CASE REPORT

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Anesthesia management of children with renal tubular acidosis

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Abstract

The primary distal renal tubular acidosis is characterized biochemically by the inability of the kidney to produce appropriately acid urine in the presence of systemic metabolic acidosis or after acid loading. 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 the perioperative management of two cases of renal tubular acidosis.

Keywords: Renal tubular acidosis, Anesthesia, Metabolic acidosis , Hypokalemia

INTRODUCTION

Renal tubular acidosis (RTA) refers to a group of disorders characterized by defective tubular acid-base regulation. As a result, there is a dysfunction in urinary acidification, resulting in net acid retention and hyperchloremic metabolic acidosis.

These cases demonstrate the perioperative management of two children who are known to be suffering from an RTA and who are proposed to undergo a chochlear-implant surgery.

CASE REPORT

Case 1: AA is a 5 years-old boy who has been diagnosed as distal renal tubular acidosis and congenital bilateral surdity at the age of 2 years. The patient was under oral sodium bicarbonate and potassium supplementation. The physical examination of the patient is normal (body temperature is 37.1, blood pressure is 121/73 mmHg, a heart rate is 90 beats per minute, a respiratory rate is 16 breaths per minute). The laboratory results were in the normal range (blood count, coagulation parameters and renal function: NA+: 140 mmol/l; K+: 3.5; CL-: 106 mmol/l). The arteriel blood gases (ABG) (room air) was pH: 7.39; Pco2: 34.2 mmhg; Po2: 96 mmhg; HCO3- : 20.8 mmol/l; BE: - 3.5 mmol/l, Saturation: 97 %.

For the preoperative preparation, the patient was put under sodium bicarbonate 0,42 % a total of 40 mEq /24h and 3 mEq/kg/24h of potassium chloride on a continuous perfusion a day before the surgery. Anesthesia is assured by continuous perfusion of propofol + remifentanil and bolus of cisatracurium. The ventilation parameters were adjusted so PetCo2 doesn't exceed 35 mmhg. The surgery took 1 hour and 40min. We continued the protocol of acid-base management during all the surgery. No peri operative incident was noticed. An immediat postoperative arterial blood gases was taken and it showed pH: 7.22; PCO2: 45 mmhg; HCO3- : 20.1 mmol/l; PO2: 107 mmhg; Saturation: 99 %. The same protocol of acid-base management was continued until the oral was authorized.

Case 2: HM is a 7 years-old girl diagnosed as distal renal acidosis and bilateral surdity since the age of one year. Since then she is taking oral bicarbonate and potassium supplementation. The physical examination is normal. The ABG on the admission: pH:7.49; PCO2: 27 mmhg; HCO3- : 20.6 mmol/l; PO2: 123 mmhg; SaO2 :99 %. The laboratory results were normal. The patient went through the same acid-base management protocol as the child AA. She also had the same anesthetic protocol. The surgery took 2 hours. No incidents were noticed. The post-operative ABG showed: pH: 7.28; PCO2: 44 mmhg PO2:180 mmhg; HCO3-: 20.7 mmol/l; Saturation: 99 %.

DISCUSSION

RTA is classified into 3 major groups which are different in mechanism. RTA can be occurred due to decreased

reabsorption of filtered HCO3 by the proximal convoluted tubule (type 2 or proximal RTA) or decreased H+ excretion in the distal nephron (type 1 or distal RTA) or aldosterone deficiency or resistance (type 3 or hyperkalemic RTA).[1,2].

Perioperative management of RTA poses two main problems: the perioperative acid-base management and the interaction of metabolic acidosis with anesthetic products and mechanical ventilation.

Renal tubular acidosis (RTA) is characterized by normal anion-gap metabolic acidosis, originating from excessive urinary loss of bicarbonate or defective urinary acidification [1]. Therefore, unlike high anion-gap acidoses (e.g., lactic acidosis or ketoacidosis), RTA must be treated with administration of sodium bicarbonate. However, perioperative acid-base management in such cases poses many difficulties because the total carbon dioxide (CO2) content in blood as well as actual blood pH must be fully taken into consideration for successful weaning from mechanical ventilation. 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.

Literature reviews of anesthetic management of patients with severe acute metabolic acidosis have focused on the maintenance of adequate blood pressure and tissue perfusion. However, in chronic cases, anesthesiologists should give attention to intraoperative acid-base status in order to ensure adequate spontaneous breathing immediately after surgery. In general, respiratory drive is stimulated by low pH, particularly in patients with chronic metabolic acidosis. Therefore, any arterial pH, which is higher than preoperative value, can be associated with a higher risk for postoperative hypoventilation to various degrees, even if normal acid-base balance without hypercapnia has been achieved at the time of weaning from mechanical ventilation. Rapid or excessive correction of chronic metabolic acidosis during surgery leads to either life-threatening hypoventilation or sudden onset of apnea [3].

Acidosis and hypokalemia which accompany RTA have pharmacodynamic and kinetic interactions with both general anesthetics (GA) and Local anesthetics. Acidosis causes exaggerated hypotensive responses to GA (both inhalational and IV) and positive pressure ventilation [4]. Acidosis and hypokalemia delay recovery from neuromuscular blockade with increased risk of postoperative residual paralysis [5].

Hypokalemia decreases the threshold for arrhythmias. Ventricular fibrillation and sudden cardiac arrest have been reported in RTA [6]. Severe hypokalemia due to RTA may cause bulbar, respiratory and limb weakness and necessitate emergency intubation, and mechanical ventilation [7].

If a urachal anomaly is not resected, the family and patient should be made aware of the potential future risk of malignancy in the future and the need for lifelong screening [5].

CONCLUSION

RTA is a rare but serious pathology. The management of anesthesia in this situation is based on a good knowledge of the biochemical changes associated with this pathology.

Conflict of Interest

None declared

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