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Late-Onset Rebound Hyperkalemia after Ritodrine Cessation in a Morbidly Obese Parturient During Emergency Surgery for Postpartum Hemorrhage

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Abstract **Case Report**

Ritodrine, a beta-2 adrenergic agonist used for tocolysis, is known to cause hypokalemia during administration, while its discontinuation can occasionally lead to "rebound hyperkalemia" due to the extracellular shift of potassium. Although this rebound effect typically occurs within 2 to 3 hours, the timing and pattern can vary based on the patient's clinical context. We report a case of delayed rebound hyperkalemia in a 34-year-old morbidly obese parturient (BMI 39.1 kg/m²) who presented with placenta accreta and massive hemorrhage following vaginal delivery. Approximately 5.5 hours after ritodrine cessation, during emergency surgery and resuscitation, her serum potassium level was found to be elevated at 6.2 mEq/L, eventually peaking at 7.0 mEq/L at the 7-hour mark. This delayed manifestation may be attributed to several factors, including the reduced extracellular fluid volume and impaired insulin sensitivity associated with morbid obesity, prior potassium supplementation, and the physiological stress of hemorrhagic shock and metabolic acidosis. Despite the severe hyperkalemia, the patient was successfully managed with insulin-glucose therapy and achieved hemodynamic stability after a subtotal hysterectomy. This case underscores the importance of extended electrolyte monitoring for up to 6 to 8 hours after ritodrine discontinuation, especially in high-risk parturients with confounding factors such as obesity or massive hemorrhage, to ensure timely intervention for delayed potassium fluctuations.

Keywords: Ritodrine, rebound hyperkalemia, ritodrine cessation, postpartum hemorrhage.

INTRODUCTION

Ritodrine is a beta-2 adrenergic agonist utilized for tocolysis to suppress preterm labor. Its primary mechanism of action involves the activation of the cell membrane-bound sodium-potassium adenosine triphosphatase pump, which facilitates the intracellular influx of potassium ions. Consequently, hypokalemia may manifest during drug administration [1]. Conversely, although rare, several cases have reported the occurrence of "rebound hyperkalemia" upon discontinuation of the medication, caused by the redistribution of intracellular potassium back into the extracellular fluid. While existing literature indicates that such hyperkalemic episodes are typically observed within 2 to 3 hours following cessation [2, 3], the timing of onset and the pattern of electrolyte fluctuations may vary depending on the patient's clinical context, with some reports extending up to 5 hours post-discontinuation [4].

The authors encountered a case of rebound hyperkalemia identified approximately 5.5 hours after ritodrine discontinuation in a morbidly obese parturient. This delayed rebound hyperkalemia, which occurred during the management of massive hemorrhage and emergency surgery due to placenta accreta following vaginal delivery, may serve as a confounding factor in the differential diagnosis of electrolyte imbalances following ritodrine cessation. Herein, we report this case of delayed potassium fluctuations following the discontinuation of ritodrine and provide a review of the relevant literature.

CASE REPORT

The study protocol was reviewed and approved by the Institutional Review Board of Presbyterian Medical Center, which waived the requirement for informed consent due to the retrospective nature of the medical record review (IRB No. PMC 2026-04-009).

A 34-year-old parturient (34 weeks and 6 days of gestation; height: 163 cm; weight: 104 kg; body mass index: 39.1 kg/m^2) was transferred to our institution due to the progression of preterm labor. Prior to admission, she had been receiving tocolytic therapy with ritodrine and potassium chloride-supplemented intravenous fluids at a referring hospital. Upon presentation, her serum potassium level was 3.9 mEq/L. As labor continued to progress, the decision was made to proceed with vaginal delivery, and ritodrine administration was subsequently discontinued. Fetal expulsion was successfully completed approximately 2 hours and 30 minutes after the cessation of ritodrine, and 100 micrograms of carbetocin was administered to facilitate uterine contraction.

Following delivery, persistent vaginal bleeding occurred due to placenta accreta, with the placenta failing to detach completely. Initially, the bleeding was manageable, and the patient was closely monitored; however, the volume of blood loss rapidly increased, necessitating emergency surgery. Immediately prior to transfer to the operating room, the patient's mental status became drowsy, with a blood pressure of 60/40 mmHg and a heart rate of 120 beats/minute. Upon arrival in the operating room, general anesthesia was induced. Given the inadequate fasting duration, rapid sequence induction was performed using 150 mg of propofol and 100 mg of rocuronium. Immediately after induction, the femoral pulse was only weakly palpable and non-invasive blood pressure measurement was unattainable; thus, 50 micrograms of epinephrine was administered intravenously. Simultaneously, arterial and right internal jugular central venous catheters were placed to facilitate rapid fluid resuscitation, resulting in hemodynamic stability.

Subsequent laboratory analysis, performed 5 hours and 30 minutes after ritodrine discontinuation, revealed a potassium level of 6.2 mEq/L. Concomitant metabolic acidosis was observed (pH 7.24, P_aCO_2 35 mmHg, HCO_3^- 15 mEq/L, and lactate 3.9 mmol/L). A follow-up test 50 minutes later showed slight improvement in the acidosis (pH 7.31, P_aCO_2 32 mmHg, HCO_3^- 16.1 mEq/L, and lactate 3.91 mmol/L), but the potassium level remained elevated at 6.4 mEq/L. After another 40 minutes, the potassium level reached 7.0 mEq/L, prompting the administration of insulin-glucose therapy. By 8 hours post-ritodrine cessation, the potassium level had normalized to 5.0 mEq/L.

Throughout these electrolyte fluctuations, the surgical procedure continued. Following a Pfannenstiel incision, the surgical team determined that separating only the placenta would be hazardous due to the severity of the hemorrhage. Consequently, with the guardian's consent, a subtotal hysterectomy was performed and completed successfully. Immediately after surgery, the patient regained consciousness and spontaneous respiration and was subsequently transferred to the general ward. The following day, the serum potassium level was 4.6 mEq/L.

DISCUSSION

This case demonstrates that the onset of rebound hyperkalemia following ritodrine cessation may be more delayed than previously described in the literature. While existing reports typically indicate that serum potassium levels peak within 2 to 3 hours after discontinuation [2, 3], a significant elevation was first identified at 5.5 hours in the present case, eventually reaching a peak concentration of 7.0 mEq/L at the 7-hour mark.

The primary factor contributing to this delayed manifestation may be the patient's morbid obesity (BMI 39.1 kg/m^2). Obese patients typically exhibit a relatively lower extracellular fluid volume compared to their total body weight, resulting in a reduced volume of distribution for potassium. Consequently, when intracellular potassium is redistributed into the extracellular compartment, the limited space available for dilution may lead to a more pronounced elevation in serum potassium levels compared to non-obese patients. Furthermore, the reduced insulin sensitivity often associated with morbid obesity may impair the compensatory mechanism of shifting extracellular potassium back into cells, potentially prolonging the duration of rebound hyperkalemia [5].

A second potential contributing factor is the administration of potassium chloride-supplemented fluids, which were given to prevent ritodrine-induced hypokalemia. Although notable changes in serum potassium concentrations typically occur within 1 to 2 hours of intravenous administration, several additional hours are required for intracellular potassium to reach complete equilibrium via renal excretion [6]. These dynamics are also influenced by the dosage and duration of both potassium and ritodrine administration. In the present case, since the patient was transferred to our institution on the day of surgery after receiving both medications at a referring hospital starting the day before admission, the exact cumulative dosage could not be accurately quantified.

Third, the physiological impact of massive hemorrhage and shock secondary to placenta accreta should be considered. Tissue hypoperfusion and acidosis resulting from acute blood loss create an environment that facilitates the extracellular efflux of potassium, potentially exacerbating hyperkalemia [5].

While blood transfusions or metabolic acidosis could also be considered as potential causes of hyperkalemia, the initial detection of hyperkalemia occurred before any substantial blood transfusion had taken place. Given the temporal progression of the serum potassium levels, we infer that the rebound shift of

potassium following ritodrine cessation is the primary underlying mechanism of the observed hyperkalemia.

Additionally, apart from the delayed onset, the prolonged duration of hyperkalemia—peaking at the 7-hour mark—was partly attributed to the delayed initiation of insulin-glucose therapy. At that time, the primary clinical focus was on managing acute massive hemorrhage and metabolic acidosis, which may have masked the clinical suspicion for ritodrine-induced rebound hyperkalemia. It was initially anticipated that the hyperkalemia would resolve spontaneously following hemodynamic stabilization and the correction of acidosis, which led to the delay in targeted treatment. Consequently, the duration of the hyperkalemic episode in the present case was more extended compared to previously reported cases where immediate therapeutic interventions were implemented. Fortunately, no serious arrhythmias were observed despite the severely elevated potassium levels.

CONCLUSION

For parturients who have received ritodrine therapy—particularly those presenting with confounding factors such as morbid obesity, massive hemorrhage, or prophylactic potassium administration—monitoring serum potassium levels for up to 6 to 8 hours after discontinuation should be considered. Such extended vigilance is crucial for the early detection and management of delayed rebound hyperkalemia, thereby ensuring patient safety in high-risk clinical scenarios.

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