ORIGINAL RESEARCH PAPER

Medicine

BARTTER'S LIKE SYNDROME, VT STORM, ANOXIC BRAINSTEM DAMAGE AND DISSEMINATED TUBERCULOSIS: A THERAPEUTIC CHALLENGE

KEY WORDS: Bartter-like syndrome, tubulopathies. , dyselectrolytemia

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ABSTRACT

We describe a case of Disseminated tuberculosis with induced Bartter-like syndrome with ventricular tachycardia storm and anoxic brain stem damage. A 26-year-old female patient presented with fever and pain abdomen for the last six months. She was being treated outside. On further investigation, disseminated tuberculosis with hypokalemia, hyponatremia, hypomagnesemia, hypocalcemia, and hypochloremic metabolic alkalosis were noted. Hypokalemia and hypomagnesemia were the cause of recurrent ventricular tachycardia and anoxic brain stem damage. Calcium, potassium, and magnesium supplementation was given and drugs prolonging the QT interval were discontinued. Electrolytes normalized in a few days after correction. Bartters syndrome can result in renal tubular dysfunction leading to Fanconi syndrome and distal tubular acidosis. Mitochondrial dysfunction in the tubular cells is the possible cause of these tubulopathies. Acquired Bartter-like syndrome phenotypically resembles autosomal dominant type 5 Bartter syndrome. Treatment consists of correction of electrolyte imbalance, potassium-sparing diuretics, and Indomethacin or Rofecoxib. Prompt diagnosis and treatment of severe dyselectrolytemia are warranted in patients on multiple drugs so that fatal ventricular arrhythmia and hypoxic brain damage could be prevented as in our case.

Case Report :

A 26 yrs old lady, presented to us with a history of fever and pain abdomen on and off for the last 6 months which increased in severity for the last 2 weeks. She had a history of recent blood transfusion after rectal bleeding. Prior to her admission, she was thoroughly evaluated at an outside hospital. She was referred to our hospital with a diagnosis of Gram-negative septicemia with Shock (on Ionotropic support) and Rt. Upper zone pneumonia,? Miliary Koch's and Dimorphic anemia. She was febrile and her initial vital signs were normal with a pulse of 118/min and blood pressure of 90/76 mm of Hg. On examination, she was thin built and tachypnoeic. On auscultation of lung fields, breath sounds were diminished on the right and fine crepitations were present. Her X-ray chest and CT-thorax and whole abdomen findings suggested the presence of disseminated tuberculosis. The rest of the physical examination was unremarkable. Initial blood-work at the time of presentation revealed low serum calcium, magnesium, and potassium level. These electrolyte abnormalities, especially hypokalemia and hypomagnesemia disturbed cardiac conduction and resulted in ventricular tachycardia. The patient suddenly collapsed in the female medical ward. Cardiopulmonary resuscitation was immediately started. She was intubated and put on mechanical ventilation Electrocardiogram showed ventricular fibrillation. She was treated with intravenous lignocaine. Her arterial blood gas analysis was done immediately which showed severe dyselectrolytemia. After the correction of the electrolyte abnormalities, the patient became hemodynamically stable. This indicates the importance of early detection and correction of severe electrolyte abnormalities. As the electrolyte abnormality was persisting along with metabolic alkalosis so we thought we are dealing with Bartter like syndrome. Coexisting with hypokalemia, hypomagnesemia, and hypocalcemia, other electrolyte abnormalities noted(Table-1) were as follows: serum sodium of 128 mEq/L, potassium of 1.8 mEq/L, chloride of 95 mEq/L, the calcium of 6.98 mg/dL, and magnesium of 1.2 mg/dL. Serum albumin was 4 g/dL. Serum creatinine was normal and remained so throughout the course of hospitalization. The arterial blood gas evaluation showed metabolic alkalosis without respiratory compensation with a pH of 7.5, HCO3 of 29.9 mEq/L, and PaCO2 of 30 mmHg. Routine urine examination revealed 2–4 pus cells, 1-2 RBCs, and 1-2 epithelial cells, in the absence of proteinuria and glycosuria. The urine albumin /creatinine ratio was within the normal limits.. Urinary prostaglandin-E level was not performed as this assay was not readily available in our hospital. Vitamin D and serum parathyroid levels were found to be in the normal range, thus ruling out hypovitaminosis D and secondary hyperparathyroidism, respectively. The patient was treated symptomatically; injectable antibiotics, antitubercular therapy, calcium, magnesium, and potassium were administered to correct electrolyte abnormalities. The patient was discharged once her symptoms resolved and the serum electrolytes were normalized. On follow-up, electrolytes remained to be in the normal range.

Table 1 : Serum electrolytes in acquired Bartter-like syndrome.

Electrolytes (normal range, unit)	Day 0	Day 7	On discharge
Na^+ (130–145 mEq/l)	128	132	136
K^+ (3.5–4.5 mEq/l)	1.8	2.7	3.4
Ca^{++} (09–11 mg/dl)	6.98	6.92	7.8
Mg^{++} (1.6–2.3 mg/dl)	1.2	1.4	1.8
Cl^- (96–105 mEq/l)	98	100	102

Na^+: sodium; K^+: potassium; Ca^{++}: calcium (total); Mg^{++}: magnesium; Cl :chloride.

3. Discussion

Bartter syndrome is an inherited renal tubular disease affecting the ascending limb of the loop of Henle that manifests as hypokalemia, hypomagnesemia, hypocalcemia, and hypochloremic metabolic alkalosis, Electrolyte abnormalities similar to that of BS can be caused by chronic diuretic use, vomiting, and drugs like Aminoglycosides, Amphotericin B, Prostaglandins, Cisplatin, and heavy metals. [2, 3]. Acquired BS is clinically similar to the autosomal dominant type 5 Bartter syndrome, which is due to a mutation in the calcium-sensing receptor (CaSR) in the thick ascending limb (TAL) of Henle's loop [6]. In our case, we reported hypochloremic metabolic alkalosis, hypokalemia, hypomagnesemia, and hypocalcemia in a normotensive patient which was indicative of Bartter-like syndrome. BS patients have increased urinary prostaglandin-E. Treatment of BS predominantly consists of symptomatic correction of

electrolytes and discontinuation of the culprit drug. Indomethacin, potassium-sparing diuretics, and/or aldosterone receptor antagonists have been also recommended [3]. Our patient showed improvement after correcting hypokalemia, hypomagnesemia hypocalcemia, and stopping the offending drug which was causing QT-prolongation. [3]. Antitubercular treatment was continued. Electrolyte abnormalities like hypomagnesemia, hypokalemia, and hypocalcemia can be potentially life-threatening due to their cardiac effects producing a VT storm. There are diagnostic difficulties in all such cases but prompt diagnosis and treatment are essential for a good outcome of this essentially reversible condition.

Conflicts of Interest: The authors declare that they have no conflicts of interest.
Financial implication- Nil

References:

1. A. Chrispal, H. Boorugu, A. T. Prabhakar, and V. Moses, "Amikacin-induced type 5 Bartter-like syndrome with severe hypocalcemia," *Journal of Postgraduate Medicine*, vol. 55, no. 3, pp. 208–210, 2009.
2. G. Deschênes and M. Fila, "Primary molecular disorders and secondary biological adaptations in bartter syndrome," *International Journal of Nephrology*, vol. 2011, Article ID 396209, 8 pages, 2011.
3. H.W. Seyberth and K. P. Schlingmann, "Bartter- and Gitelman-like syndromes: salt-losing tubulopathies with loop or DCT defects," *Pediatric Nephrology*, vol. 26, no. 10, pp. 1789–1802, 2011.
4. A. M. Hall, P. Bass, and R. J. Unwin, "Drug-induced renal fanconi syndrome," *QJM*, vol. 107, no. 4, pp. 261–269, 2014.
5. Y.-S. Chen, H.-C. Fang, K.-J. Chou et al., "Gentamicin-induced Bartter-like syndrome," *American Journal of Kidney Diseases*, vol. 54, no. 6, pp. 1158–1161, 2009.
6. C.-C. Hung, J.-Y. Guh, M.-C. Kuo, Y.-H. Lai, and H.-C. Chen, "Gentamicin-induced diffuse renal tubular dysfunction [3]," *Nephrology Dialysis Transplantation*, vol. 21, no. 2, pp. 547-548, 2006.
7. R. W. Steiner and A. S. Omachi, "A Bartter's-like syndrome from capreomycin, and a similar gentamicin tubulopathy," *American Journal of Kidney Diseases*, vol. 7, no. 3, pp. 245–249, 1986.