

CASE REPORTS

Anesthesiology
1997; 86:501-3
© 1997 American Society of Anesthesiologists, Inc.
Lippincott-Raven Publishers

Dilutional Acidosis: Is It a Real Clinical Entity?

Donald D. Mathes, M.D.,* Robert C. Morell, M.D.,* Michael S. Rohr, M.D., Ph.D.†

DILUTIONAL acidosis occurs as a result of rapid extracellular volume expansion decreasing the measured serum bicarbonate. This phenomenon is not well described in the literature. Searching the English medical literature, we found only one case report of dilutional acidosis¹ and no case reports of dilutional acidosis occurring within the perioperative period. Several authors have concluded that dilutional acidosis has little actual clinical significance because of cellular buffering in which rapid extracellular volume expansion causes only minimal changes in measured extracellular bicarbonate and pH levels.²⁻⁴ We report a case of dilutional acidosis occurring intraoperatively as a direct result of giving a large volume of isotonic saline.

Case Report

A 46-year-old, 65-kg woman presented with end-stage renal disease secondary to polycystic kidney disease. She was scheduled to undergo a bilateral nephrectomy and removal of Tenckhoff catheter. The patient had been undergoing peritoneal dialysis for 4 months before surgery and was to eventually receive a living related kidney transplant 1 month after her planned bilateral nephrectomy. She had no other sequelae from her polycystic kidney disease except for cystic involvement of the liver. Otherwise, her medical history was remarkable only for hypertension, treated with 2.5 mg lisinopril daily. Preoperative laboratory values included: sodium, 137 mEq/l; potassium, 4.9 mEq/l; chloride, 90 mEq/l; bicarbonate, 27 mEq/l; blood urea nitrogen, 60 mg/dl; and creatinine 12.6 mg/dl.

In addition to routine monitors, a radial arterial catheter was inserted before the start of surgery. Anesthesia was induced with sodium thiopental, fentanyl, and cisatracurium and maintained with isoflurane/fentanyl/air/oxygen, and the lungs were mechanically ven-

tilated after tracheal intubation. Baseline arterial blood gas was pH 7.40, PCO₂ 39 mmHg, PO₂ 202 mmHg, with a base deficit of -0.5 mEq/l, and HCO₃⁻ 24.5 mEq/l.

The patient required large amounts of crystalloid and blood products due to continued blood loss secondary to a difficult dissection and removal of the grossly enlarged cystic kidneys. The patient remained hemodynamically stable, but was noted to have periods of increasing systolic pressure variation on the arterial blood pressure tracing, which resolved with further fluid boluses. No central venous pressure monitoring was used because of the patient's stable hemodynamics and the ability to monitor intraoperatively the arterial blood pressure for changes in systolic pressure variation.

After 4 h of surgery, arterial blood gas revealed a pH of 7.28, PCO₂ of 34 mmHg, PO₂ of 214 mmHg, HCO₃⁻ of 15.9 mEq/l, and base deficit of 9.5 mEq/l. At that time, the patient's estimated blood loss was 1,000 ml, and the patient had received 5,000 ml 0.9 normal saline, 1,000 ml 5% albumin, and 2 units of packed red blood cells. Fifty-five milliequivalents of sodium bicarbonate was given intravenously. Thirty-seven minutes after sodium bicarbonate was given, repeat arterial blood gas was pH 7.32, PCO₂ 35 mmHg, PO₂ 209 mmHg, HCO₃⁻ 17.8 mEq/l, and base deficit 7.2 mEq/l.

After 8 h of surgery, the patient's estimated blood loss was 3,500 ml, and the patient had received 20 l 0.9 normal saline, 9 units of packed red blood cells, 1,750 ml 5% albumin and 2 units of fresh frozen plasma. Arterial blood gas values were: pH 7.16, PCO₂ 37 mmHg, PO₂ 154 mmHg, HCO₃⁻ 14.4 mEq/l, and base deficit 14.5 mEq/l. The patient continued to be hemodynamically stable. Because of concerns regarding the etiology of the worsening metabolic acidosis possibly indicating end organ hypoperfusion, serum electrolyte and lactic acid concentrations were measured, and were as follows: Na⁺, 144 mEq/l; K⁺, 3.3 mEq/l; Cl⁻, 128 mEq/l (normal 95-106 mEq/l); HCO₃⁻, 13.2 mEq/l; glucose, 89 mg/dl; arterial lactic acid 0.8 mEq/l (normal 0.5-2.2 mEq/l). The anion gap was found to be only 3 (normal 12 ± 2 calculated from Na⁺ - [Cl⁻ + HCO₃⁻]). The metabolic acidosis was diagnosed as a dilutional nonanion gap hyperchloremic metabolic acidosis resulting from the large volume of normal saline given during surgery and not from inadequate end organ perfusion. Shortly afterwards, surgery was completed and the patient was transferred to the intensive care unit (ICU).

On arrival to the ICU, a pulmonary artery (PA) catheter was inserted because of concern with continued postoperative fluid shifts. Initial pulmonary artery pressures were: pulmonary artery systolic, 21 mmHg; pulmonary artery diastolic, 7 mmHg; pulmonary capillary wedge pressure, 4 mmHg; cardiac output, 5.2 l/min; and systemic vascular resistance, 1,460 dynes · s⁻¹ · cm⁻⁵. Four hours after arrival in the ICU, the patient underwent hemodialysis with a bicarbonate and low potassium dialysate. No volume was removed with the dialysis. Arterial blood gas obtained after the conclusion of hemodialysis was: pH, 7.39; PCO₂, 30 mmHg; PO₂, 158 mmHg; base deficit, 4.8 mEq/l. Postdialysis electrolytes were: Na⁺, 140 mEq/l; K, 3.7 mEq/l; Cl⁻, 107 mEq/l; HCO₃⁻, 18 mEq/l. The patient had no further prob-

* Assistant Professor, Department of Anesthesia.

† Professor, Department of Surgery.

From the Departments of Anesthesia and Surgery, The Bowman Gray School of Medicine of Wake Forest University, Winston-Salem, North Carolina. Submitted for publication July 12, 1996. Accepted for publication October 18, 1996.

Address reprint requests to Dr. Mathes: Department of Anesthesia, Bowman Gray School of Medicine, Medical Center Blvd., Winston-Salem, North Carolina 27157-1009.

Key words: metabolic acidosis, anion gap acidosis, hyperchloremia, dilutional acidosis.

lems with metabolic acidosis and was subsequently discharged home 9 days after surgery.

Discussion

Dilutional acidosis occurs when the plasma bicarbonate concentration is decreased by extracellular volume expansion with solutions that contain neither acid nor alkali.⁴ The actual total amount of extracellular bicarbonate does not decrease with rapid volume expansion; rather, it is simply decreased in concentration.^{2,3} In our patient, the infusion of large volumes of isotonic normal saline caused a hyperchloremic dilutional metabolic acidosis with a decreased anion gap.

Many authors have concluded, after studying dilutional acidosis in dogs, that rapid extracellular volume expansion caused only minimal decreases in measured extracellular bicarbonate due to cellular buffering.²⁻⁴ Those authors found that with rapid volume expansion, total extracellular bicarbonate actually increased because of the release of bicarbonate from intracellular and bone sources. Therefore, this increase in total extracellular bicarbonate prevented any significant decrease in measured bicarbonate or *pH* concentrations. In contrast to these authors' findings, our case report reveals that the normal buffering effect of increasing extracellular bicarbonate with volume expansion is inadequate to prevent a significant decrease in the measured bicarbonate concentration and *pH*.

We believe that dilutional acidosis is a genuine clinical entity, as shown by a marked hyperchloremic nonanion gap acidosis with a normal lactic acid concentration seen in our patient at the end of surgery. Another potential cause of this patient's metabolic acidosis was her preexisting renal failure, resulting in an increased retention of inorganic and organic acids. This is unlikely, because the patient had a normal preoperative bicarbonate and continued peritoneal dialysis until the morning of surgery. In addition, an increase in inorganic or organic acids would also have caused an increase in the calculated anion gap.

This case is unique for dilutional acidosis given the patient had end-stage renal disease and underwent a bilateral nephrectomy. Of interest, Garella *et al.*⁵ found that, in dogs with normal renal function, acute volume expansion caused increased renal bicarbonate wasting. The decreased bicarbonate reabsorption occurred because of increased sodium diuresis and decreased reabsorption of solute in the proximal tubule, including bicarbonate. Therefore, patients with normal renal func-

tion may have the potential for worsening dilutional acidosis with acute volume expansion compared with our patient without kidney function. However, we believe the kidney has very little time to contribute to or prevent dilutional acidosis in the acute setting. This conclusion also was made by Rosenbaum *et al.*,² who studied dilutional acidosis in nephrectomized dogs.

Despite the large amount of normal saline, albumin, and blood products, the patient had no evidence of increased intravascular volume, as suggested by a continued need for additional volume administration to correct the systolic pressure variation on the arterial waveform with unchanging positive pressure ventilation and the low filling pressures present in the ICU immediately after surgery.⁵ This indicates that dilutional acidosis can occur in the presence of intravascular volume depletion. Bicarbonate and chloride are measured per liter of extracellular volume. Intravascular volume only makes an average of 25% of the extracellular volume and becomes a smaller percentage of extracellular volume with increasing third space loss into the interstitial tissues. Therefore, dilutional acidosis can occur in the perioperative setting with large third space losses and intravascular volume depletion.

We do not believe that dilutional acidosis needs to be treated with sodium bicarbonate. Total body extracellular bicarbonate actually increases, even though the measured bicarbonate is decreased due to the extracellular expansion. Rosenberg *et al.*² found no change in the intracellular *pH* with a large increase in extracellular volume expansion. Therefore, dilutional metabolic acidosis is likely not harmful. Normally, dilutional acidosis will correct itself as the excess extracellular volume is reabsorbed and diuresed and bicarbonate remains in the extracellular space. In our patient, hemodialysis corrected the dilutional effect.

Surgeons and anesthesiologists may interpret blood gas data that indicate worsening metabolic acidosis and an increasing base deficit as a marker of end organ hypoperfusion. However, the perioperative physician must be aware that dilutional acidosis can cause these same changes. This case stresses the importance of obtaining additional values, including calculating the anion gap from Na^+ , Cl^- , and HCO_3^- and measuring lactic acid concentration. Although our patient had progressive severe metabolic acidosis, the decreased anion gap, stable hemodynamics, and normal lactic acid at the end of surgery indicated that this patient had adequate end organ perfusion. Her worsening acidosis was due to the

CASE REPORTS

large volume of normal saline and albumin administered.

References

1. Goodkin DA, Raja RM, Saven A: Dilutional acidosis. *South Med J* 1990; 83:354-5
2. Rosenbaum BJ, Makoff DL, Maxwell MH: Acid-base and electro-

lyte changes induced by acute isotonic saline infusion in the nephrectomized dog. *J Lab Clin Med* 1969; 74:427-35

3. Garella S, Tzamaloukas AH, Chazan JA: Effect of isotonic volume expansion on extracellular bicarbonate stores in normal dogs. *Am J Physiol* 1973; 225:628-36

4. Garella S, Chang BS, Kahn SF: Dilutional acidosis and contraction alkalosis: Review of a concept. *Kidney Int* 1975; 8:279-83

5. Perel A, Pizov R, Cotev S: Systolic blood pressure variation is a sensitive indicator of hypovolemia in ventilated dogs subjected to graded hemorrhage. *ANESTHESIOLOGY* 1987; 67:498-502

Anesthesiology
1997; 86:503-5
© 1997 American Society of Anesthesiologists, Inc.
Lippincott-Raven Publishers

Cardiac Arrest during Segmental Thoracic Epidural Anesthesia

Kevin K. M. Chan, M.D.,* Kathleen J. Welch, M.D., M.P.H.†

AT our institution, thoracic epidural anesthesia (TEA) is being used with increasing frequency to provide surgical anesthesia for breast surgery.¹ Until recently, we have not seen any morbidity associated with this technique. Although profound bradycardia and asystole after spinal anesthesia have been described,²⁻¹⁰ to our knowledge, there has been no clinical report describing such cardiovascular event after TEA. Therefore, we report a case of postoperative bradycardia and asystole complicating TEA performed on a healthy woman for surgical removal of bilateral breast tissue expanders.

Case Report

A 35-yr-old, 60-kg, 165 cm woman, American Society of Anesthesiologists (ASA) physical status 1, underwent removal of bilateral breast tissue expanders and replacement with saline implants. Before this admission, she had undergone bilateral modified radical mastectomy and insertion of tissue expanders during TEA. Her perioperative course was uneventful. She requested TEA again for this procedure. On the morning of admission to the day surgery unit, her physical

examination was normal, but she was anxious. After insertion of an intravenous catheter, she received 100 µg fentanyl and 2 mg midazolam intravenously. With the patient in the sitting position, an epidural catheter was inserted at the T5-6 level with loss of resistance technique. Lidocaine 2% with 1:200000 epinephrine was given in incremental doses of 2 ml to a total of 12 ml. A surgical level of sensory anesthesia was obtained from C8 to T10. During the surgery, she was comfortable and hemodynamically stable. Intraoperatively, she was sedated with 150 µg fentanyl and 2 mg midazolam intravenously. To maintain surgical anesthesia, a total of 7 ml of 2% lidocaine with 1:200000 epinephrine was given in incremental doses throughout the 1-h operation. The epidural catheter was removed at the end of surgery without any complications. On arrival at the postanesthesia care unit, she was awake, relaxed, and comfortable. Vital signs were: blood pressure 102/55 mmHg, heart rate 65 beats/min in normal sinus rhythm, respiratory rate 12 breaths/min, and hemoglobin oxygen saturation of 96% on room air. The sensory level extended from T2 to T10. The patient was placed in a semi-sitting position. Ten minutes later, she complained of nausea; vital signs were still stable. Ondansetron (4 mg in 100 ml normal saline) was given intravenously over 15 min. Approximately 15 min later, she again complained of nausea. The postanesthesia care unit nurse noticed that heart rate had suddenly decreased from the 60s to the 40s. The cardiac rhythm was sinus bradycardia in the 40s for approximately 1 min, during which time blood pressure was normal. Then, she abruptly became unresponsive and pulseless, with 45 s of asystole on the electrocardiograph. Cardiopulmonary resuscitation was promptly started, and 1 mg atropine was administered intravenously. She responded immediately to treatment, with return of sinus rhythm of 75 beats/min, a stable blood pressure of 110/60 mmHg, and a respiratory rate of 12 breaths/min. Repeat physical examination was unremarkable; the patient was calm and hemodynamically stable. Neurologic examination revealed residual sensory blockade from T2 to T10 level 1 h after the last dose of local anesthetic had been administered *via* the epidural catheter. She was admitted overnight for close observation. No dysrhythmia or myocardial ischemia had been documented before or after the cardiac arrest. However, postoperative 24-h Holter moni-

* Resident in Anesthesiology.

†Instructor in Anesthesiology.

Received from the Department of Anesthesia, Brigham and Women's Hospital and Harvard Medical School, Boston, Massachusetts. Submitted for publication May 30, 1996. Accepted for publication October 25, 1996.

Address reprint requests to Dr. Welch: Department of Anesthesia, Brigham and Women's Hospital, 75 Francis Street, Boston, Massachusetts 02115.

Key words: epidural; cardiac arrest; bradycardia; thoracic epidural.