

Regional anesthesia is safe and effective for lower limb orthopedic surgery in patient with renal tubular acidosis and hypokalemia

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Abstract

Renal tubular acidosis (RTA) with hypokalemia may precipitate acute respiratory failure and potentially fatal arrhythmias like ventricular fibrillation. Though there are random reports of respiratory failure needing mechanical ventilation and sudden death in patients with RTA and hypokalemia, the anesthetic management of these patients has not been clearly elucidated. Acidosis and hypokalemia have significant interactions with both general and local anesthetics and alter their effect substantially. Proper preoperative planning and optimization are required for the safe conduct of anesthesia in this subset of patients. We describe a case of distal RTA, hypokalemia, and metabolic bone disease in whom central neuraxial anesthesia was effectively used for lower limb orthopedic surgery with no complications.

Keywords: Combined spinal epidural anesthesia, hypokalemia, renal tubular acidosis

Introduction

Renal tubular acidosis (RTA) is characterized by normal anion gap hyperchloremic acidosis and occurs either due to decreased reabsorption of filtered HCO_3^- by the proximal convoluted tubule (type 2 or proximal RTA) or decreased H^+ excretion in the distal nephron (type 1 or distal RTA). RTA is associated with normo- hypo- or hyperkalemia. Both acidosis and hypokalemia can significantly alter the pharmacokinetics and -dynamics of local anesthetics (LA). Though there are reports of patients with RTA and hypokalemia causing life-threatening respiratory failure in literature, the use of regional anesthesia in these patients has not been reported.

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Case Report

A 28-year-old female patient presented to the orthopedic department with pain in left lower limb after trivial trauma. Past history was significant for repeated episodes of muscle weakness. At that time, she was diagnosed to have hypokalemic periodic paralysis (HPP) and started on oral potassium supplementation. Her physical examination was normal except for subtrochanteric fractures of bilateral femora. There was no muscle weakness of both upper limbs. The power in the lower limbs could not be tested due to fractures of both femora. On investigation, serum creatinine was 1.4 mg/dl, BUN 26 mg/dl, S.Na^+ - 133 mmol/L, S.K^+ 2.7 mmol/L, S.Ca^{++} 9.7 mg/dl, S.phosphorus 2.1 mg/dl, uric acid 2 mg/dl, S.bilirubin 0.5 mg/dl, SGOT 42 U/L, SGPT 23 U/L, S.albumin 3.2 mg/dl, and alkaline phosphatase 837 mg/dl. Her Vitamin D3 was 10

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ng/ml and S.PTH 38.5 pg/ml. Her thyroid function tests were normal. Analysis of arterial blood gases (ABGs) (on room air) revealed pH of 7.22, PaO₂ of 61, PCO₂ of 23, HCO₃ of 11.3 with base deficit of - 17 mmol/L, Na⁺ - 139 meq/L, K⁺ 2.3 mmol/L, Ca⁺⁺ 0.41 mmol/L, Cl⁻ - 122 mmol/L, and an anion gap of 8 meq/L. The urinary pH was 7.5. On radiological evaluation, there was an acute pathological subtrochanteric fracture of left femur, old subtrochanteric fracture of the right femur and left proximal fibula. On ultrasound abdomen, there was dystrophic parenchymal calcification in the left kidney and mild hydronephrosis and hydroureter on the right side. Tc-99m diethylene triamine penta acetic acid revealed nonfunctioning left kidney with the normally functioning right kidney. Electrocardiography (ECG) was normal except for frequent ventricular ectopics. Transthoracic echocardiography was normal. A bone scan showed avid uptake of tracer in right sixth rib, left seventh rib, bilateral proximal femora, left proximal fibula, shaft of the right humerus, and renal cortex [Figure 1].

Based on the above findings, the patient was diagnosed to have RTA (distal or type 1, normal anion gap hyperchloremic acidosis) with hypokalemia, nephrocalcinosis, and metabolic bone disease. Surgery was deferred in order to optimize the condition of the patient. The goal of therapy was to maintain serum pH of >7.3 and S.K⁺ >3 mmol/L. She was started on potassium supplementation intravenously (IV), followed by oral potassium 21 meq tid, tablet alfacalcidol 0.25 mcg, oral sodium bicarbonate 1 g (11.9 mmol) thrice a day, and oral calcium 500 mg BD. After 4 days, ABG analysis showed pH 7.307, PaO₂ 96 mmHg, PCO₂ 25 mm Hg, HCO₃ 12.3 mmol/L with base deficit of - 12.6 mmol/L, K⁺ 3.1 mmol/L, and Ca⁺⁺ 0.92 mmol/L. She was posted for interlocking nailing of fracture of left femur to be followed by right femur after 2 days.

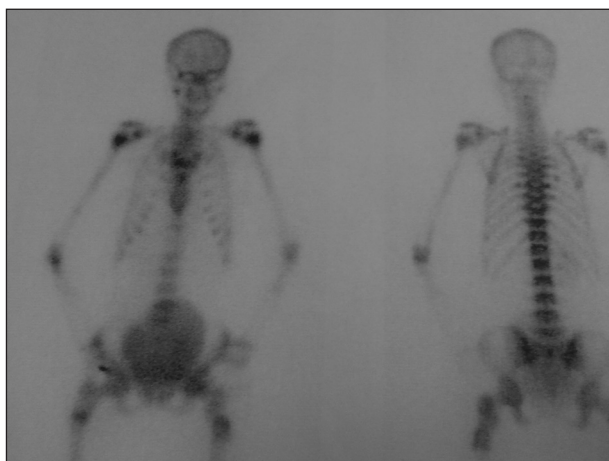


Figure 1: Bone scan of the patient consistent with metabolic bone disease and associated insufficiency fractures

On the day of surgery, she was premedicated with tablet alprazolam 0.25 mg and tablet pantoprazole 40 mg 2 h prior to shifting to the operation theatre (OT). In the OT, standard monitors were attached and wide bore IV access was secured. Radial arterial cannulation was done for continuous blood pressure monitoring and ABG analysis. Under strict aseptic precautions, epidural space was located in sitting position at L3-4 intervertebral space through a median approach by the loss of resistance to air with an 18G Tuohy needle. A subarachnoid block was established with the administration of 3 ml of 0.5% heavy bupivacaine with 25 µg of fentanyl via 27G spinal needle (needle through needle technique). A 22G epidural catheter was passed into the epidural space and fixed at 9 cm. The time taken for onset of the subarachnoid block was approximately 5-6 min and the highest level of the block (T8) was achieved within 13 min. During the procedure, which lasted for 120 min, 1.5 L of ringer lactate was infused and normal saline was avoided. IV sodium bicarbonate and potassium supplementation were given based on ABG findings so as to maintain pH of more than 7.3 and >3 -5 mEq/L. A total of 50 meq of 7.5% sodium bicarbonate and 10 meq of potassium chloride were infused. Intraoperative vitals were stable, blood loss was approximately 300 ml, and urine output was 20-40 ml/h. For postoperative analgesia, a bolus of 8 ml of 0.2% ropivacaine (after a test dose of 3 ml of 2% lignocaine without epinephrine) was given epidurally and an infusion of 0.2% ropivacaine was started at 6 ml/h. The operation on the contralateral femur was done under epidural anesthesia 2 days later. Postoperatively, ECG was continuously monitored and ABG analysis was done twice a day for 3 days. Oral bicarbonate and potassium supplements were continued and additional IV supplementation was given as required. She was discharged on the 10th postoperative day.

Discussion

Distal RTA (type 1) is caused by impaired distal acidification and inability to lower urinary pH maximally. It is either inherited or acquired and often associated with hypokalemia and nephrocalcinosis. The main concerns of medical therapy are to protect the bones and reduce the risk of nephrocalcinosis. Treatment includes alkali therapy, K⁺ and Vitamin D supplementation, and long-term follow-up for renal stone complications.

Acidosis and hypokalemia which accompany RTA have pharmacodynamic and kinetic interactions with both general anesthetics (GA) and LA. Acidosis causes exaggerated hypotensive responses to GA (both inhalational and IV) and positive pressure ventilation.^[1] Acidosis and hypokalemia delay

recovery from neuromuscular blockade with increased risk of postoperative residual paralysis.^[2] With neuraxial techniques, central nervous depression, and muscle weakness due to GA and neuromuscular blockade are avoided. Therefore, combined spinal epidural anesthesia was preferred over GA in our patient.

Patients with acidosis can become hemodynamically unstable.^[3] The systemic vascular resistance (SVR) may decrease if pH is sufficiently reduced.^[4] Interaction of acidosis and subarachnoid anesthesia has potential to cause a deleterious decrease in SVR and result in profound hypotension. Acidosis interferes with the action of inotropes and the response to vasopressors may be suboptimal. Hence, we aimed at correction of pH to >7.3. With correction and maintenance of pH >7.3, institution of SA did not cause significant fall in blood pressure in our patient and hemodynamic instability was not observed at any stage of the perioperative period.

Acidosis increases the potential for central nervous system toxicity due to decreased protein binding of LA.^[5,6] The hyperdynamic circulation accompanying acidosis may increase the cerebral blood flow and delivery of LA to the brain and thereby enhance the toxic effects of LA.^[7] As the potential for toxicity is more with bupivacaine, low dose (0.2%) ropivacaine infusion with fentanyl was administered epidurally for postoperative pain relief. Cellular hypoxia due to tourniquet results in increased lactate production which may aggravate acidosis.^[8] Though this is of little concern in healthy patients, it may produce clinically significant acidosis in patients with underlying acidosis. Hence, the use of tourniquet must be done judiciously in RTA. In the perioperative period, lactated ringer was preferred over normal saline infusion as the latter is found to cause hyperchloremic acidosis.^[9,10]

Hypokalemia decreases the threshold for arrhythmias. Ventricular fibrillation and sudden cardiac death have been reported in RTA.^[11] Severe hypokalemia due to RTA may cause bulbar, respiratory and limb weakness and necessitate emergency intubation, and mechanical ventilation.^[12] Our patient had previous episodes of profound muscle weakness which were treated as HPP. It is important to differentiate hypokalemia due to RTA from HPP as the treatment of the latter with acetazolamide may aggravate acidosis and is contraindicated in RTA. Correction of acidosis with bicarbonate aggravates hypokalemia. To avoid the exacerbation of hypokalemia, our patient was started on K⁺ supplementation, which was followed by a HCO₃ correction. The K⁺ supplementation was continued perioperatively and

continuous ECG monitoring was done for early detection of arrhythmias. Epidural anesthesia has been found to decrease serum potassium by 0.3-0.7 mmol/L.^[13] This has been attributed to epinephrine co-administered with LA. Hence, the addition of epinephrine in the epidural test dose was avoided.

Conclusion

Renal tubular acidosis with hypokalemia may result in acute complications such as respiratory failure and ventricular fibrillation. The anesthetic management and its implications in this group has not been clearly elucidated. We found that with correction and maintenance of pH with normal levels of serum potassium, ensure safe and effective central neuraxial anesthesia for lower limb orthopedic surgery with no complications.

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