Asploro Journal of Biomedical and Clinical Case Reports
(ISSN: 2582-0370)
Case Report
DOI: https://doi.org/10.36502/2023/ASJBCCR.6321

Transfusion-Related Hyperkalemia Causing Ventricular Arrhythmia in Neonatal Hepatectomy: A Critical Case Report

Xuewei Liu[1], Yuyi Zhao[1*]
[1]*Department of Anesthesiology, West China Hospital, Sichuan University, Chengdu, Sichuan, People's Republic of China*

Corresponding Author: **Yuyi Zhao**
Address: Department of Anesthesiology, West China Hospital, Sichuan University & The Research Units of West China (2018RU012), Chinese Academy of Medical Sciences, 37 Guoxuexiang, Chengdu, Sichuan 610041, China; Tel: +189 80606837; Email: zhaoyuyi@wchscu.cn
Received date: 16 August 2023; **Accepted date**: 25 August 2023; **Published date**: 29 August 2023

Citation: Liu X, Zhao Y. Transfusion-Related Hyperkalemia Causing Ventricular Arrhythmia in Neonatal Hepatectomy: A Critical Case Report. Asp Biomed Clin Case Rep. 2023 Aug 29;6(3):229-32.

Summary

Transfusion-related hyperkalemia leading to ventricular arrhythmia is rare in neonates. We report a case of a 9-day-old neonate who developed severe hyperkalemia and ventricular arrhythmia after receiving a packed red blood cell transfusion during hepatectomy. After successful resuscitation, further transfusion was required. Subsequently, hyperkalemia was prevented by infusing packed red blood cells with saline in a ratio of 2:1, based on careful calculation.

Keywords

Transfusion, Hyperkalemia, Ventricular Arrhythmia

Introduction

The blood volume of neonates is small, making them more susceptible to blood loss during surgery, which can result in hypovolemia and necessitate blood transfusions. However, the transfusion of blood products can give rise to various complications. Among these, a particularly perilous complication is blood transfusion-related hyperkalemia, which can lead to severe arrhythmia, cardiac arrest, and even death. Hence, it is crucial for pediatric anesthesiologists to be well-informed about adverse reactions associated with blood transfusions, with special attention to the potentially life-threatening transfusion-related hyperkalemia.

Case Presentation

A 9-day-old female infant (weight: 3.5 kg, height: 53 cm) presented with a highly vascular liver mass measuring 11×6.8 cm, and she was scheduled for a laparoscopic liver mass biopsy. Preoperative assessments showed a hemoglobin level of 152 g/L, hematocrit (HCT) of 0.46%, and a plasma potassium concentration of 4.54 mmol/L. In anticipation of potential significant blood loss during the surgery, we prepared packed red blood cells (PRBCs) and fresh frozen plasma (FFP).

Considering the potential impact on electrolyte balance and liver function, we opted for anesthesia agents with minimal implications. For anesthesia induction, we administered midazolam, propofol, fentanyl, and cis-atracurium at appropriate doses based on the baby's weight. Throughout the anesthesia maintenance phase, we utilized sevoflurane (1.5-2%), remifentanil (0.1-0.3 μg/kg/min), and cis-atracurium (bolus as required), all of which have limited potential

impact on electrolyte balance.

Following endotracheal intubation, we established a radial arterial line for continuous blood pressure monitoring and inserted a central venous catheter via the right internal jugular vein. Due to severe hemorrhage, the laparoscopic biopsy was converted into an open hepatectomy. Packed red blood cells (PRBCs), which were two weeks old, were infused at a rate of 80 ml/h (seventy minutes before the onset of arrhythmia). The transfusion was halted after 90 ml, and the plasma potassium level surged to 6.6 mmol/L (twenty-four minutes before arrhythmia).

Despite the absence of arrhythmia signs on the electrocardiogram (ECG), the heart rate suddenly plummeted from 130 to 67 bpm. The ECG displayed absent P waves, peaked T waves, and irregular wide QRS complexes. Blood pressure decreased to 51/39 mmHg, and oxygen saturation (SpO_2) dropped from 100% to 68%. Swift administration of intravenous epinephrine (5 μg), atropine (0.1 mg), and norepinephrine (2 μg) was initiated. An arterial blood gas analysis revealed a plasma potassium level of 9.0 mmol/L. Immediate and aggressive treatment for hyperkalemia was initiated (**Fig-1**), involving insulin (1.5 U), calcium gluconate (60 mg), 5% sodium bicarbonate (7 ml), and furosemide (0.7 mg). The ventricular arrhythmia persisted for approximately 2 minutes, during which the heart rate surged to 174 bpm while blood pressure reached 73/64 mmHg.

Around 20 minutes after the arrhythmia episode, the plasma potassium level dropped to 4.3 mmol/L, with the hemoglobin level at 119.7 g/L. However, significant bleeding persisted, resulting in an estimated blood loss of over 140 ml within 30 minutes. The infusion rate of norepinephrine was escalated from 0.05 μg/kg/min to 0.3 μg/kg/min, and repeated boluses of 5 μg norepinephrine were necessary to sustain stable circulation.

To address the unstable circulation and subsequent impaired tissue perfusion, we initiated PRBC infusion. To avert the development of hyperkalemia with subsequent PRBC transfusion, we assessed the potassium concentration of the stored PRBCs, revealing a concentration as high as 19 mmol/L. To mitigate this, PRBCs were infused with a 2:1 ratio of saline to decrease the potassium concentration [1]. After infusing 60 ml of PRBCs over 20 minutes, the infant's plasma potassium concentration decreased to

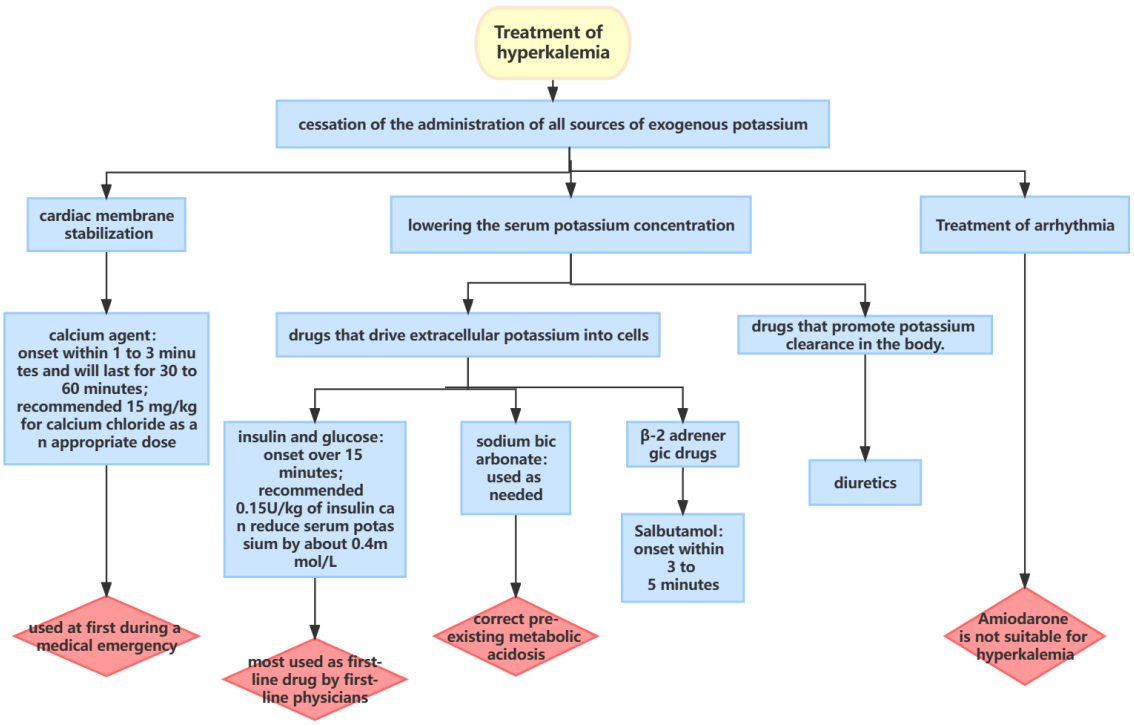

Fig-1: Treatment of Hyperkalemia

3.9 mmol/L, and her hemoglobin level was 113.6 g/L (forty-five minutes after the arrhythmia). The infant's hemodynamics remained stable, and the surgery concluded without further issues.

The entire operation lasted nearly 6 hours, with a total blood loss of 200 ml. A cumulative 150 ml of PRBCs was administered, along with 267 ml of crystalloid infusion. Following the procedure, the infant was transferred to the Pediatric Intensive Care Unit (PICU) and was discharged home after a 10-day post-surgery stay. The final pathological diagnosis for the removed liver mass was congenital hepatic hemangioma.

Discussion

The destruction of red blood cells during blood storage has been established as a cause for elevated potassium concentrations in stock blood products, with levels as high as 27.3±13.5 mmol/L confirmed [2]. This phenomenon accounts for the rise in the neonate's blood potassium from 4.5 to 6.6 mmol/L following our initial blood transfusion (the PRBCs had been stored for 2 weeks). Temporary post-transfusion elevation of potassium is a common occurrence, usually resolving when PRBC transfusion is halted. However, in this case, the serum potassium surged from 6.6 to 9.0 mmol/L within 24 minutes after ceasing the transfusion. This abrupt increase might be linked to exacerbated acidosis due to hypotension.

The metabolic acidosis and accumulation of lactic acid resulting from impaired tissue perfusion due to hypotension can accelerate the extracellular shift of potassium as a result of hydrogen ion exchange, thereby exacerbating hyperkalemia [3,4]. A study by Hall TL [5] indicated that elevated plasma potassium levels (5-7 mmol/L) can prolong the ECG P-R interval and the T wave ascent in the heart. Moreover, potassium concentrations surpassing 8 mmol/L could impede myocardial contraction and broaden the QRS complex. Prolongation of the P-R interval contributes to the rapid decline in heart rate. Nonetheless, slight to moderate P-R interval prolongation might not manifest as arrhythmias on the initial ECG. As the P-R interval continues to elongate, and the QRS complex becomes more prominent, it eventually progresses to Ventricular Arrhythmia.

This case underscores the imperative of aggressively treating hyperkalemia, even in the presence of a seemingly normal ECG. At times, ongoing hemorrhage necessitates transfusions despite the correction of hyperkalemia-induced ventricular arrhythmia, introducing a challenging scenario. To prevent recurrent hyperkalemia-induced ventricular arrhythmia, we employed the computational approach detailed in the study by Vraets A et al [1]. By considering the PRBC potassium level (19 mmol/L), the neonate's current serum potassium level (4.3 mmol/L), and the anticipated volume of required blood transfusion (60 ml), we projected the neonate's post-transfusion potassium level to be 6.3 mmol/L. Subsequently, we calculated the quantity of potassium-free saline needed for infusion alongside the PRBCs at a 2:1 ratio. This case serves as a reminder of the necessity to consistently assess the potassium levels of stored PRBCs and to concomitantly administer an appropriate amount of saline, especially in neonatal cases.

Tips

- Prior to transfusion, assess both the potassium concentration and hematocrit in the stored PRBCs.
- Hyperkalemia has various treatment options; however, addressing acidosis should be a primary focus.
- Always calculate the safe transfusion threshold for red blood cells and counteract the potassium concentration of stored PRBCs by simultaneously infusing saline.

Acknowledgment

We extend our gratitude to Ian Yuan, Assistant Professor in the Department of Anesthesiology and Critical Care Medicine at The Children's Hospital of Philadelphia, Perelman School of Medicine, University of Pennsylvania, for his invaluable contributions in editing this manuscript.

Conflicts of Interest

No conflicts of interest have been declared.

Funding

This research was conducted independently without

any external funding.

References

[1] Vraets A, Lin Y, Callum JL. Transfusion-associated hyperkalemia. Transfus Med Rev. 2011 Jul;25(3):184-96. [**PMID**: 21498041]

[2] Smith HM, Farrow SJ, Ackerman JD, Stubbs JR, Sprung J. Cardiac arrests associated with hyperkalemia during red blood cell transfusion: a case series. Anesth Analg. 2008 Apr;106(4):1062-69. [**PMID**: 18349174]

[3] Mitani A, Shattock MJ. Role of Na-activated K channel, Na-K-Cl cotransport, and Na-K pump in [K]e changes during ischemia in rat heart. Am J Physiol. 1992 Aug;263(2 Pt 2):H333-40. [**PMID**: 1324611]

[4] Terkildsen JR, Crampin EJ, Smith NP. The balance between inactivation and activation of the Na+-K+ pump underlies the triphasic accumulation of extracellular K+ during myocardial ischemia. Am J Physiol Heart Circ Physiol. 2007 Nov;293(5):H3036-45. [**PMID**: 17873015]

[5] Hall TL, Barnes A, Miller JR, Bethencourt DM, Nestor L. Neonatal mortality following transfusion of red cells with high plasma potassium levels. Transfusion. 1993 Jul;33(7):606-609. [**PMID**: 8333025]