was treated with bicarbonate dialysate in the ICU. We are in agreement in this therapy. We believe that the correction of metabolic acidosis that followed the dialysis in this case is in part due to the removal of the unmeasured fixed acids.

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Treatment of dilutional acidosis with sodium bicarbonate should be approached cautiously. We agree with Russo that severe metabolic acidosis such as lactic acidosis combined with worsening acidosis from normal saline infusion should likely be treated with conservative amounts of sodium bicarbonate and by changing to a lower chloride solution such as Ringer’s lactate, if not contraindicated. However, we disagree on the necessity of treating dilutional acidosis alone without any other metabolic insult. We are not aware of any studies showing dilutional acidosis to be harmful.

Traverso et al. in the hemorrhagic swine model did find statistically nonsignificant improved survival in swine resuscitated with Ringer’s lactate over 0.9% saline. Russo in his letter implied the lower chloride level with higher pH and bicarbonate level in the Ringer’s lactate group to be the reason for improved survival. However, the group of swine who received PlasmaLyte had a lower survival rate than the 0.9% saline group despite having lower chloride and higher bicarbonate and pH levels.

In our case report, no further sodium bicarbonate was given after the diagnosis of dilutional acidosis was made. Rapid extracellular changes in bicarbonate and chloride do not cause such changes at the intracellular level. Rosenbaum et al. found no change in intracellular pH in dogs with extracellular expansion from 0.9% saline. Sodium bicarbonate therapy has the potential for increasing intracellular acidosis by increasing the diffusion of molecular CO₂.

Much investigation still needs to be performed on the etiology of dilutional acidosis and the need for treatment. Clinical studies on extracellular volume expansion comparing the effects of 0.9% saline and lower physiologic chloride solutions without any base buffer need to be done to clarify whether dilutional acidosis is caused by an increase in chloride or an actual dilution of bicarbonate. These studies should also examine changes in strong ion difference to see if Stewart’s analysis is applicable to explain dilutional acidosis. Further, the measurement of intracellular pH before and after sodium bicarbonate is needed to determine if dilutional acidosis by itself is potentially harmful or should be corrected by pharmacologic means.

We appreciate Khorasani and Appavi’s comments concerning other possible causes of metabolic acidosis. Although there is no way to measure the amount of bicarbonate lost secondary to blood loss or from electrolyte exchange across the open abdominal surgical field, we believe these losses were negligible and in no way accounted for the hyperchloremic metabolic acidosis. Nine units of packed erythrocytes were given for blood replacement, and we are not aware of any literature supporting the loss of significant bicarbonate through the bowel and peritoneum from surgical irrigation with saline.

In our case report, the patient had a normal bicarbonate at the start of surgery, and we believe the fixed acid load was not significant after only 8 h without dialysis. We certainly recognize the potential for multiple causes of a metabolic acidosis to occur simultaneously such as dilutional acidosis combined with a lactic acidosis, ketones, or inorganic acids. Dilutional acidosis from isotonic saline has the potential for hindging an underlying anion gap acidosis by decreasing the existing anion gap from chloride elevation. Hence, we believe other causes of a metabolic acidosis such as lactic acidosis should be excluded before a clinician attributes a metabolic acidosis solely to the infusion of isotonic normal saline and extracellular volume expansion.

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