weaning in a patient with hypercapnic respiratory failure.

Keywords:

Bicarbonate buffer, respiratory acidosis, venovenous extracorporeal membrane oxygenation

Respiratory acidosis is common in patients with respiratory failure[1] and is acceptable if tolerated by patients; administration of alkali buffer in patients with respiratory acidosis is controversial and not routinely recommended owing to concerns.[2] However, bicarbonate buffer is sporadically used in patients with respiratory acidosis. We describe a patient who first failed to wean off venovenous extracorporeal membrane oxygenation (VV-ECMO) due to respiratory acidosis and was finally removed from VV-ECMO after the planned administration of sodium bicarbonate.

Case Report

A 48-year-old man presented to the emergency room with emphysematous cystitis and a prostatic abscess. Chest computed tomography (CT) in the emergency room showed multifocal consolidations in the right middle and lower lobes of his lungs [Figure 1a]. *Klebsiella pneumoniae* was cultured from blood, urine, and sputum specimens. With the administration of ceftriaxone, cystitis seemed to improve; however, pneumonia was aggravated and tension pneumothorax developed. Follow-up of the chest and abdominal CT also showed pneumonia aggravation, with a larger extent of consolidation and thickened

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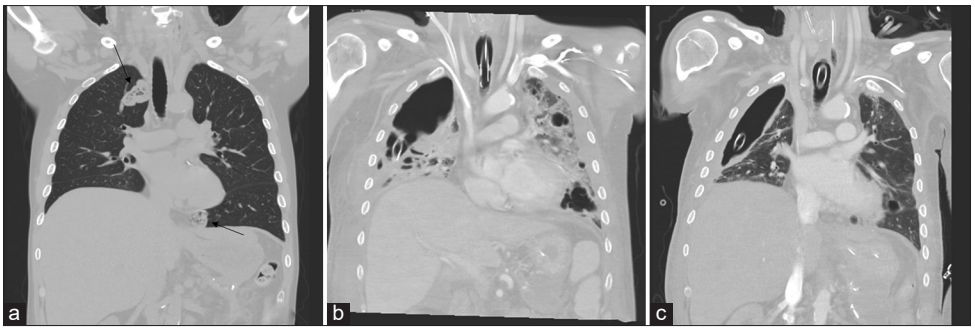

Figure 1: Chest CT of the patient at each point of the treatment period (a-c). CT: Computed tomography

pleura [Figure 1b]. A bronchopulmonary fistula (BPF) was suspected in the right upper lung. Renal infarction in the right kidney and subcapsular hemorrhages in the left kidney were newly observed. Methicillin-resistant *Staphylococcus aureus*, *Pseudomonas aeruginosa*, and multidrug-resistant *Acinetobacter baumannii* were newly cultured in sputum and pleural fluid samples. Therefore, the antibiotics were changed to a combination of teicoplanin, meropenem, and colistin.

VV-ECMO was started due to Types I and II respiratory failure on hospital day 14; arterial blood gas analysis showed a pH of 7.260, PCO_2 of 58.5 mmHg, PO_2 of 50.7 mmHg, HCO_3– of 25.7 mmol/L, oxygen saturation of 78.7% with a positive end-expiratory pressure of 5 cmH_2O, and a fraction of inspiration of oxygen of 1.0. Continuous venovenous hemodiafiltration (CVVHDF) was commenced due to decrease in hourly urine output and increase in serum blood urea nitrogen and creatinine levels (95.5 and 2.74 mg/dL, respectively). During the 1st month of ECMO, the expiratory tidal volume was <150 mL (2 mL/kg of predicted body weight). After ECMO support for >2 months with antibiotic treatment, the pneumonic consolidation in the left lung was much improved [Figure 1c], although the BPF in the right lung persisted. The PO_2 was >100 mmHg with blood flow through the ECMO at 1 L/min. The patient's expiratory tidal volume was 300 mL. However, when we reduced the ECMO sweep gas from 0.5 to 0 L/min, the PCO_2 increased from 52.0 to 61.6 mmHg and the pH decreased from 7.31 to 7.24, with a serum bicarbonate concentration of 25.9 mmol/L. The patient was agitated, his heart rate was elevated, and his blood pressure decreased. Because the patient was anuric and supported by CVVHDF until the day when we first tried to wean him off VV-ECMO, we added 80–120 mEq of sodium bicarbonate to every 5 L of CVVHDF replacement fluid. After 6 days of sodium bicarbonate infusion to the replacement fluid from ECMO day 64 to 69, the basal serum bicarbonate concentration increased to >30 mmol/L. Then, pH was >7.30 even when the PCO_2 exceeded 60 mmHg due to the sweep gas off. Although the patient was tachypneic, his respiration rate was <30 breaths/min, with no change in blood pressure. Finally, we weaned the patient off VV-ECMO on ECMO day 70 [Figure 2]. With CVVHDF support, we observed no volume overload or hypernatremia due to the sodium bicarbonate infusion.

Discussion

A hypercapnic state is commonly permitted in several conditions in patients with respiratory failure. However, severe acidemia can cause deleterious effects, as severe acidemia and tissue acidosis impair organ perfusion through vasoconstriction. Moreover, cardiac output and renal blood flow decrease, and pulmonary vascular resistance increases. Metabolic problems are also triggered, including effects on brain metabolism, insulin resistance, inhibited anaerobic glycolysis, and decreased lactate uptake in the liver.[3] Therefore, some patients cannot tolerate respiratory acidosis; thus, management of respiratory acidosis is important in these patients.

In the acute phase of respiratory acidosis, nonbicarbonate body buffers titrate acidemia based on the negative charges of proteins, hemoglobin, and phosphates (a). Bicarbonate does not directly buffer respiratory acidosis (b). During the chronic phase (3–5 days) of respiratory acidosis, a compensatory renal response develops. The kidney excretes H^+, producing NH_4^+, and absorbing HCO_3^-. The increased HCO_3– concentration results in less chance of arterial hydrogen ion concentration and blood pH (c). However, sodium bicarbonate buffer may increase PCO_2, considering the Henderson–Hasselbalch equation (d).[4]

a. $H_2CO_3 + Buf^- > HBuf^- + HCO_3^-$
b. $H_2CO_3 + HCO_3^- > HCO_3^- + H_2CO_3$
c. $[H^+] = 24 \times PCO_2/[HCO_3^-]$
d. Henderson–Hasselbalch equation:
$H_2O + CO_2 > H_2CO_3 > H^+ + HCO_3^-$

Our patient had no renal compensation. The patient was anuric and was supported by CVVHDF. The plasma bicarbonate concentration in arterial blood gas analysis was 22–26 mmol/L when sodium bicarbonate was not administered. Furthermore, he had been in

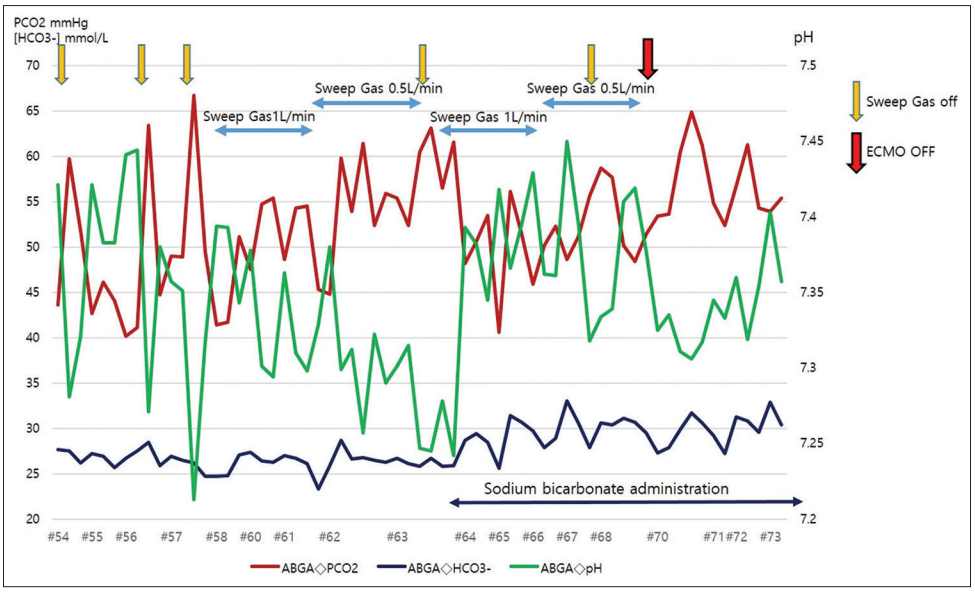

Figure 2: A daily record of arterial blood gas analysis in the weaning period of VV-ECMO and the changes in the VV-ECMO sweep gas flow rate. VV-ECMO: Venovenous extracorporeal membrane oxygenation

a chronic, critically ill state with VV-ECMO support for >2 months. The possible buffers to compensate for the respiratory acidosis were poor. At his best, his hemoglobin level was 7–8 g/dL, serum protein was 5.5 g/dL, and serum albumin was 2.5 g/dL. The phosphorous level was also marginal. Under such conditions, the response of the patient to decreased ECMO sweep gas was a decrease in pH; namely, 7.24 at a PCO_2 of 60 mmHg, followed by tachypnea, tachycardia, and hypotension. The mean arterial blood pressure decreased from 79 to 49 mmHg. Therefore, we commenced additive bicarbonate administration targeting a bicarbonate concentration of >30 mmol/L through the CVVHDF replacement fluid. Simultaneously, the ECMO sweep gas was tapered slowly over 1 week to 0 L/min. There was no volume overload or sodium retention due to CVVHDF support. When PCO_2 exceeded 60 mmHg, the pH was maintained at >7.30, the vital signs were stable, and the patient did not complain of dyspnea.[5] When the patient was stable for >1 day without sweep gas flow, VV-ECMO was successfully decannulated.

The use of bicarbonate buffer to compensate for respiratory acidosis remains controversial because bicarbonate can increase PCO_2 (D) and the sodium salt included in the buffer can cause hypernatremia and hyperosmolality. Another concern is that bicarbonate cannot increase intracellular pH.[6] Finally, there are no clinical data supporting bicarbonate buffer use in patients with respiratory acidosis. However, our patient did not have any risk of sodium retention under CVVHDF support. PCO_2 was planned to increase as the sweep gas flow decreased. The amount of CO_2 expected to be produced from the infused bicarbonate is much smaller than that from daily metabolism.[3]

We presented the case of a patient with hypercapnic respiratory failure who was successfully weaned off VV-ECMO using bicarbonate buffer through CVVHDF. Although the administration of sodium bicarbonate should not be the routine practice in patients with respiratory acidosis, the strategic compensation of respiratory acidosis using bicarbonate buffer in patients with an abnormal metabolic compensation system could be helpful in the weaning phase of VV-ECMO, if the complications of sodium bicarbonate can be properly controlled.

Ethical comment

Informed consent for the publication of the case report was obtained.

Author contributions

Conceptualization: Kim JH, Investigation, Methodology: Hwang JW, Writing original draft: Kim S, Writing and editing: Kim JH, Kim S, Hwang JW.

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Conflicts of interest

There are no conflicts of interest.

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