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Internal Medicine

A RARE CASE OF VITAMIN D DEFICIENCY PRESENTING AS QUADRIPARESIS

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ABSTRACT Vitamin D deficiency is often underdiagnosed and has vague symptomatology & it is uncommon for Vitamin D deficiency to progress to Renal Tubular Acidosis 2, therefore leading to several electrolyte imbalances & motor weakness. We present a case of a young Indian female with a history of trivial fall followed by ascending weakness and numbness. After a careful review of the patient's history, evaluation and diagnostic testing led to the diagnosis of type 2 Renal Tubular Acidosis with Vitamin D deficiency. Patient improved with specific treatment for the aetiology i.e. Vitamin D deficiency and was discharged following improvement.

KEYWORDS : Quadriparesis, Vitamin D, Type 2 Renal tubular acidosis

INTRODUCTION

Vitamin D deficiency is a global public health issue. About 1 billion people worldwide have vitamin D deficiency, while 50% of the population has vitamin D insufficiency[1]. Eighty percent of adults and ninety six percent of elderly population in India are Vitamin D deficient.[2] The majority of patients with vitamin D deficiency are asymptomatic, and symptoms occurring at best are inconclusive to arrive at a diagnosis. Hence even with best of imaging and diagnostic modalities, It is difficult to pin point single aetiology, causing financial burden to the patient.

We present an Indian female patient who presents with a history of sudden onset of ascending weakness of both lower limbs along with numbness, the patient had a history of fall ~15 days back leading to #Right Distal radius. Eventually, after a careful review of the patient's history, and laboratory data, diagnostic testing led to the diagnosis of renal tubular acidosis type 2 (RTA 2) associated with vitamin D deficiency that resulted in significant hypokalaemia.

Case report

Mrs. A 42Y/Female, Housewife presented to our unit with history of sudden onset of ascending weakness and numbness of both lower limbs for past 3 days, the patient had a history of fall ~15 days back and on presentation GCS was E4V5M6, on general examination patient appeared well nourished, and cast was present on Rt forearm, CNS examination patient was conscious, oriented to time, place and person. Knee reflexes were found to be absent however ankle reflexes were preserved and quadriparesis was noted. On respiratory system examination patient had tachypnea 22/min, smooth but shallow respirations. CVS and P/a examination revealed no significant findings. All routine investigations were within normal limits except ECG which showed flattening of t waves and serum potassium level which was low (1.4 meq/L). Initial differential diagnosis was Gullian barre syndrome (GBS)/ transverse myelitis / suspected cervical cord contusion/ hypokalaemia induced.

MRI Brain with spine revealed a normal study which excluded the diagnosis of cervical cord contusion & transverse myelitis. Patient underwent nerve conduction velocity (NCV) testing in which no action potential was seen which was highly suggestive of GBS/ Hypokalaemia.

Further CSF testing revealed no significant abnormality or albumino-cytological dissociation; along with history and clinical examination, GBS and transverse myelitis were effectively ruled out. The probable diagnosis by exclusion was hypokalaemia Induced paralysis but as there was no history of similar episodes in the past in view of which the periodic paralysis was ruled out. As the patient gave a history of burning micturition and increase in frequency, hence sepsis profile was sent to rule out Sepsis, PCT and CRP were within the normal limits and also the cultures were negative effectively ruling out sepsis. In view of history of non- specific pains, and history of right distal radius fracture following fall, patient's vit d3 and vit b12 were sent, post which patients' vitamin D3 result came out < 3, however vitamin B12 levels were within normal limits.

Patient was managed conservatively by a team of doctors consisting of a neurosurgeon, neurologist and intensivist. Patient was put on potassium, vitamin D3 and calcium replacement, however despite the Potassium being replaced at almost 40meq/l/hr the patient's serum potassium continued to be between 1.5 to 1.9meq/l, raising the possibility of Renal tubular acidosis for which the patient's urine electrolyte /protein /HCO3 /creatinine were sent following which the diagnosis renal tubular acidosis type 2 was made in view of which bicarbonate supplementation was started and post which the patient serum potassium improved and reached a level of 3.5meq/l. The patient's motor functions also improved significantly with the return of the deep tendon reflexes. Infusion of potassium and sodium bicarbonate was stopped and tablet sodium bicarbonate was added and potassium was replaced when it fell below 3.5 mg/L. The patient was discharged to her house with advice for regular follow-up.

DISCUSSION

Severe vitamin D deficiency can present as osteomalacia in adults but the association of vitamin D deficiency with RTA 2 is a rare entity and there are only a few references in literature[3]. Failure to recognize the association between vitamin D deficiency and RTA 2 can lead to missed diagnoses, increased healthcare costs, and increased morbidity and mortality due to falls, secondary to weakness owing to the electrolyte imbalances of calcium, phosphorus, and potassium, hypokalaemia occasionally leading to death [4,5]. There are several aetiologies for calcium deficiency, one of which is the deficiency of vitamin D. Vitamin D increases calcium and phosphorus absorption in the intestines. In the absence of this action, calcium levels decrease in the human body, which leads to the secretion of parathyroid hormone from the parathyroid glands. The parathyroid hormone reacts by an increase in bone resorption as well as a decrease in calcium excretion in exchange for increased phosphate excretion by the kidneys. Our patient was diagnosed with RTA 2 owing to vitamin D deficiency. Vitamin D deficiency is commonly seen in patients who lack sufficient sun exposure for the normal synthesis of vitamin D. Briefly, the skin absorbs ultraviolet light from the sun and synthesises 7-dehydrocholesterol in the skin, which is metabolised to 25-hydroxyvitamin D in the liver by the action of the enzyme 25 hydroxylase. It is finally converted to its active form calcitriol (1, 25-dihydroxy vitamin D) in the proximal tubules of the kidneys through the action of the enzyme1- alpha-hydroxylase. Vitamin D deficiency results in low serum calcium and phosphorus, elevated alkaline phosphatase, and parathyroid hormone levels that in effect reduce the bicarbonate recovery at the proximal renal tubules[6].

Impaired reclamation of the bicarbonate ion in the proximal tubule leads to bicarbonate loss in the urine, but serum bicarbonate levels typically do not fall below 15 mEq/L because of the ability of the collecting ducts to reclaim some of the bicarbonate missed by the proximal tubules[7]. Patients with RTA 2 generally have hypokalaemia and increased urinary potassium wasting due to increased urine flow to the distal nephron caused by the distal delivery of bicarbonate ions. The renin-angiotensin-aldosterone system is also activated from the

mild hypovolemia induced by bicarbonate loss in urine, leading to increased collecting duct sodium reabsorption and potassium excretion[7]. Administration of alkali in these patients increases bicarbonate wasting in urine and can worsen hypokalaemia unless potassium is replaced simultaneously[7]

Isolated proximal type 2 RTA is characterised by defects in the reabsorption of filtered HCO3 – in the proximal tubule without defects in the transport of other solutes. The threshold serum concentration for HCO3 – reabsorption (normally approximately 25 mmol/L) is reduced, leading to delivery of larger quantities of filtered HCO3 – to the distal nephron (which has a low capacity for HCO3 – reabsorption) and urinary HCO3 – wastage. Reductions in serum HCO3 – cause acidosis; however, the urine pH remains alkaline because of the presence of urinary HCO3- . When serum HCO3 – concentrations decrease below the lower threshold (16–20 mmol/L), a new steady state is reached, whereby all filtered HCO3 – is reabsorbed.

At this point, the urine contains no HCO3 – and is maximally acidic. A diagnosis of proximal RTA may be suspected in patients who present with hypokalaemia, normal anion gap metabolic acidosis, and acidic urine (pH 5.5).

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Take Home Message

This case report is presented as a gentle reminder to consider vitamin D3 deficiency-induced type 2 RTA as a differential diagnosis while evaluating a case of quadriparesis and hypokalaemia.

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Conflict of interest statement

The authors declare that there is no conflict of interest.

Specialized Chemistry	Reference Values
Potassium = 1.4meq/l	3.5-4.5 meq/l
Phosphorus = 1.6 mg/dl	2.5-4.5 mg/dl
Bicarbonate = 18.3meq/dl	22-26meq/dl
Fraction excretion Bicarbonate = 27%	5-15%
Calcium = 6.58 mg/dl	8.4-10.2 mg/dl
Vitamin D = <3 ng/ml	30-100ng/ml
Parathyroid Hormone = 423.80 pg/ml	14-72 pg/ml

Table 1. Laboratory Values

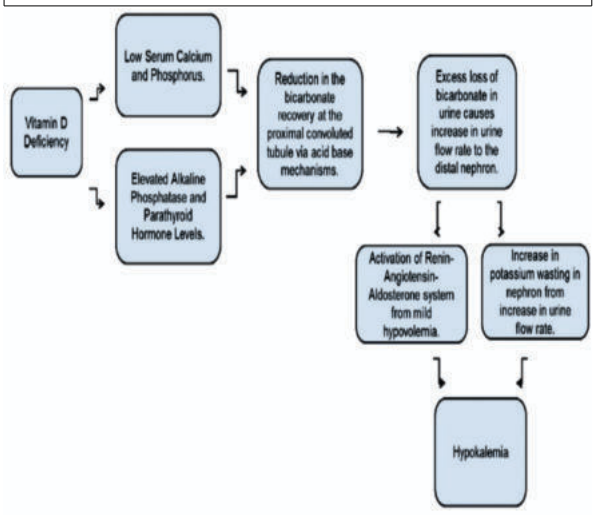

Figure 1. Vitamin D Deficiency leading to Hypokalemia Causing Weakness.

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