The Turkish Journal of Pediatrics 2024; 66: 364-368
https://doi.org/10.24953/turkjpediatr.2024.4541

Case Report

Extremely rare cause of hyperkalemia: ileostomy–induced hyperkalemia in extremely low birth weight infants

Mehmet Mutlu[1], Yakup Aslan[1], Şebnem Kader[1], Emine Ayça Cimbek[2], Gül Şalcı[3]

[1]Division of Neonatology, Karadeniz Technical University, Faculty of Medicine, Trabzon; [2]Division of Pediatric Endocrinology, Karadeniz Technical University, Faculty of Medicine, Trabzon; [3]Department of Pediatric Surgery, Karadeniz Technical University, Faculty of Medicine, Trabzon, Türkiye.

ABSTRACT

Background. Hyperkalemia is one of the most serious electrolyte disturbances, and it can cause lethal cardiac arrhythmia. Although hyperkalemia associated with ileostomies has been reported in adults, to the best of our knowledge, it has not previously been reported in neonates.

Case. We report ileostomy–induced hyperkalemia that persisted during the ileostomy and resolved promptly after the closure of the ileostomy in two extremely low birth weight (ELBW) infants, with birth weights of 850 g and 840 g and gestational ages of 27 weeks and 27 weeks 6 days.

Conclusions. These cases highlight that disruption of intestinal integrity in ELBW infants may cause hyperkalemia. Ensuring the integrity of the gastrointestinal tract plays an important role in the treatment of electrolyte disorders such as hyperkalemia in ELBW infants with an ileostomy.

Key words: extremely low birth weight, premature, ileostomy, hyperkalemia.

Hyperkalemia is defined as a serum or plasma potassium concentration exceeding 6 mEq/L in a non-hemolyzed specimen in neonates.[1] Renal dysfunction or immaturity, medications, massive tissue breakdown (asphyxia, hypotension, intraventricular hemorrhage, necrotizing enterocolitis and intravascular hemolysis), congenital adrenal hyperplasia or acute adrenal insufficiency may cause hyperkalemia in extremely low birth weight (ELBW) neonates. It can be life-threatening because of its effect on cardiac rhythm. Although hyperkalemia associated with ileostomies has been reported in adults, it has not previously been reported in neonates.[2-4] Herein, we present two ELBW premature newborns with ileostomy–induced hyperkalemia that persisted during ileostomy and resolved promptly after closure of the ileostomy.

✉ Mehmet Mutlu
drmehmetmutlu38@hotmail.com

Received 30th Mar 2024, revised 8th May 2024, accepted 13th May 2024.

Case Presentations

Case 1

A female preterm infant with a birth weight of 850 g was delivered by caesarean section at 27 weeks of gestation. The mother did not receive antenatal steroids. The infant was admitted to the neonatal intensive care unit (NICU) due to being extremely preterm and respiratory distress syndrome. The patient had normal external genitalia without areolar or genital hyperpigmentation. Noninvasive mechanical ventilation support and less-invasive surfactant administration were provided. Total parental nutrition and minimal enteric nutrition with maternal milk were given just after the birth. On the 10th day of life, the patient had recurrent episodes of apnea, bradycardia, lethargy, abdominal distension and vomiting.

An abdominal X-ray showed evidence of intestinal pneumatosis and ileus. The patient was diagnosed with necrotizing enterocolitis (NEC) according to a combination of clinical and radiographic features. Enteral nutrition was discontinued, antibiotics (ampicillin, gentamicin and metronidazole) and parenteral nutrition were started. Nasogastric decompression was applied. On the 16th day of life, despite medical treatment, the patient did not improve, leading to the performance of an ileostomy 10 cm proximal to the cecum. Measured serum potassium levels were between 4.2 and 4.9 mEq/L before the operation. Serum potassium levels in non-hemolyzed serum were between 5.7 and 6.9 mEq/L during the ileostomy period and between 4.4 and 5.0 mEq/L after closure of the ileostomy (Fig. 1). Potassium chloride (KCl) (1 mEq/kg/d) was added to parenteral nutrition fluids before surgery. The patient was not receiving medications that contained potassium (total parenteral nutrition and antibiotics) or potassium sparing diuretics during the ileostomy period. The ileostomy closure was performed on the 113th day of life. No signs of hyperkalemia were detected on the electrocardiogram (ECG). Ultrasound imaging of the adrenal glands and urinary systems showed no pathology. Serum creatinine, blood urea nitrogen (BUN), sodium (Na), blood gases and urine output (ml/kg/h) were within normal limits during the ileostomy period (Table I). Renin, aldosterone and 17-hydroxyprogesterone levels were within the normal range (Table I). The low dose (1 mcg) adrenocorticotropic hormone (ACTH) stimulation test revealed a normal response (Table I).

Case 2

A preterm male infant with a birth weight of 840 g from a twin pregnancy was delivered by caesarean section at 27 weeks and 6 days of gestation. The pregnancy was complicated by preeclampsia. The mother received antenatal steroids. Neonatal resuscitation was performed in the delivery room. The Apgar scores were 3 and 6 at the 1st and 5th minute, respectively. The patient had normal external genitalia without areolar or genital hyperpigmentation. The infant was admitted to the NICU due to prematurity,

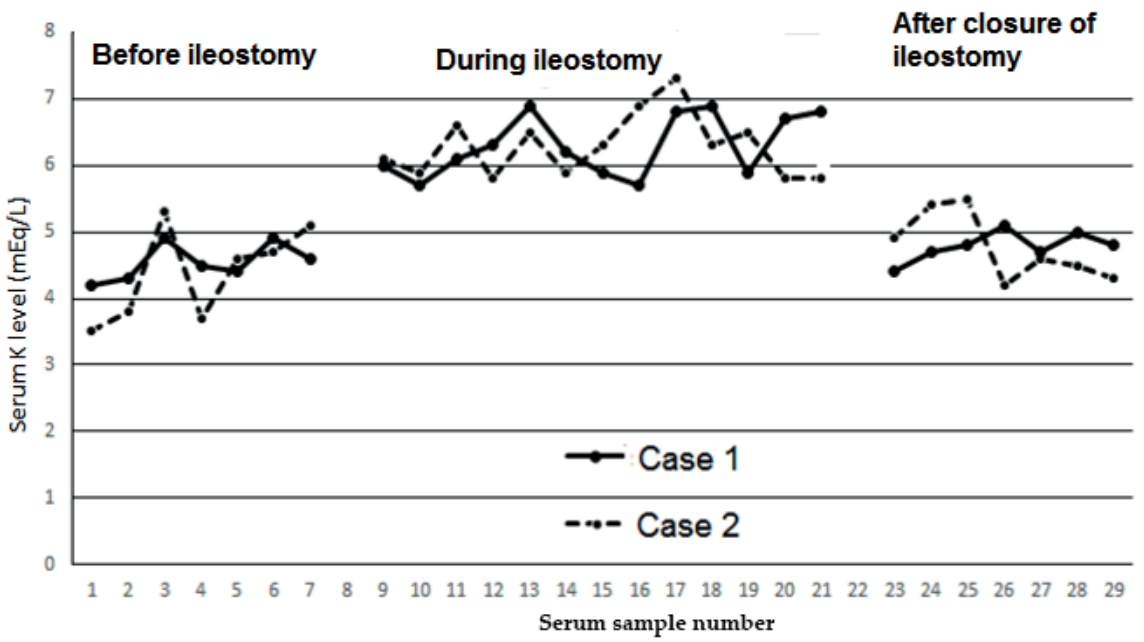

Fig. 1. Changes in serum potassium concentration before ileostomy, during ileostomy and after closure of ileostomy.

Table I. Laboratory values of the cases during ileostomy.

	Case 1	Case 2
Glucose (mg/dL)	70-95	82-122
BUN (mg/dL)	4-21	2-20
Cr (mg/dL)	0.13-0.4	0.14-0.49
Na (mEq/L)	131-140	131-137
K (mEq/L)	5.7-6.9	5.8-7.3
Urine Na (mEq/L)	5-7	6.2-25
Urine K (mEq/L)	11.2-48	1.6-71
Blood pH	7.34-7.418	7.34-7.42
HCO_3^- (mEq/L)	19.9-22.8	21.0-23.0
Renin (N: 2.4 – 37 ng/ml/h)	8.99	-
Aldosterone (N: 19-141 ng/dL)	75.69	-
ACTH (N: <46 ng/L)	13.7	23.7
Cortisol (μg/dL)	5.97	11.62
30-min cortisol	48.4	-
17OHP (N: 6.3-10 ng/mL)	1.82	3.56

Intervals indicate min-max values.
ACTH: Adrenocorticotropic hormone,
17OHP: 17-hydroxyprogesterone.

being an ELBW infant, and respiratory distress syndrome. Surfactant and mechanical ventilation support were administered. Total parental nutrition was immediately started, and minimal enteric nutrition with maternal milk was started at 24 hours of life. The patient had bradycardia, lethargy, feeding intolerance, recurrent vomiting, and abdominal distension and vomiting on the 12th day of life. Abdominal X-ray showed evidence of dilated loops of the bowel, pneumatosis intestinalis, and portal venous air. Thrombocytopenia and metabolic acidosis were determined on laboratory examination. The patient was diagnosed with NEC according to a combination of clinical, laboratory and radiographic features. Enteral nutrition was discontinued, antibiotics (meropenem and vancomycin) and parenteral nutrition were started. Nasogastric decompression was applied. KCl (2 mEq/kg/d) was added into the parenteral nutrition fluid before surgery. The patient underwent an ileostomy on the 18th day of life. Measured serum potassium levels were between 3.5 and 5.1 mEq/L before the operation. Serum potassium levels in non-hemolyzed serum were between 5.8 and 7.3 mEq/L during the ileostomy period and between 4.2 and 5.5 mEq/L after closure of the ileostomy (Fig. 1). When the serum potassium level was measured at 7.3 mEq/L, sinus rhythm with peaked T-waves was observed on the ECG and was treated with insulin and dextrose, bicarbonate and calcium gluconate. The patient was not receiving medications containing potassium (total parenteral nutrition and antibiotics) or potassium-sparing diuretics during the ileostomy period. Serum creatinine and BUN, Na, pH, HCO_3, urine output (ml/kg/h) were within normal limits during the ileostomy period (Table I). ACTH, cortisol and 17-hydroxyprogesterone levels were within normal limits (Table I). Ultrasound imaging of the adrenal glands and urinary systems showed no pathology. The ileostomy closure was performed on the 91st day of life. A blood transfusion was not given to the patients after the ileostomy was opened.

A written consent form was obtained from the families for this publication.

Discussion

In the present report, ileostomy–induced hyperkalemia was presented in two extremely preterm infants. To the best of our knowledge, this has not been previously reported in neonates. In both cases, although the serum potassium levels were within normal limits before the ileostomy, it increased and persisted in the ileostomy period, and recovered after the closured of the ileostomy.

Potassium is critical for maintaining cellular function. 98% of total body potassium is intracellular. Plasma potassium concentration is kept within narrow limits (3.5-5.0 mEq/L). The plasma potassium level in neonates is elevated compared to that of older infants. In the early postnatal period, premature neonates with a birthweight of <1000 grams and a postmenstrual age of <30 weeks have higher serum potassium concentrations due to renal function immaturity

and an inadequate response to hormones that control potassium levels. The highest level (>6 mEq/L) is observed at around 24 hours of life. After the third day of life, it starts to gradually decrease and stabilize over the next 4-5 days.[5]

Hyperkalemia is one of the most serious electrolyte disturbances because it can cause lethal cardiac arrhythmia. Hyperkalemia may be caused by excess potassium intake, impaired potassium excretion, drugs that can cause hyperkalemia, maldistribution between intra- and extracellular space or pseudohyperkalemia.[6] Acute kidney injury, adrenal insufficiency, congenital adrenal hyperplasia, metabolic acidosis or increased plasma osmolality, such as hyperglycemia, massive tissue breakdown such as asphyxia, hypotension, intraventricular hemorrhage, rhabdomyolysis, and blood cell transfusions can result in hyperkalemia. None of these conditions were present in these patients. Potassium-sparing diuretics (spironolactone), trimethoprim, non-steroidal anti-inflammatory drugs (ibuprofen, indometacin) angiotensin converting enzyme inhibitors, digoxin, heparin, beta blockers, mannitol and calcium channel blockers may cause hyperkalemia.[7] The patients did not receive any medication that could cause hyperkalemia during the hyperkalemic period. No potassium was added to the total parenteral nutrition fluid, and a potassium rich formula was not given in the ileostomy period. There was no condition such as mechanical hemolysis, lymphocytosis or thrombocytosis.

Ou et al.[8] reported that seven newborns with pseudo-hyperkalemia and high levels of aldosterone and renin were diagnosed with secondary pseudohypoaldosteronism, due to excessive gastrointestinal losses from ileostomy or jejunostomy. In our cases, hyponatremia and dehydration were not observed, aldosterone and renin levels were measured in only one patient, and the level was within normal limits.

Potassium homeostasis is maintained by oral intake, absorption from the gastrointestinal tract, and excretion through the colon and kidney. The kidneys are responsible for approximately 90% of excess potassium excretion in the body, the remaining 10% of potassium is excreted in the colon.[6,7] The colon has the capacity to absorb or excrete potassium depending on the serum potassium status. When the renal excretion of potassium is limited, as in ELBW infants or in individuals with chronic kidney disease, colonic excretion acquires a more prominent role in regulating extracellular potassium.[5] We assume that the hyperkalemia observed in our patients was caused by reduced colonic potassium excretion due to the disabling of the colon as a result of the ileostomy.

Hyperkalemia has been reported in adult patients with an ileostomy due to impaired intestinal continuity.[6,7] After the bowel continuity was restored, hyperkalemia improved as in our patients. High fecal potassium levels after ostomy closure could explain this situation. We could not measure the fecal potassium concentration before and after the stoma. However, the high serum potassium during the ileostomy period, which decreased to the normal range after closure of the ileostomy, supports this situation.

Hyperkalemia (≥7 mEq/L) is a severe acute problem in ELBW infants[1] and should be treated when hyperkalemia when it exceeds 7 mEq/L or hyperkalemic changes occur on the ECG with insulin and dextrose, bicarbonate and calcium gluconate. It should be kept in mind that an ileostomy may lead to hyperkalemia when other conditions that may cause hyperkalemia in ELBW neonates are excluded.

Ethical approval

A written consent form was obtained from the families for this publication.

Author contribution

Study conception and design: MM, YA; data collection: MM, ŞK, EAC, GŞ; analysis and interpretation of results: MM, YA, ŞK; draft manuscript preparation: MM, ŞK, EAC, GŞ. All

authors reviewed the results and approved the final version of the manuscript.

Source of funding

The authors declare the study received no funding.

Conflict of interest

The authors declare that there is no conflict of interest.

REFERENCES

1. Wright CJ, Posencheg MA, Seri I, Evans JR. Fluid, Electrolyte, and Acid-Base Balance. In: Gleason CA, Juul SE, editors. Avery's Diseases of the Newborn. 10th ed. Philadelphia: Elsevier; 2018: 368-389. https://doi.org/10.1016/B978-0-323-40139-5.00030-9
2. Weise WJ, Serrano FA, Fought J, Gennari FJ. Acute electrolyte and acid-base disorders in patients with ileostomies: a case series. Am J Kidney Dis 2008; 52: 494-500. https://doi.org/10.1053/j.ajkd.2008.04.015
3. Kononowa N, Dickenmann MJ, Kim MJ. Severe hyperkalemia following colon diversion surgery in a patient undergoing chronic hemodialysis: a case report. J Med Case Rep 2013; 7: 207. https://doi.org/10.1186/1752-1947-7-207
4. Yorimitsu D, Sasaki T, Horike H, et al. Severe hyperkalemia following ileostomy not colostomy in a patient undergoing chronic hemodialysis. Kawasaki Medical Journal 2015; 41: 65-69. https://doi.org/10.11482/KMJ-E41(2)65
5. Bonilla-Félix M. Potassium regulation in the neonate. Pediatr Nephrol 2017; 32: 2037-2049. https://doi.org/10.1007/s00467-017-3635-2
6. Viera AJ, Wouk N. Potassium disorders: hypokalemia and hyperkalemia. Am Fam Physician 2015; 92: 487-495.
7. Lehnhardt A, Kemper MJ. Pathogenesis, diagnosis and management of hyperkalemia. Pediatr Nephrol 2011; 26: 377-384. https://doi.org/10.1007/s00467-010-1699-3
8. Ou CY, Chen YJ, Lin GB, Chen MF, Chia ST. Case report: newborns with pseudohypoaldosteronism secondary to excessive gastrointestinal losses through high output stoma. Front Pediatr 2021; 9: 773246. https://doi.org/10.3389/fped.2021.773246