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Acute Hypokalemic Paralysis Secondary to Distal Renal Tubular Acidosis Presenting as Guillain Barre Syndrome

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ABSTRACT: A 52 year old female patient presented to emergency casualty with Acute onset quadriparesis with respiratory muscle involvement in. During initial work up Serum electrolytes showed severe hypokalemia. Arterial blood gas showed normal anionic gap metabolic acidosis with positive urine anion gap and urine examination showed alkaline PH in the presence of systemic acidosis. All these findings suggest Distal Tubular Acidosis. Further evaluation revealed strongly positive Anti nuclear antibodies with SS-A also being positive suggesting Sjogren's Syndrome.

KEYWORDS: Distal renal tubular acidosis, gbs, Hypokalemic paralysis, sjogren's syndrome.

BACKGROUND

Hypokalemic Periodic Paralysis of the extremities is a well known complication of hypokalemia due to Distal Tubular Acidosis.[1] [2] It almost resembles and can be misdiagnosed as Guillian- Barre Syndrome. Distal Tubular Acidosis is rather uncommon condition with complex pathophysiology that can present with life threatening electrolyte abnormalities.[3].Distal Tubular Acidosis is denoted by failure to acidify the urine in the distal part of the nephron. In children distal renal tubular acidosis is usually correlated with genetic defect or anatomic abnormality of urinary system, while in contrast Distal Renal tubular acidosis in adults is frequently related to the acquired conditions such as infections, drugs and autoimmune diseases.[4]

CASE REPORT

A 52 year old female presented with weakness of both upper and lower limbs which was sudden in onset associated with shortness of breath. She was a known case of Hypothyroidism. History of dry eyes is present. No history of fever, cough, cold, vomiting, diarrhea, paresthesias, neck pain, sensory disturbances, or headache. No history suggestive of recent drug use or native medication. No history of similar complaints in the past.

O/E: patient is conscious coherent, afebrile, pulse rate: 96bpm, BP:110/70mm Hg, SPO2:98% with Room Air. CNS Examination: Power:1/5 in all limbs, Hypotonia and Areflexia in all limbs. Sensory Examination, Cranial Nerve Examination Normal. No features suggestive of autonomic disturbances.

INVESTIGATIONS

Complete Blood Counts: Normal

Liver Function Tests, Kidney Function Tests are normal.

Sodium:136mmol/L, Potassium:1.8mmol/L, Chlorine:128mmol/L. Serum Calcium- 9.5mmol/L, Serum magnesium-2.0 mmol/L. Urine Potassium was 27.1 mmol/g , Trans tubular potassium gradient (TTKG) >4 indicating Distal K+ secretion. Arterial Blood Gas showed Normal Anion Gap Metabolic Acidosis. Urine pH was 7.5 and Urine Sodium: 148 mmol/L, Urine Potassium :27mmol/L, Urine Chlorine:104 mmol/L with positive urine anion gap.

Ultrasound abdomen showed few tiny caliculi in both kidneys.

Anti nuclear antibodies immune fluorescence was Strong Positive with Granular Pattern and SS-A was positive.

Schirmer's Test Showed dry eye features.

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DISCUSSION

A case of flaccid Hypokalemic Quadriparesis with respiratory involvement. Her clinical manifestations included severe muscle weakness and characteristic acid base disorders such as Hyperchloremic Normal Anion Gap Metabolic Acidosis and Severe Hypokalemia.[5] [6] Her urine study showed persistent alkaline urine, a positive Urine anion gap indicating distal Renal TubularAcidosis. On further evaluation, she was diagnosed with Sjogren's Syndrome.[4] She was treated with prednisone and Hydroxychloroquine in addition to Potasium and Bicarbonate supplementation which allowed to maintain adequate serum potassium levels.[5][7]

CONCLUSION

Cases of renal tubular acidosis should be carefully assessed for secondary causes to prevent progression to complications, and to uncover a potentially treatable condition which if untreated can unfold into chronic kidney disease. Repeated episodes of unexplained hypokalaemia could be a vital clue to a probable secondary cause. Awareness on part of treating Physician will assist in early diagnosis of such treatable conditions.

DECLARATION OF PATIENT CONSENT

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her clinical examination and information to be reported in the journal. The patient understand that her name and initials will not be published.

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