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A CASE OF SUDDEN ONSET OF WEAKNESS IN ADOLESCENT MALE : HYPOKALEMIC PERIODIC PARALYSIS

General Medicine

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ABSTRACT

Hypokalemic periodic paralysis is an uncommon neuromuscular disorder characterised by transient episodes of flaccid muscle weakness. Secondary hypokalemic periodic paralysis is caused by loss of potassium from kidney/gastrointestinal tract/skin. Here I report a case of sudden onset of weakness of all four limbs in an adolescent patient due to Hypokalemic periodic paralysis secondary to renal tubular acidosis type 2, improved on treatment with potassium and bicarbonate supplements resulting in no residual weakness.

KEYWORDS

INTRODUCTION:

Hypokalemic periodic paralysis is the most common of the periodic paralysis, but is still quite rare, with an estimated prevalence of 1 in 100,000 . Hypokalemic Periodic paralysis (PP) is a rare neuromuscular disorder related to a defect in muscle ion channels, characterised by episodes of painless muscle weakness, which may be precipitated by heavy exercise, fasting, or high-carbohydrate meals. Periodic paralysis is classified as hypokalemic when episodes occur in association with low potassium blood levels or as hyperkalemic when episodes can be induced by elevated potassium. Most cases of PP are hereditary, usually with an autosomal dominant inheritance pattern. Acquired cases of hypokalemic Periodic paralysis have been described in association with hyperthyroidism.

CASE REPORT:

A 18 year right handed boy studying in 12th standard, born from non consanguineous marriage belonging to lower socioeconomic class presented with complain of weakness in both legs more over left side since 2 days which was associated with difficulty while getting up from sitting position-initially required one hand support which was gradually progressed over three days that he is unable to get up independently , squatting position and climbing stairs. (Not associated with wearing/slippage of chappals/no sensory abnormalities) which progressed to involve a similar type of weakness in the right leg. Then after he developed weakness in bilateral arms in the form of difficulty putting objects above the head shelf and combing hairs. Then he developed difficulty in raising head above the pillow and turning side by side on the bed. However he didn't have difficulty in breaking chapatis, buttoning-unbuttoning. At present the patient is dependent for his daily activities.

No history of similar complain
No history of abnormal enlargement of calf muscle.
No history of difficulty in releasing objects after tight grip.
No h/o pain/myalgia/cramps/contractures
No h/o difficulty closing eyes, whistling or drinking through straw
No h/o change in voice (nasal intonation) or difficulty in swallowing or nasal regurgitation of fluids
No h/o drooping of eyes/diplopia/facial numbness/facial deviation/hearing loss
No h/o cola coloured urine/ easy fatigability /exercise intolerance
No h/o tightness of limbs
No h/o tingling /numbness / paresthesias
No h/o thinning of limbs or rippling movement under the skin
No h/o diurnal variation
No h/o chest pain /palpitation
No h/o urinary frequency /urgency/ incontinence
No h/o sudden darkening of visual fields on standing / palpitation / diarrhoea /constipation/excessive sweating or dryness of skin
No h/o seizure/altered sensorium/difficulty speaking/understanding what other are saying/cognitive impairment
No h/o fever/ rash / joint pain / dryness of mouth / oral genital ulcers
No h/o weight gain/ loss
No h/o ayurvedic medicine
No h/o drug/toxin exposure
No complaints of neck pain/back pain. No history of trauma/insect bite
No complain of rashes, joint pain, dryness of mouth,dryness/redness of eyes, parotitis, raynaud's,dyspnea on exertion, frothing from urine, photosensitivity
No past history of DM / TB / HTN / Thyroid disorder / CAD /jaundice

Born as full term normal delivery with adequate weight, operated for cleft palate and lip soon after birth, fully vaccinated. Having two siblings and parents with nil comorbidities.

On examination: Conscious/oriented/cooperative/comfortably lying on bed.
Temp= Normal
Pulse= 60/min
Bp=110/70 mmhg
Rs= Blae + , Clear ,No added sounds
Cvs= s1s2
Cns=
Single Breath Count: >35
Higher Mental functions: NAD
Pupils: B/L equal sized reactive to light
Tone: Bilateral upper and lower limb hypotonia
No wasting
Power: Neck flexors weakness
2/2//1/1 across all range of joint movements of upper and lower limb
DTR: Absent
All 12 pairs of cranial nerve examination normal
Planter: Bilateral flexors
Sensory Examination: NAD
With normal back and spine
Cerebellar examination normal

P/A: Soft, NT

INVESTIGATION:

- ECG: ST depression , Presence of U waves
- Cbc - 10.2/7890/195
- TSh : 3.81
- HBA1C: 5.2
- CPK: 1355
- ANA: Negative
- MRI Brain with WSS: NAD
- Vitamin B-12: 1687
- ABG: hyperchloremic metabolic acidosis
- Urinary ph : 5.4
- Plasma bicarbonate : 13 mmol/l
- Renal Function Test: 0.90/32.3/139/2.2
- USG ABDOMEN : NAD

DIAGNOSIS: Hypokalemic Periodic Paralysis Due to Renal Tubular

Acidosis Type 2

Patient has been treated with Injectable Potassium correction with oral supplements. Serum Potassium improved to 4.2 mEq/L with symptomatic improvement (Power improved to 4+ in bilateral upper and lower limbs with all range of joint movements and reestablishment of DTRs) After that bicarb therapy was given and continued.

DISCUSSION:

Hypokalemic periodic paralysis is an autosomal dominant disorder with onset in adolescence with Male preponderance . Exacerbating factor being meals high in carbohydrates or sodium or after prolonged exercise.

Weakness usually affects proximal limb muscles. Ocular, bulbar muscles and respiratory muscles are mostly spared. A low serum potassium level during an attack, excluding secondary causes, establishes the diagnosis.

In the midst of an attack of weakness, motor conduction studies may demonstrate reduced amplitudes, whereas EMG may show electrical silence in severely weak muscles. Mild attacks usually do not require medical treatment. However, severe attacks of weakness can be improved by the administration of potassium. Oral KCl (0.2–0.4 mmol/kg) can be given every 30 min. Only rarely is IV therapy necessary (e.g., when swallowing problems or vomiting is present). In proximal (type 2) RTA, the proximal tubule has a decreased capacity to reabsorb filtered bicarbonate. Renal NaHCO3 losses lead to intravascular volume depletion, which in turn activates the renin-angiotensin-aldosterone system. Because of the associated hyperaldosteronism and increased distal nephron Na+ reabsorption, there is increased K+ secretion. The net result is renal potassium wasting and the development of hypokalemia.

CONCLUSION:

Rapid identification of correctable electrophysiological parameters.

The long-term goal of therapy is to avoid attacks. Patients should be made aware of the importance of a low-carbohydrate, low-sodium diet, and consequences of intense exercise. Prophylactic administration of acetazolamide or dichlorphenamide can reduce attacks of periodic weakness.

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