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Acquired Bartter's Syndrome: A Rare Metabolic Abnormality Induced by Streptomycin

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Abstract

Streptomycin associated nephrotoxicity can manifest either as nonoliguric acute kidney injury, tubular dysfunction or electrolyte abnormalities including Fanconi-like syndrome or Bartter syndrome. We report a case of Streptomycin-induced renal electrolyte wasting mimicking Bartter's syndrome.

Keywords: Acquired Bartter's Syndrome • Electrolyte wasting • Streptomycin

Introduction

Aminoglycosides, polyvalent cationic antibiotics, are widely prescribed in the treatment of gram negative infections. Aminoglycoside induced nephrotoxicities can have varied presentations such as interstitial nephritis and nonoliguric renal failure [1]. Other less common adverse effects include Fanconi-like syndrome or Bartter syndrome [2]. Aminoglycoside induced renal tubular dysfunction has been reported with gentamicin and tobramycin [3], however, there are only few case reports of bartter's syndrome associated with streptomycin to the best of our knowledge [4].

Case Presentation

A 38-years-old male, diagnosed as a case of pulmonary tuberculosis, on antituberculous treatment for 1 month presented to our hospital with complains of frequent episodes of vomiting. On admission, his blood pressure was 100/60 mmHg, pulse 105 beats/min, respiratory rate 22 breaths/min, and body temperature 98.2°F. Right upper quadrant tenderness without rebound tenderness was noted. The remainder of the physical examination was unremarkable. Serum biochemistries suggested deranged liver function tests [AST-219 U/L, ALT-42 U/L, ALP-13I U/L, T. Bil-5.1 mg/dl, D. Bil-3.8 mg/dl] however renal function tests and serum electrolytes, were within the normal range. Ultrasonographic scan suggested mildly coarse echo texture of liver with minimal free fluid in perisplenic and interbowel spaces. Alternative causes for elevated liver function tests including alcohol, acetaminophen, viral hepatitis, gallstones, and biliary obstruction were excluded, and a diagnosis of ATT-induced hepatitis was made and subsequently patient was shifted to modified ATT regimen containing Ethambutol, Levofloxacin and Streptomycin. On the fifth day of streptomycin administration, patient reported anorexia, lethargy and generalized weakness. Chvostek and Trousseau signs were although, absent. ECG showed low-voltage QRS complexes. Laboratory investigations revealed pH 7.59, PCO_2 33 mmHg, PO_2 160 mmHg, SO_2 98%, sodium 126 mmol/L, potassium 1.2 mmol/L, serum magnesium 1.05 mg/dl, bicarbonate 32.4 mmol/L, and ionized calcium 0.41 mmol/L suggesting profound hypokalemia, hyponatremia, hypomagnesemia, hypocalcemia, and metabolic alkalosis. Serum immunoreactive parathyroid hormone concentration was elevated secondary to hypocalcemia. These abnormalities persisted for many days as evidenced by Table 1. Streptomycin was subsequently discontinued, and intravenous supplementation of magnesium sulfate, calcium gluconate, and potassium chloride was started, and serum electrolytes returned to normal, as depicted in Table 1.

Table 1. Showing patient's serum and urine biochemistries and other relevant investigations during course of hospital admission.

Baseline investigations of patient at presentation			
Serum Biochemistries		**Urine Biochemistries**	
S. Na^+ (135-145 mmol/L)	126 mmol/L	Urine volume (mL/24 hours)	2950
S. K^+ (3.5-5.5 mmol/L)	1.2 mmol/L	K^+ (mEq/L) (25-120)	40.62 mEq/L
S.Cl (98-107 meq/L)	99.4 meq/L	Cl (mmol/L)	120.1 mmol/L
HCO_3 (22-26 mmol/L)	32.4 mmol/L	24-hr Calcium (0.10-0.25 gms/24 hours)	0.49 gms/24 hours
S. Mg^{2+} (1.6-2.5 mg/dL)	1.05 mg/dL	Osmolality (50-1200)	396 mOsm/KgH_2O
S. Ca^{2+} (8.4-11 mg/dL)	6.4 mg/dL	TTKG	17
S. Phosphate (3.4-4.5 mg/dL)	2.5 mg/dL		
Albumin (3.2-5.5 mg/dL)	2.5 mg/dL		
BUN (9-20 mg/dL)	<2 mg/dL		
Creatinine (0.5-1.0 mg/dl)	0.3 mg/dL		
i-PTH (15-68 pg/mL)	222.1 pg/mL		
S. Cortisol (133-537 mmol/L)	329.1 nmol/L		
Amylase	<30 U/L		
Lipase	129 U/L		

Relevant investigations of patient during hospital stay							
Biochemical parameters	**Day 1[a] (On Streptomycin)**	**Day 2**	**Day 3**	**Day 5**	**Day 6[b] (Streptomycin stopped)**	**Day 8**	**Day 11**
pH	7.59	7.55	7.47	7.50	7.26	7.42	7.46
S. Na^+ (mmol/L)	126	129	132	133	139	140	125
S. K^+ (mmol/L)	1.2	1.6	2.0	2.6	2.8	2.8	3.4
HCO^{3-} (mmol/L)	32.4	36.0	34.2	24.7	18.2	25.3	27.7
i.Ca^{2+} (mmol/L)	0.41	0.47	0.49	0.43	0.68	0.70	0.90
Mg^{2+} (mg/dL)	1.05	-	-	-	-	2.04	-

[a.] Day 1 of electrolyte imbalance (2nd day of streptomycin administration); b. Day 6 of electrolyte imbalance when streptomycin injection was stopped.

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Discussion

Aminoglycosides are associated with various toxicities including vestibular, cochlear and renal toxicity. Aminoglycoside-induced renal tubular dysfunction can either present as Fanconi-like syndrome with involvement of proximal tubules and manifesting as metabolic acidosis, hypophosphatemia, aminoaciduria and glucosuria as well as frequent elevations in serum creatinine, or as Bartter-like syndrome with distal renal tubule dysfunction, characterized by hypokalemic metabolic alkalosis, hypocalcemia, and hypomagnesemia without a significant elevation of serum creatinine [5]. In our patient, streptomycin-induced Bartter-like syndrome was diagnosed as other causative factors such as poor intake, vomiting, persistent diarrhea, massive glucosuria, and diuretic use were excluded, and the features of Bartter-like syndrome resolved upon withdrawal of streptomycin. Exact mechanisms culminating in aminoglycoside induced Bartter's syndrome remains elusive; however, it may be similar to hereditary variant of Bartter's disease involving a transporter defect in thick ascending limb of loop of Henle with subsequent renal wasting of Sodium, Potassium, Chloride, Ca, and Magnesium.

Conclusion

This case report highlights the occurrence of Bartter-like syndrome as one of the rare complications of streptomycin therapy emphasizing the fact that streptomycin-induced renal dysfunction may represent significantly underreported cause of Bartter-like syndrome. Thus, acquired Bartter-like syndrome should be kept in mind as a complication of aminoglycoside therapy, and close monitoring of serum and urine electrolytes is warranted during aminoglycoside therapy. Early diagnosis and prompt correction of these electrolyte and acid-base imbalances can reduce the risk of potentially life-threatening complications.

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