Laparoscopic cholecystectomy in a patient with distal renal tubular acidoses associated with Sjogren’s syndrome: An anesthetic challenge

Madam,

Distal renal tubular acidoses (Distal-RTA) is a condition of chronic metabolic acidoses. Creation of pneumoperitoneum in these patients aggravates acidoses by increasing the P$\text{CO}_2$ in blood gases. Hence, we report here a rare combination of pneumoperitoneum in a patient with partially compensated distal-RTA.

We describe here the case of a 26 Y/F (Wt-55 kg, Ht-148 cm) with cholelithiasis having a past history of two episodes of quadriplegia 4 years back (6 months apart) secondary to hypokalemia. During both the episodes, she was managed medically and fully recovered. She was diagnosed to have type-1 RTA following the diagnostic workup for hypokalemia. She satisfied the “Sjögren’s International Collaborative Clinical Alliance” (SICCA) criteria for Sjogren’s syndrome. She was being treated with tablet sodium bicarbonate, tablet hydroxychloroquine, potassium chloride syrup, and tablet pilocarpine. Her serum potassium level was well maintained >3 mEq/L and she was completely asymptomatic. Currently, she presented with symptomatic gallstone disease and was posted for laparoscopic cholecystectomy. Preoperative blood investigations, chest X-ray, and electrocardiogram were within normal limits. All drugs were continued.

On the day of surgery, preoperative arterial blood gas (ABG) analysis showed; pH-7.42, PaCO$_2$ - 26.7 mmHg, PaO$_2$ 93.8 mm Hg, HCO$_3$- 15.4 mmol/L, BE- 7.7, SpO$_2$ 99%, Na$^+$-134.8 mEq/L, K$^+$-3.17 mEq/L, ionized Ca$^{2+}$-1.06 mmol/L, and Lactate- 2.01 mmol/L. As the pH was compensated to normal, we proceeded with elective surgery. In the operation theater, standard ASA monitors were attached. The patient was induced with injection morphine 7.5 mg and injection propofol 1–2 mg/kg (titrated to loss of verbal response), and injection atracurium 30 mg was used to facilitate tracheal intubation. Anesthesia was maintained with propofol infusion @ 50–70 mcg/kg/min and O$_2$:N$_2$O (40%) mixture. Ventilation was adjusted to maintain end-tidal carbon-di-oxide (EtCO$_2$) to 26 to 27 mmHg. Warming blankets and warm fluids were used to prevent hypothermia, and a total of 700 ml Ringer’s lactate solution was used intraoperatively. The surgery lasted for 70 min and was uneventful. ABG before extubation showed; pH-7.4, PaCO$_2$-28.2 mmHg, PaO$_2$-212.1 mmHg, HCO$_3$-15.4 mEq/L, BE-6.9 mmol/L, SpO$_2$-98.7%, Na$^+$-135.2 mEq/L, K$^+$-3.51 mEq/L, ionized Ca$^{2+}$-1.09 mmol/L, Lactate-2.01 mmol/L, and Hct-36. At the end of surgery, neuromuscular blockade was reversed using neostigmine 2.5 mg, and glycopyrrolate 0.5 mg and trachea was extubated successfully without any residual muscle weakness.

To maintain blood pH, acid-base homeostasis involves the balance of carbonic acid (H$_2$CO$_3$), bicarbonate ion (HCO$_3^-$), and carbon dioxcide (CO$_2$). CO$_2$ reacts with water (H$_2$O) to form (H$_2$CO$_3$), which rapidly dissociates to form HCO$_3^-$ and a hydrogen ion (H$^+$) as shown in the following equation, H$_2$O + CO$_2$ $\rightleftharpoons$ H$_2$CO$_3$ $\rightleftharpoons$ H$^+$ + HCO$_3^-$. This reaction is catalyzed by carbonic anhydrase enzyme.[1]

In distal RTA, there is increased luminal membrane permeability to H$^+$. Impaired H$^+$ ion secretion results in the shift of balance toward left resulting in decreased intracellular HCO$_3^-$ and metabolic acidoses (normal anion gap acidoses). To maintain electrical neutrality, K$^+$ secretion is proportionately increased. Hypokalemia (<2 mEq/L) potentiates the neuromuscular blockade, causes muscle paralysis, respiratory arrest, and arrhythmias.[2,4] The respiratory system compensates for low pH by hyperventilation that decreases the pressure of arterial CO$_2$. Distal RTA is mostly caused by autoimmune disorders such as Sjogren’s syndrome and rheumatoid arthritis.[12,5] High autoantibody titeres in Sjogren’s are directed against carbonic anhydrase type II, which can impair the intracellular production of H$^+$ and hence its secretion.

The primary goal of the anesthesiologist in the case of chronic metabolic acidoses is to maintain the intraoperative acid-base balance to ensure early extubation. Patients with RTA have a chronically low pH with low bicarbonate ions and low partial pressure of arterial CO$_2$(PaCO$_2$). Laparoscopic surgeries become a challenge here as there is an increase in PaCO$_2$ that aggravates the acidoses. Acute acidoses leads to the shift of potassium from the intracellular compartment to the intravascular compartment, which may result in alarming hyperkalemia in a well-optimized patient.[6]

Hence, ventilation should be titrated to maintain PaCO$_2$ around the pre-induction baseline values. Rapid correction of chronic metabolic acidoses is not advised intraoperatively during surgery as it can take away respiratory drive. Intraoperative hypothermia also aggravates acidoses by compromising peripheral circulation and should be avoided. Serum K$^+$ levels should be monitored to avoid both hypo and hyperkalemia. Non-steroidal anti-inflammatory drugs should be avoided as they may aggravate distal RTA. The dosage of long-acting opioids should be titrated closely as excessive sedation also increases PaCO$_2$.
Proper precautions to avoid hypothermia, strict vigilance with regard to ventilator settings avoiding both hypo and hyperventilation, maintenance of serum potassium levels, avoiding isotonic saline (prevent hyperchloremic metabolic acidosis), and avoiding the use of nephrotoxic drugs is the key for successful intraoperative management of a patient with distal RTA for laparoscopic surgeries.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest
There are no conflicts of interest.

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References
1. Rodríguez Soriano J. Renal tubular acidosis: The clinical entity. J Am Soc Nephrol 2002;13:2160.

2. Rose BD, Post TW. Clinical Physiology of Acid-Base and Electrolytes Disorders. 5th ed. New York: McGraw-Hill; 2001. p. 616.
3. Sebastian A, McSherry E, Morris RC Jr. Renal potassium wasting in renal tubular acidosis (RTA): Its occurrence in types 1 and 2 RTA despite sustained correction of systemic acidosis. J Clin Invest 1971;50:667.
4. PouxJM, Peyronnet P, Le MeurY, Favereau JP, Charmes JP, Leroux-Robert C. Hypokalemic quadriplegia and respiratory arrest revealing primary Sjogren's syndrome. Clin Nephrol 1992;37:189-91.
5. Battle D, Haque SK. Genetic causes and mechanisms of distal renal tubular acidosis. Nephrol Dial Transplant 2012;27:3691.
6. Srivastava A, Niranjan A. Secrets of safe laparoscopic surgery: Anaesthetic and surgical considerations. J Minim Access Surg 2010;6:91.

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