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Refractory Hypokalemia in a Patient with HIV Infection

Case Report

Ivaturi Venkata Nagesh[1*], T Murari[2], VK Sashindran[3], VA Arun[4]

[1] Reader, Department of Internal Medicine, AFMC, Pune, India.
[2] Assoc Professor, Department of Internal Medicine, AFMC, Pune, India.
[3] Professor & HOD, Department of Internal Medicine, AFMC, Pune, India.
[4] Assistant Professor, Department of Internal Medicine, AFMC, Pune, India.

Background

Renal involvement in HIV can manifest in various forms like immune-mediated glomerulonephritis, HIV-associated nephropathy, renal tubular acidosis, acute/chronic renal failure and HIV induced distal RTA is usually associated with hypergammaglobulinemia and use of trimethoprim [1]. Distal renal tubular acidosis is characterized by hypokalemia, non-anion gap metabolic acidosis and inability of the urine to decrease the urine pH below 5.5. In review of literature only two cases of HIV induced distal RTA were described [2].

Case Summary

35 yrs old male a known case of case of HIV infection on Zidovudine, Lamivudine and Efavirenz for 07 years, and detected to have multi drug resistant tuberculosis on Inj Capreomycin, levofloxacin, Ethambutol, Pyrazinamide, Amikacin, and on trimethoprim and sulphamethoxazole prophylaxis for Pneumocystis jiroveci pneumonia presented with diarrhea, weakness and significant weight loss of 7 days duration. There was no history joint pain, rash, hair loss, breathlessness, or decreased urine output. Clinical examination revealed BMI 16.1kg/m^2, tachycardia, tachypnoea and hypotension. Rest of the general examination was normal. Systemic examination revealed pleural effusion on the right. Investigation revealed Hb- 6.5 gm%, Total leucocyte count 2500/cmm, and platelet count of 95000/cmm. Blood urea, serum creatinine, blood sugar F/PP, serum lipid profile were normal. Serum K+ and Na+ levels were 1.6 meq/l, and 136 meq/l, respectively. Arterial blood gas analysis revealed normal anion gap metabolic acidosis (pH – 7.2, PCO_2– 41.3 mmHg, Serum HCO_3- 9.6mEq/L, Anion gap- 11meq/l). ECG was suggestive of hypokalemia. Urine examination revealed Osmolality - 362 mosm/kg, (Plasma osmolality - 302 mosm/kg), Urine potassium 29 mEq/l, Urine sodium 129meq/L, urine chloride 147 meq/l, Trans tubular K+ gradient of 15 , urine anion gap of +3. Twenty four hour hr urine protein and phosphate were 232 mg, and 299mg respectively. Dipstick test for glycosuria was negative.

Urinary pH and CO_2 were 6.4 and 44 mmHg respectively. USG abdomen and kidneys were normal. Chest radiograph revealed right sided pleural effusion. Anti nuclear antibodies level, anti Ro/La were normal. HBS Ag and Anti HCV antibodies negative. Serum Caeruloplasmin, VitB 12 levels and bone marrow studies were normal. He was managed with intravenous sodium bicarbonate 50 mg in 500 ml DNS x 3 days followed by Tab Sodiumbicarbonate 2 gm QID. Potassium was supplemented to keep serum K+ above 3.5meq/l and the individual remains normokalemic on potassium supplementation even after two months.

Discussion

The relationship between HIV and RTA was first described by Laing in 2006 [3]. The incidence of distal RTA in patients with HIV is 9% [3]. In our case patient presented with hypokalemia preceded by diarrhea. Initially we managed diarrhea with fluid replacement and potassium supplementation. Hypokalemia persisted even after correction of fluid deficit and cessation of diarrhea and normalization of renal function. Investigations revealed hypokalemia with non anion gap metabolic acidosis and urine analysis revealed findings consistent with distal RTA. Distal RTA is usually associated with Fanconi's syndrome but in our patient no features suggestive of generalized tubulopathy or features of chronic distal RTA such as rickets or nephrocalcinosis were present. HIV induced distal RTA is usually associated with hypergammaglobulinemia, due to non-specific polyclonal plasma cell from B-lymphocyte differentiation [4]. but in our patient the

***Corresponding Author**:
Ivaturi Venkata Nagesh ,
Reader, Department of Internal Medicine, AFMC, Pune, India.
Tel: 009109618024173
E-mail: ivaturi_venkata@rediffmail.com

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globulins were just in the higher range of the normal. Our search for tuberculosis or anti tubercular therapy causing distal RTA, did not reveal any such association. Trimethoprim may cause distal RTA but is usually associated with hyperkalemia (Type 4 RTA) and hypokalemia persisted even after stopping sulfamethoxazole and trimethoprim tablets. Hence we propose that distal RTA in this patient was most probably due to HIV per se. HIV induced distal is due to the cytopathic effect sof the HIV virus itself and the mechanism is by apoptosis mediated by HIV proteins [5].

Conclusion

The present case demonstrated the difficulty in identifying renal tubular acidosis especially in an HIV patient with multiple co-morbid conditions and the importance of early recognition and treatment to avoid potential complications.

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