DISTAL RENAL TUBULAR ACIDOSIS ASSOCIATED WITH AUTOIMMUNE HYPOTHYROIDISM-
A CASE REPORT
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PRESENTATION OF CASE
We report hypokalaemic quadripareisis presenting in a 45-year-old woman. Evaluation of the case revealed that hypokalaemic quadripareisis was secondary to underlying distal RTA (known as type 1 RTA), metabolic acidosis and alkaline urine. TSH was raised and anti-TPO antibodies were positive suggesting autoimmune basis for pathogenesis of functional tubular defect causing hypokalaemia. Bicarbonate therapy resulted in sustained clinical recovery.

DIFFERENTIAL DIAGNOSIS
In 1946, Albright et al described distal renal tubular acidosis as a distinct entity. The classical syndrome described consists of hypokalaemia, hyperchloremic metabolic acidosis, inability to decrease urinary pH below 5.5 in face of systemic acidosis, hypercalcaemia, hypotitraturia, nephrocalcinosis and nephrolithiasis, hypokalaemia, progressive renal failure and growth retardation. Additional features include osteoporosis, osteomalacia and autoimmune primary hypothyroidism. The syndrome was designated as distal RTA since the establishment of pH gradient between urine and blood is a function of distal nephron.

Renal Tubular Acidosis is non-uraemic defects of urinary acidification. RTA is characterised by normal anion gap, and hyperchloremic metabolic acidosis. Plasma K+ may be normal, low or high depending on the type of RTA. These syndromes differ from uraemic acidosis, which is associated with high anion gap, decreased GFR with enhanced H+ secretion by each remaining nephron. This disorder classically presents in familial form, but occasionally presents as a result of excessive gastrointestinal and/or urinary loss of K+. The disorder has been found endemic in Thailand, but has been reported in many countries as sporadic and/or hereditary form with variable modes of inheritance.

CLINICAL DIAGNOSIS
A 45-year-old female presented in emergency room with history of acute onset weakness of all four limbs. There was no preceding history of fever, drug intake, diarrhoea, trauma or headache. The patient had experienced similar episodes of weakness one year back and improved with some home remedies. She was never investigated further.

General physical examination was normal except for firm thyromegaly with mild tenderness. Neurological examination revealed grade 2 quadripareisis with depressed deep tendon reflexes. The rest of systemic examination was normal.

Routine investigations were normal except a serum K+ level of 1.26 mEq/L, pH of 7.23 pCO2 25.2 mmHg, pO2 87 mmHg and standard HCO3 16.2 mEq/L, TSH-49 IU/mL, FT3-0.79 pg/mL, FT4-0.65 ng/mL and anti-TPO~128.67 IU/mL. Serum urea was 17 mg/dL, creatinine was 0.7 mg/dL, glucose (F) 78 mg/dL, Ca2+ (ionised) 1.21 mmol/L and serum Na+ was 137 mg/L. Phosphate albumin, aspartate and alanine aminotransferase were normal.

Antibodies to nuclear antigen, antimitochondrial antibodies and anti-smooth muscle antibodies were negative. Anti-dsDNA, Anti-Sm, Anti-RNP, Anti-Ro and AntiLA were negative. Levels of serum immunoglobulins, compliments C3 and C4 were normal.

Ultrasoundography of abdomen was normal. Radiograph of chest and electrocardiogram were also normal. Urine was negative for blood, protein, glucose and urine microscopy was normal. Urine pH was alkaline at corresponding blood pH of 7.23. NCV and EMG were normal.

FNAC thyroid showed follicular cells, Hurthle cells and reactive lymphoid cells suggestive of lymphocytic thyroiditis.

In view of above clinical and biochemical parameters, a diagnosis of distal RTA presenting as hypokalaemic periodic paralysis with Hashimoto thyroiditis was established. Patient was treated with IV infusion of K+ and weakness improved over a period of three days. Patient was given L-thyroxine and Lugol’s solution at a dose of 1 mmol/kg body wt./day in divided doses. At the time of discharge, patient’s K+ was 4.2 mEq/L, chloride 102 mEq/L, pH 7.38 and HCO3 24.2 mEq/L. The patient is being followed up and remains asymptomatic.

DISCUSSION OF MANAGEMENT
Our patient presented with generalised weakness and was diagnosed as distal RTA and hypothyroidism on evaluation. The normal resting potential of myofibers is about ~85 mV at normal extracellular K+ of 4 mEq/L. Hyperpolarisation occurs with decreased extracellular potassium, which causes muscle excitability and thus weakness.

In renal failure, the underlying mechanisms of H+ secretion are preserved and kidney responds normally to systemic acid overload. However, in RTA, the kidney is not able to excrete an acid (H+) load and loses its normal acid-base balance. Distal RTA is a disorder of distal renal tubules, which is characterised by hyperchloremic metabolic acidosis, a normal serum anion gap, nephrolithiasis and daily
replacement needs of bicarbonate ≤ 4 mmol/kg. Distal RTA is the final common pathway for hypokalaemic paralysis in a variety of diseases of the endocrine disorders.⁵

RTA has been rarely reported in patients with thyroid dysfunction involving hyperthyroidism, Hashimoto’s thyroiditis and hypothyroidism. Various mechanisms have been reported in autoimmune thyroiditis leading to dRTA. One such mechanism is a defect in H⁺-ATPase pump of the cortical and/or medullary collecting tubules.⁶

Thyroid hormone increase membrane cell Na⁺, K⁺ and ATPase pumps.⁷ In hypothyroidism, the content and function of these pumps are decreased, which cause decreased elimination of H⁺, exacerbating the acidotic state caused by RTA.⁸

CONCLUSION
It is important to differentiate RTA from FPP. During an acute attack, administration of sodium bicarbonate in FPP would facilitate intracellular potassium influx and total hypokalaemia can occur. In FPP, oral K⁺ acetazolamide useful in preventing attack, but acetazolamide is contraindicated in dRTA as it produces acidosis. Recurrent hypokalaemic paralysis with apparently progressive symptoms should be evaluated for underlying disorders such as RTA.

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