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Case Report

Distal renal tubular acidosis in systemic lupus erythematosus

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ABSTRACT

Systemic lupus erythematosus (SLE) is a multisystem disorder of autoimmune etiology. Renal involvement is frequently seen in SLE. Tubular dysfunction is also seen in SLE. Authors report a case of distal renal tubular acidosis in patient with SLE.

Keywords: SLE, Distal renal tubular acidosis, Hypokalemia

INTRODUCTION

Systemic lupus erythematosus (SLE) is the prototypic systemic autoimmune disease, characterized by diverse multisystem involvement and production of an array of autoantibodies.[1] Renal involvement is common in SLE and is a significant cause of morbidity and mortality.[2] Tubulointerstitial disease has been observed in as many as 66% of SLE renal biopsy specimens.[3] The presence of tubulointerstitial disease is a strong predictor of poor long term renal outcome.[4] Renal tubular acidosis is a manifestation of tubular dysfunction and can be seen in SLE and may present as chronic metabolic acidosis.[5] Authors report a case of SLE who presented with hypokalemia and was diagnosed with distal renal tubular acidosis.

CASE REPORT

A 38-year-old woman $G_3P_3L_3$ presented to the emergency with complain of rash over malar prominences, lesions on hands that started gradually in the last pregnancy at the gestation period of 7 months and progressed over 5 months after delivery. Patient also complained of oral ulcers, hair loss, weight loss, loss of appetite, easy fatigability, early morning stiffness that gets relieved within 30 min of work, low grade fever not associated with burning micturition, cough, pain abdomen, loose stools, vomiting, headache. She complained of weakness gradual in onset but progressive such that she had difficulty in combing her hair, getting up from sitting position but no history of difficulty in buttoning and unbuttoning of shirt or slipping of slippers. There was no history of blurring of vision, diplopia, facial asymmetry, sensory impairment or bladder or bowel disturbance. There was no history of band like sensation, root pains. There was no history of dryness of mouth, difficulty in swallowing, gritty sensation under eyelids. There was no history of any drug intake. She had clear sensorium.

On examination, blood pressure was 116/76 mmHg; pallor was present. There was no icterus, cyanosis, clubbing, edema and lymphadenopathy. There were multiple irregular shaped lesions on the palmar surface of the hands. These lesions had hyperpigmented rim at the periphery and hypopigmentation in the centre-Figure 1. Neurological examination revealed grade of muscle power in the proximal muscles of upper and lower limbs to be 4-/5 and muscles were tender on examination. Grade of power in distal muscles was 5/5 in both upper limbs as well as lower limbs. Deep tendon reflexes were intact and plantar reflexes were flexor. Chest auscultation revealed normal vesicular breath sounds and S_1 and S_2 were normally audible with no additional sounds audible.

The abdomen was soft non tender with normal bowel sounds.

Initial lab tests showed blood urea 32 mg/dl; creatinine 1.01 mg/dl; hypokalemia serum potassium 2.3 mmol/L. radiograph of the chest was unremarkable. Ultrasonography of the abdomen showed normal size and echotexture of liver and both kidneys. We kept possibility of inflammatory myopathy secondary to rheumatic disorder, thyroid disorder and acid base electrolyte disturbance and investigated her on the lines of our differential diagnosis. Further serological tests revealed ANA 152 (Negative <25), anti-ds DNA 2.10 (Positive >1.1) ESR 80 mm (normal 0-15) CRP 41.2 (Normal <6 mg/dl) Anti CCP 1.59 (Negative <25) Tests for HIV, hepatitis B and hepatitis C were negative. She was diagnosed as SLE based on SLICC criteria.[6] Investigations showed that our patient did not have inflammatory myopathy as her creatine kinase levels were normal and muscle biopsy was unremarkable. Thyroid function tests revealed no abnormality. Liver function tests were within normal limits. ABG analysis showed pH 7.21 chloride 112 mEq/L anion gap 6 mmol/L-hyperchloremic metabolic acidosis. Urine analysis showed urinary pH 6, positive urinary anion gap 4 mmol/L. Patient was started on oral potassium supplements, oral steroids, hydroxychloroquine 200 mg twice a day and has been asymptomatic on regular follow up Figure 2. Table 1 shows pretreatment and post treatment summary of the laboratory data.

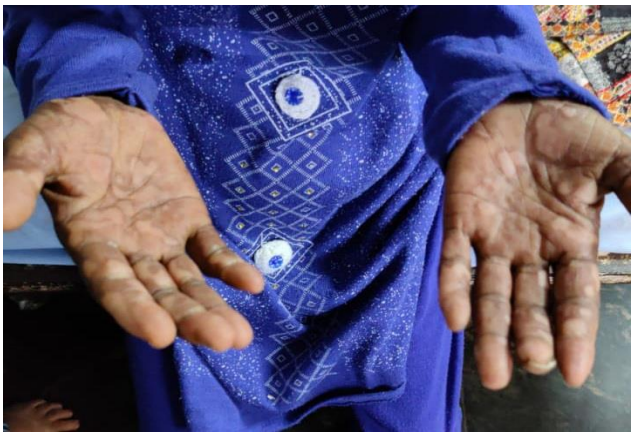

Figure 1: On presentation.

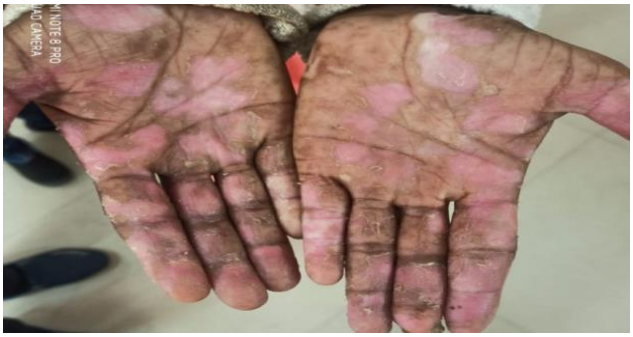

Figure 2: After treatment.

Table 1: Summary of laboratory data.

Parameters	Pre treatment	Post treatment
Arterial blood gas Ph	7.21	7.24
S. Potassium (mmol/L)	2.3	4.6

DISCUSSION

SLE is an autoimmune disease in which organs and cells undergo damage initially mediated by tissue binding autoantibodies and immune complexes. Nephritis is usually the most serious manifestation of SLE.[7] Severe tubular dysfunction is often seen in active proliferative lupus nephritis and is amenable to treatment with corticosteroids and cytostatics.[8] Distal renal tubular acidosis is not uncommon in SLE patients.[9] It leads to recurrent hypokalemia and acidemia, urolithiasis and nephrolithiasis.[10] In view of hypokalemia, hyperchloremic metabolic acidosis, positive urinary anion gap , the diagnosis of distal renal tubular acidosis was made in our case. Our case is considered to be acquired form of distal renal tubular acidosis. The pathogenesis of RTA has not clearly been elucidated and is presumed to be a sequel to deposition of immune complex.[11]Thus, tubular function should be carefully assessed in patients with SLE.

CONCLUSION

The evaluation of SLE patients with myalgias should not only focus on the work up inflammatory myopathy seen with SLE but should also focus on acid base disorder to identify an underlying tubular dysfunction.

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