CASE REPORT

Amikacin-Induced Bartter-Like Syndrome, Profound Hyponatremia, and Acute Kidney Injury: A Case Report

Kwannate Intarawongchot, MD[1], Kittrawee Kritmetapak, MD[1]

[1] Division of Nephrology, Department of Medicine, Faculty of Medicine, Khon Kaen University, Khon Kaen, Thailand

Amikacin is a commonly used aminoglycoside antibiotic for the treatment of severe gram-negative bacterial infections, infective endocarditis, and is also employed as a second-line medication for tuberculosis. Potential side effects of amikacin include nephrotoxicity and ototoxicity, which may occur in high-risk patients, including those on prolonged high-dose therapy, the elderly, and individuals with pre-existing kidney disease or hypovolemia. In addition to acute tubular necrosis, amikacin may cause Bartter-like syndrome, manifested with polyuria, hyponatremia, hypokalemia, hypochloremic metabolic alkalosis, hypocalcemia, hypercalciuria, and hypomagnesemia. We report a case of an elderly woman who developed non-oliguric acute kidney injury and Bartter-like syndrome, with life-threatening hyponatremia (Na^+ 104 mEq/L) and hypokalemia (K^+ 1.5 mEq/L), after a 4-week amikacin therapy with a cumulative dose of 28 g. The patient required cautious intravenous fluid and electrolyte replacement, and kidney function recovered approximately 6 weeks after discontinuing amikacin and providing supportive care. In summary, prudent prescribing, close monitoring of kidney function and electrolytes, and prompt cessation at signs of aminoglycoside nephrotoxicity may aid kidney recovery.

Keywords: Acute kidney injury; Aminoglycoside; Bartter syndrome; Hyponatremia; Nephrotoxicity

Received 18 January 2024 | Revised 14 March 2024 | Accepted 2 May 2024

J Med Assoc Thai 2024;107(Suppl. 1):S136-41

Website: http://www.jmatonline.com

Bartter syndrome is a rare inherited renal tubular disorder primarily affecting the thick ascending limb of Henle's loop (TALH), resulting in excessive urinary salt loss and secondary hyperreninemic hyperaldosteronism[1]. Furthermore, some medications, including aminoglycosides, can cause acute kidney injury (AKI) and renal tubulopathy, manifesting as Bartter-like syndrome, which can be life-threatening if not promptly recognized[2]. Nevertheless, the occurrence of severe hyponatremia, especially in conjunction with other profound electrolyte disturbances, has been rarely reported in patients receiving amikacin. The present study was approved by the Human Research Ethics Committee of Khon Kaen University (HE661531).

Correspondence to:

Kritmetapak K.

Division of Nephrology, Department of Medicine, Faculty of Medicine, Khon Kaen University, Khon Kaen 40002, Thailand.

Phone: +66-43-348888

Email: kittrawee@kku.ac.th

How to cite this article:

Intarawongchot K, Kritmetapak K. Amikacin-Induced Bartter-Like Syndrome, Profound Hyponatremia, and Acute Kidney Injury: A Case Report. J Med Assoc Thai 2024;107(Suppl.1):S136-41

DOI: 10.35755/jmedassocthai.2024.S01.S136-S141

Case Report

A 64-year-old woman presented with progressive jaundice for one week. She had a 4-month history of chronic back pain, significant weight loss, and a diagnosis of tuberculous spondylitis based on magnetic resonance imaging of the lumbosacral spine. One month earlier, she had initiated anti-tuberculosis treatment with isoniazid 300 mg/day, rifampicin 600 mg/day, pyrazinamide 1,500 mg/day, and ethambutol 800 mg/day. Upon admission, she was diagnosed with drug-induced cholestatic hepatitis due to anti-tuberculosis medications. The infectious disease physicians then adjusted her treatment to include intravenous amikacin 1 g/day, levofloxacin 750 mg/day, and ethambutol 800 mg/day. Serial measurements of liver function tests showed improvement after modifying the anti-tuberculosis regimen. However, after 4 weeks of amikacin therapy (on the 28th day of admission), the patient developed nausea, vomiting, generalized muscle weakness, and progressive drowsiness. Her vital signs included a body temperature of 36.8°C, blood pressure of 109/67 mmHg, a pulse rate of 80 beats/min, and a respiratory rate of 18 breaths/min. Physical examination revealed a drowsy woman with dry oral mucosa, flat neck veins, a body weight of 64 kg, a height of 160 cm, and a body mass index of 25 kg/m². Neurological

examination noted proximal muscle weakness but no focal neurological deficits, and the patient produced approximately 2 to 3 liters of urine per day.

Investigations

Blood chemistry tests revealed AKI and mild transaminitis. Blood electrolyte analysis showed profound hyponatremia, hypokalemia, hypochloremic metabolic alkalosis, hypocalcemia, and hypomagnesemia (Table 1). During the current hospital admission, the patient did not receive diuretic or laxative medication. Urinalysis revealed a specific gravity of 1.008, a pH of 7.0, trace protein, 3 to 5 red blood cells, and 1 to 2 white blood cells per high-power field. The spot urine showed an osmolality of 216 mOsm/kg, sodium of 59 mEq/L, potassium of 28 mEq/L, and chloride of 53 mEq/L. A urine potassium-to-creatinine ratio was 140 mEq/g and a transtubular potassium gradient was 18.6, suggesting urinary potassium loss. Additional tests showed hypercalciuria (urine calcium-to-creatinine ratio 0.214 mg/mg) and high fractional excretion of urea (59.7%), sodium (6.3%), chloride (10.3%), calcium (7.5%), and magnesium (33.3%). The arterial blood gas analysis revealed pH 7.57, PCO_2 32 mmHg, PO_2 89 mmHg, ionized calcium 0.46 mmol/L, and oxygen saturation 99%. The electrocardiogram revealed prolonged QT intervals (565 ms), flattened T waves, and U waves. The results of serum parathyroid hormone (PTH) and 25-hydroxyvitamin D levels are lacking.

Differential diagnosis

The presence of low-normal blood pressure, salt-wasting hypovolemic hyponatremia, hypokalemic metabolic alkalosis, hypocalcemia, and hypomagnesemia is consistent with Bartter-like syndrome. Considering that the serum electrolyte values were within the normal range before amikacin therapy, it is likely that the acquired renal tubular dysfunction in this patient was caused by amikacin. The differential diagnoses were Gitelman-like syndrome and Fanconi syndrome. The presence of hypercalciuria (>0.2 mg/mg) and pronounced urinary salt loss pointed towards Bartter-like syndrome,

Table 1. Laboratory values

Variable	Reference Range, Adults	1 Month before Current Admission	Current Admission, 1st Day	Current Admission, 28th Day	Hospital Discharge, 70th Day	2 Weeks after Hospital Discharge
Plasma glucose (mg/dL)	70 to 100		110	104		
Blood urea nitrogen (mg/dL)	6 to 20	21.3	23	26.4	10.5	12.5
Creatinine (mg/dL)	0.51 to 0.95	1.17	0.83	2.23	1.31	1.12
Uric acid (mg/dL)	2.5 to 6.9	5.2		4.9		
Osmolality in serum (mOsm/kg)	275 to 295			215		
Sodium (mEq/L)	136 to 145	138	135	104	137	138
Potassium (mEq/L)	3.5 to 5.0	3.8	4.8	1.5	3.3	3.5
Chloride (mEq/L)	98 to 106	103	97	57	100	98
Bicarbonate (mEq/L)	23 to 26	23	22.7	29.2	26	27
Calcium (mg/dL)	8.8 to 10.2			6.4	9.6	9.1
Phosphorus (mg/dL)	2.7 to 4.5			3.8	3.9	3.7
Magnesium (mg/dL)	1.6 to 2.6			1.0	2.0	1.9
Albumin (g/dL)	3.5 to 5.2	3.9	3.4	3.0	3.5	3.9
Total bilirubin (mg/dL)	0.3 to 1.2	0.4	22.5	3.2	0.6	0.7
Direct bilirubin (mg/dL)	0.1 to 0.5	0.2	19.1	2.4	0.4	0.4
Alanine transaminase (U/L)	0 to 25	16	107	28	16	15
Aspartate transaminase (U/L)	0 to 32	18	99	55	19	21
Alkaline phosphatase (U/L)	35 to 104	125	122	54	68	60

making the diagnosis of Gitelman-like syndrome unlikely, whereas hypocalciuria (<0.07 mg/mg) would suggest Gitelman syndrome[3]. Fanconi syndrome was excluded due to the absence of euglycemic glycosuria, normal anion gap metabolic acidosis, hypophosphatemia, and hypouricemia. The altered mental status in this patient was largely attributed to profound hyponatremia, which was caused by massive natriuresis and vasopressin-mediated water retention. Moreover, the high fractional excretion of sodium (>2%) and urea (>50%) in combination with isosthenuria support the diagnosis of AKI secondary to acute tubular necrosis (ATN). We could not exclude the possibility that AKI in this patient might have been contributed to by levofloxacin or ethambutol-induced tubulointerstitial nephritis[4,5] and crystal nephropathy secondary to levofloxacin[6]. Renal tuberculosis may lead to granulomatous interstitial nephritis, but the improvement in tuberculosis symptoms in this patient makes this scenario unlikely.

Treatment

Amikacin, levofloxacin, and ethambutol were discontinued, and the patient received intravenous infusions of 0.9% sodium chloride, potassium chloride, calcium gluconate, and magnesium sulfate. We withheld 3% NaCl owing to the patient's hypotension, polyuria, and marked hypovolemia. Due to the patient's high risk of osmotic demyelination syndrome, marked by severely low serum sodium (<105 mEq/L) and concurrent hypokalemia, our aim was to gradually raise serum sodium by less than 8 mEq/L over 24 hours. The authors adjusted intravenous fluids between 0.9% sodium chloride, 0.45% sodium chloride, and 5% dextrose in water based on serum sodium level changes. As a response to the life-threatening hypokalemia, we administered intravenous potassium chloride at a rate of 20 mEq per hour via a central line and adjusted as needed. Neurological symptoms including consciousness and muscle power, along with serum creatinine and electrolyte levels, gradually improved after intermittent electrolyte replacement during the hospitalization. The patient was discharged on isoniazid, ethambutol, and levofloxacin for tuberculous spondylitis, along with electrolyte supplements including elixir KCl, NaCl tablets, and calcium carbonate.

Follow-up and outcomes

Six weeks after stopping amikacin and providing supportive care, serum creatinine improved markedly from 2.23 to 1.31 mg/dL, and serum electrolytes normalized with the use of supplements. At 8 weeks post-amikacin discontinuation, serum creatinine returned to the baseline level of 1.12 mg/dL, allowing for the discontinuation of electrolyte supplements without any subsequent drop in serum electrolytes. The delayed kidney recovery may result from limited tubular regeneration capacity in elderly patients, particularly after prolonged exposure to high-dose nephrotoxic drugs.

Discussion

Amikacin is a small-molecule aminoglycoside, highly hydrophilic, and minimally protein-bound. It is renally excreted via glomerular filtration, and nephrotoxicity is due to drug accumulation in proximal tubular cells (Figure 1). Aminoglycosides are attracted to the negatively charged phospholipids in the apical membrane of the proximal tubule, thereby promoting their cellular uptake via the endocytic receptors megalin and cubilin[7]. The positive charge in aminoglycoside structure augments nephrotoxicity. The decreasing number of positive charges in the structure of aminoglycosides follows this order: neomycin > gentamicin > tobramycin > amikacin > netilmicin > streptomycin. Aminoglycoside nephrotoxicity can manifest as non-oliguric AKI resulting from ATN, with an incidence ranging

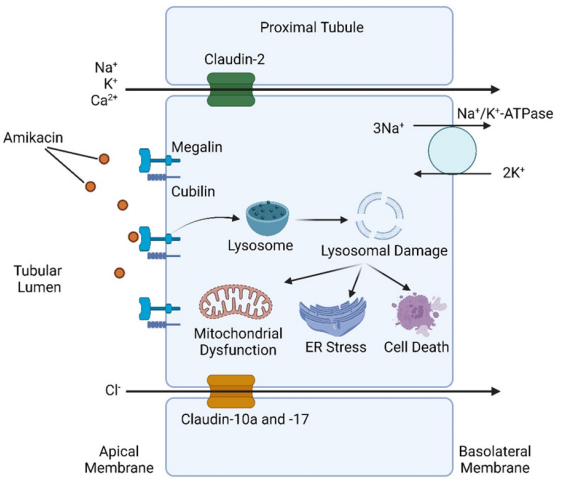

Figure 1. Mechanisms of amikacin-induced acute tubular necrosis. After being freely filtered through the glomerulus, polycationic amikacin is attracted to anionic phospholipid membranes, where it binds to the megalin/cubilin receptor complex on the apical membrane of proximal tubule. Amikacin is then endocytosed, entering the cell, and subsequently translocated into lysosomes. Lysosomal damage, rupture, along with mitochondrial dysfunction and endoplasmic reticulum (ER) stress, lead to necrosis of proximal tubular cells.

(Adapted from Perazella MA, Rosner MH. Drug-Induced Acute Kidney Injury. Clin J Am Soc Nephrol. 2022;17:1220-1233)

from 10% to 20%, and it typically develops 5 to 7 days after the initial exposure[8]. It can also manifest as Bartter-like syndrome, Fanconi syndrome, and distal tubular dysfunction. The risk factors for aminoglycoside nephrotoxicity present in this patient were extended high-dose therapy, female sex, advanced age, hypovolemia, and hypoalbuminemia. Other established risk factors for nephrotoxicity included pre-existing chronic kidney disease (CKD), diabetes mellitus, liver disease, sepsis, concurrent use of nephrotoxic agents, and a multiple daily dosing regimen of aminoglycosides[2,9]. Pharmacological guidelines recommend a maximum cumulative amikacin dose of 15 g per treatment course to prevent nephrotoxicity and ototoxicity.

Bartter-like syndrome, characterized by hyponatremia, hypokalemia, hypochloremic metabolic alkalosis, hypocalcemia, hypercalciuria, and hypomagnesemia, has been described in patients treated with aminoglycosides, especially gentamicin[10-14], with an approximate incidence of 2%[7]. Bartter-like syndrome can also result from other medications (such as loop diuretics, amphotericin B, colistin, cisplatin)[13,15,16], autoimmune diseases (such as Sjogren's syndrome)[17], and sarcoidosis[18]. Bartter-like syndrome typically manifests after a minimum of 1 to 2 weeks of aminoglycoside treatment. However, we could not determine the precise onset of amikacin nephrotoxicity in our patient due to vague clinical symptoms, resulting in delayed blood chemistry assessment, which occurred 4 weeks after the initiation of amikacin.

In the TALH of the nephron, the transcellular reabsorption of sodium chloride mainly occurs through Na^+-K^+-2Cl- cotransporter (NKCC2) in the apical membrane, which is driven by the activity of Na^+/K^+-ATPase in the basolateral membrane. The basolateral efflux of sodium and chloride occurs through Na^+/K^+-ATPase and the ClC-Kb chloride channel, respectively. Apical recycling of potassium to the tubular lumen through renal outer medullary K^+ channel (ROMK) generates a lumen-positive transepithelial potential, leading to paracellular reabsorption of sodium, calcium, and magnesium. The tight junction protein claudin-14 engages in a physical interaction with claudin-16/19 complex and directly inhibits its paracellular permeability to calcium and magnesium.

The primary function of the extracellular calcium-sensing receptor (CaSR) in the kidney involves regulating electrolyte and acid-base homeostasis. Although the CaSR is expressed throughout the entire length of the nephron, it exhibits the highest expression within the basolateral membrane of the TALH[19]. Here, it detects peritubular calcium fluctuations and influences the renal tubular transport of various electrolytes. Polycationic aminoglycosides, including amikacin, are recognized as ligands for CaSR. When amikacin activates basolateral CaSR in the TALH, it generates inhibitory signals to suppress the activity of NKCC2, ROMK, and Na^+/K^+-ATPase, which leads to a subsequent reduction in apical potassium recycling and the positive voltage in the tubular lumen. This causes diminished tubular reabsorption and enhanced urinary losses of sodium, potassium, chloride, calcium, and magnesium (Figure 2). Additionally, activation of the CaSR in the TALH by amikacin results in an increase in claudin-14 expression, which subsequently impedes the paracellular reabsorption of divalent cations through claudin-16/19 (Figure 3). Moreover, aminoglycosides may act through the CaSR in the distal tubule and inhibit PTH-stimulated magnesium reabsorption, potentially causing renal magnesium loss[20].

Traditionally, there were 5 primary types of Bartter syndrome, with Bartter syndrome type 5 previously categorized as autosomal dominant hypocalcemia due to the activating mutation in the CaSR. Nevertheless, current classification defines Bartter syndrome type 5 as caused by the X-linked recessive mutation in the melanoma-associated antigen D2 (MAGED2) gene[1,21]. Hence, aminoglycoside-induced Bartter-like syndrome should be classified as the acquired form of CaSR activating syndrome, resembling historical Bartter syndrome type 5.

Hypomagnesemia in this patient exacerbates hypokalemia and hypocalcemia via several mechanisms. A decrease in intracellular magnesium, induced by hypomagnesemia, increases the activity of apical ROMK channels in the distal nephron and enhances renal potassium excretion[22]. While data

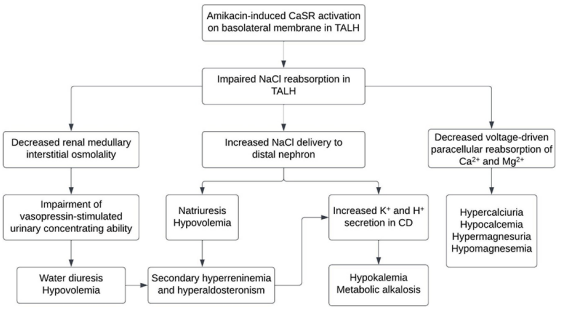

Figure 2. Pathogenesis of amikacin-induced Bartter-like syndrome.

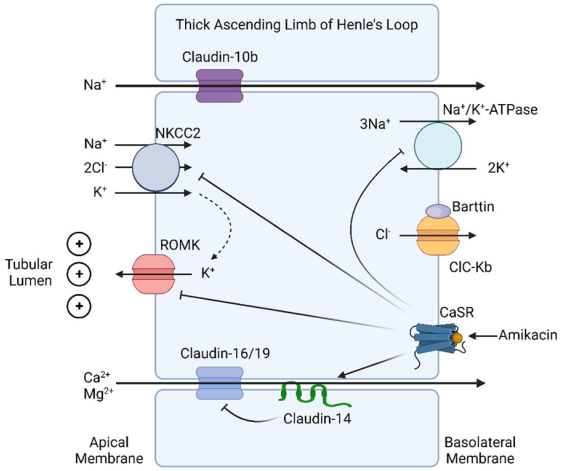

Figure 3. Mechanisms of amikacin-induced Bartter-like syndrome. Amikacin can activate the calcium-sensing receptor (CaSR) on the basolateral membrane of the thick ascending limb of Henle's loop. This activation can interfere with electrolyte transport by inhibiting four distinct pathways, involving Na^+-K^+-2Cl- cotransporter (NKCC2), renal outer medullary K^+ channel (ROMK), Na^+/K^+-ATPase, and paracellular diffusion mediated by claudin-16/19 complex. The inhibition of these transport mechanisms results in excessive urinary excretion of Na^+, K^+, Cl^-, Ca^{2+}, and Mg^{2+}, consequently leading to the development of hyponatremia, hypokalemia, hypochloremic metabolic alkalosis, hypocalcemia, and hypomagnesemia.

(Adapted from Zietse R, Zoutendijk R, Hoorn EJ. Fluid, electrolyte and acid-base disorders associated with antibiotic therapy. Nat Rev Nephrol. 2009;5:193-202)

on serum PTH and vitamin D levels are lacking, a previous report suggested that the mechanisms leading to hypocalcemia in hypomagnesemic patients are PTH resistance, PTH hyposecretion, and calcitriol underproduction[23]. Despite having AKI, the patient developed metabolic alkalosis instead of metabolic acidosis, as we would expect. The generation phase of metabolic alkalosis in this patient is due to hypovolemia-induced secondary hyperaldosteronism, resulting in increased H+ excretion in collecting duct. The important factors for maintaining metabolic alkalosis are volume contraction (causing low HCO_3^- filtration), hypochloremia (causing low HCO_3^- secretion in the collecting duct), and hypokalemia. Hypokalemia worsens metabolic alkalosis, particularly in volume-depleted states, by (1) promoting ammoniagenesis in proximal tubular cells, leading to new HCO_3^- generation; (2) enhancing proximal tubular reabsorption of filtered HCO_3^-; (3) increasing HCO_3^- generation and absorption in the collecting duct; and (4) reducing HCO_3^- secretion in the collecting duct[24]. Hypokalemia, hypocalcemia, and hypomagnesemia can lead to prolonged QT intervals, potentially resulting in life-threatening arrhythmias if left unrecognized. Metabolic alkalosis lowers ionized calcium levels, potentially exacerbating symptoms associated with hypocalcemia, such as changes in mental status or seizures.

Preventive strategies for aminoglycoside nephrotoxicity involve a limited duration of once-daily dosing, dose adjustments based on kidney function, maintaining euvolemia, and avoiding concomitant nephrotoxins[8,25]. General management of aminoglycoside-induced Bartter-like syndrome and AKI is prompt discontinuation of culprit medication and cautious replacement of fluids and electrolytes, especially sodium, potassium, chloride, calcium, and magnesium. Some patients may require spironolactone as a potassium- and magnesium-sparing medication. Aminoglycoside-induced Bartter-like syndrome generally resolves after 2 to 6 weeks following drug cessation, as reflected by our patient. The serum creatinine level usually returns to the baseline value within 3 weeks after cessation of therapy. However, recovery of kidney function may be delayed under certain conditions such as hypovolemia, sepsis, pre-existing CKD, or prolonged exposure to high-dose nephrotoxic agents. Long-term follow-up of kidney function is necessary to monitor the potential development of CKD.

Conclusion

Aminoglycoside nephrotoxicity encompasses AKI and Bartter-like syndrome, which is characterized by hyponatremia, hypokalemia, hypochloremic metabolic alkalosis, hypocalcemia, hypomagnesemia, and hypercalciuria. Prudent short-term prescribing, vigilant monitoring of kidney function and electrolytes, and prompt discontinuation upon signs of aminoglycoside nephrotoxicity may contribute to kidney function recovery.

What is already known on this topic?

Previous reports have shown that amikacin is nephrotoxic and can cause AKI and electrolyte disturbances resembling Bartter-like syndrome.

What this study adds?

Amikacin can lead to life-threatening hyponatremia due to polyuric salt wasting, especially in high-risk patients, including those on prolonged high-dose therapy, advanced age, pre-existing CKD, hypovolemia, and hypoalbuminemia. Close monitoring of serum creatinine, electrolytes, and urine output is warranted during aminoglycoside therapy, with prompt discontinuation upon the observation of laboratory evidence of nephrotoxicity.

Acknowledgements

The authors are grateful to the Department of Medicine, Faculty of Medicine, Khon Kaen University, for their support in publication.

Conflicts of interest

The authors declare no conflict of interest.

References

1. Konrad M, Nijenhuis T, Ariceta G, Bertholet-Thomas A, Calo LA, Capasso G, et al. Diagnosis and management of Bartter syndrome: executive summary of the consensus and recommendations from the European Rare Kidney Disease Reference Network Working Group for Tubular Disorders. Kidney Int 2021;99:324-35.
2. Perazella MA. Pharmacology behind common drug nephrotoxicities. Clin J Am Soc Nephrol 2018;13:1897-908.
3. Blanchard A, Bockenhauer D, Bolignano D, Calò LA, Cosyns E, Devuyst O, et al. Gitelman syndrome: consensus and guidance from a Kidney Disease: Improving Global Outcomes (KDIGO) Controversies Conference. Kidney Int 2017;91:24-33.
4. Kwon SH, Kim JH, Yang JO, Lee EY, Hong SY. Ethambutol-induced acute renal failure. Nephrol Dial Transplant 2004;19:1335-6.
5. Ramalakshmi S, Bastacky S, Johnson JP. Levofloxacin-induced granulomatous interstitial nephritis. Am J Kidney Dis 2003;41:E7.
6. Ansari FA, Manuel S, Dwivedi R, Boraiah SK, Raju SB, Uppin M, et al. A rare case of acute kidney injury due to levofloxacin-induced crystal nephropathy. Indian J Nephrol 2019;29:424-6.
7. Zietse R, Zoutendijk R, Hoorn EJ. Fluid, electrolyte and acid-base disorders associated with antibiotic therapy. Nat Rev Nephrol 2009;5:193-202.
8. Perazella MA, Rosner MH. Drug-induced acute kidney injury. Clin J Am Soc Nephrol 2022;17:1220-33.
9. Le TA, Hiba T, Chaudhari D, Preston AN, Palowsky ZR, Ahmadzadeh S, et al. Aminoglycoside-related nephrotoxicity and ototoxicity in clinical practice: A review of pathophysiological mechanism and treatment options. Adv Ther 2023;40:1357-65.
10. Chrispal A, Boorugu H, Prabhakar AT, Moses V. Amikacin-induced type 5 Bartter-like syndrome with severe hypocalcemia. J Postgrad Med 2009;55:208-10.
11. Chen YS, Fang HC, Chou KJ, Lee PT, Hsu CY, Huang WC, et al. Gentamicin-induced Bartter-like syndrome. Am J Kidney Dis 2009;54:1158-61.
12. Hung CC, Guh JY, Kuo MC, Lai YH, Chen HC. Gentamicin-induced diffuse renal tubular dysfunction. Nephrol Dial Transplant 2006;21:547-8.
13. Chou CL, Chen YH, Chau T, Lin SH. Acquired bartter-like syndrome associated with gentamicin administration. Am J Med Sci 2005;329:144-9.
14. Landau D, Kher KK. Gentamicin-induced Bartter-like syndrome. Pediatr Nephrol 1997;11:737-40.
15. Yavas DP, Ekinci F, Horoz OO, Gundeslioglu OO, Atmis B, Yildizdas D. Acquired Bartter-like syndrome presenting with polyuria and reversible hypokalemia associated with colistin use in a critically ill pediatric patient. Indian J Crit Care Med 2021;25:822-4.
16. Tabish M, Mahendran M, Ray A, Vikram NK. Colistin-induced acquired Bartter-like syndrome: an unusual cause of meltdown. BMJ Case Rep 2020;13:e232630.
17. Fraj A, El Euch M, Jaziri F, Kefi A, Abdelghani KB, Turki S, et al. Acquired Bartter syndrome in primary Sjögren syndrome. Saudi J Kidney Dis Transpl 2020;31:1144-7.
18. Abu Ayyach A, Le Moine A, Kaci L, Royer-Chardon C, Ghisdal L, Marangoni M, et al. Salt-losing tubulopathy worsening the prognosis of renal sarcoidosis. J Nephrol 2023;36:627-30.
19. Riccardi D, Valenti G. Localization and function of the renal calcium-sensing receptor. Nat Rev Nephrol 2016;12:414-25.
20. Kang HS, Kerstan D, Dai L, Ritchie G, Quamme GA. Aminoglycosides inhibit hormone-stimulated Mg2+ uptake in mouse distal convoluted tubule cells. Can J Physiol Pharmacol 2000;78:595-602.
21. Laghmani K, Beck BB, Yang SS, Seaayfan E, Wenzel A, Reusch B, et al. Polyhydramnios, transient antenatal Bartter's syndrome, and MAGED2 mutations. N Engl J Med 2016;374:1853-63.
22. Huang CL, Kuo E. Mechanism of hypokalemia in magnesium deficiency. J Am Soc Nephrol 2007;18:2649-52.
23. Rosner MH, Ha N, Palmer BF, Perazella MA. Acquired disorders of hypomagnesemia. Mayo Clin Proc 2023;98:581-96.
24. Do C, Vasquez PC, Soleimani M. Metabolic alkalosis pathogenesis, diagnosis, and treatment: Core curriculum 2022. Am J Kidney Dis 2022;80:536-51.
25. Yousif Z, Awdishu L. Drug-induced acute kidney injury risk prediction models. Nephron 2023;147:44-7.