Case Report

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Hypokalemic Periodic Paralysis Mimicking as CVA

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Abstract

Hypokalemic periodic paralysis (hypoKPP) is characterized by muscle weakness or paralysis along with fall in potassium levels in the blood . This clinical entity is seen in adolescence and most commonly occur on awakening or after sleep or rest following strenuous exercise, high carbohydrate meals, meals with high sodium content, sudden changes in temperature. Weakness may be mild and limited to certain muscle groups, or more severe full-body paralysis. Attacks may last for a few hours or persist for several days. Some patients may fall into an abortive attack or develop chronic muscle weakness later in life.

Keywords: Hypkalemia; Paralysis; CVA; Potassium.

Introduction

Hypokalemic periodic paralysis (hypoKPP) is a rare, autosomal dominant channelopathy characterized by muscle weakness or paralysis with a matching fall in potassium levels in the blood (primarily due to defect in a voltage-gated calcium channel). In individuals with this mutation, attacks often begin in adolescence and most commonly occur on awakening or after sleep or rest following strenuous exercise (attacks during exercise are rare), high carbohydrate meals, meals with high sodium content, sudden changes in temperature, and even excitement, noise, flashing lights and induced by cold temperatures. Weakness may be mild and limited to certain muscle groups, or more severe full-body paralysis. Attacks may last for a few hours or persist for several days. Recovery is usually sudden when it occurs, due to release of potassium from swollen muscles as they recover. Some patients may fall into an abortive attack or develop chronic muscle weakness later in life.

Treatment of hypokalemic periodic paralysis focuses on preventing further attacks and relieving acute symptoms. Avoiding carbohydrate-rich meals, strenuous exercise and other identified triggers, and taking acetazolamide (Diamox®) or another carbonic anhydrase inhibitor, may help prevent attacks of weakness. Some patients also take potassium-sparing diuretics such as spironolactone (Aldactone®) to help maintain potassium levels. Paralysis attacks can be managed by drinking one of various potassium salts dissolved in water (debate exists over which, if any one in particular, is best used, but potassium chloride and bicarbonate are common). Rapidly absorbed boluses of liquid potassium are generally needed to abort an attack, but some patients also find positive maintenance results with time-released potassium tablets. IV potassium is seldom justified unless the patient is unable to swallow. Daily potassium dosage may need to be much higher than for potassium replacement from simple hypokalemia: 100-150 mEqs of potassium is often needed to manage daily fluctuations in muscle strength and function.

Here in this case report we present such a case of hypokalemic paralysis initially mimicking as CVA.

Case Report

A 30yr old male patient presented to the emergency department with complaints of fever since 3 days,

associated with generalised weakness, excessive joint pain, inability to move both left lower limbs and left upper limb since last night.There is no history of any loss of consciousness and no seizure. He is otherwise very fit and is not having any major medical problems and not on any regular medications. There is no history of any loose motion and no vomiting and no history of any recent travel. There is no history of any sick contact at home. He also gave the history that the fever started 3 days ago and he took some paracetamol tablets only. There is no associated cough and there is no expectoration. Today he came to the ED as the fever has subsided but the joints pain is very sever and since last night he started difficulty in moving his left side of the body. There is no headache and there is no associated slurring of speech and no deviation of the mouth. But he is having some nausea and body pain was still persisting.However he had 4-5 episodes of loose motion and mild abdominal cramps. No blood or mucus in the stool.

On Examination

Airway Assessment : Patent and talking and protected

Breathing Assessment

Respiration(RR / min) :22 / Min

Laboured: No

SpO2: 98% on Room Air

Circulation

Pulse :88 / Min

BP :120 / 70 MM HG

Peripheral Pulses :Yes

Temperature :98 F

GRBS : 120mg / dl

Disability: GCS-15 / 15, Conscious and oriented and normal Higher Mental function and oriented to time, place and person.

Pupil:- B / L equal and equally reacting

Sensory examination: Normal B / L

Motor function

power: Right upper limb -5 / 5

Left upper limb -2 / 5

Both lower limbs -2 / 5

B / L plantar-mute

DTR:- WNL

No deviation of angle of mouth

No Nystagmus and no Neck rigidity.

Review of Systems

HEENT: No pallor, No icterus, No scars, No JVD and No neck swelling.

CHEST: Normal Vesicular breath sound heard, b / l air entry equal, no adventitious sounds

CVS: s1s2 heard no murmurs

ABD: soft non tender no organomegaly, bowel sounds+

EXT: warm, no edema

Past Medical History: Nothing Significant

Past Surgical History: Nothing Significant.

Hospital Course

1. Immediate Neurology Consult was taken in view of significant Neurological finding and with a differential of CVA.
2. Iv fluid started with 0.9% NS @ 100ml / hr
3. Inj. Paracetamol 1000mg iv stat
4. Inj. Pantoprazole 40 mg iv stat
5. ECG done- Showed NSR @ 88 / m and no ST-T changes and no ectopy.
6. VBG was sent
7. Plan for an Imaging taken: MRI brain was orderd after discussion with the Neurologist.
8. Admission was planned in ICU
9. Routine investigations sent: CBC, LFT, RFT, Blood C / s, Urine C / s, Chest X-ray , Serum Electrolytes sent.
10. Inj. Monocef 1gm started empirically , pending culture report.

While in the ED the VBG done and was found to be normal with no acidosis and normal Lactate.

The patient was sent for MRI Brain to rule out any Stroke. But the MRI came out to be normal with no abnormality .

The patient was then shifted to ICU. After admission in the ICU the rest of the blood reports were chased.

CBC- N55, TLC 18000, E 0, M 0, HB 12.2, PLATELET 3.4 LAKH, ESR 2 0

LFT- SGOT / SGPT WNL, TOTAL BIL 1.1,

RFT- WNL

ELECTROLYTES- Na-135, K(Potassium)-2.3mg / dl, Mg-normal.

Chest X ray –normal

Blood C / S and Urine C / S was sterile after 48 hrs of culture.

Treatment in ICU (on follow up)

1. IV fluid with KCL 40meq in 500ml 0.9%NS started @ 100ml / hr
2. Syp PotKlor 60ml thrice daily with lime juice PO.
3. Inj. MVI 10ml amp inj 10ml iv q24h(14hrs) in 500ml dns iv over 6 hours
4. Econorm sachet (250mg) BID
5. IV fluid with alternate RL& NS to continue.
6. IV Monocef 1gm BID.
7. Inj. Pantoprazole 40mg iv BD.

Course in the Hospital

Patient continued to do well in the ICU and after 24hrs of ICU stay there was significant improvement in the limb strength. The patient had no other complain and the fever also did not recur and the headache and joint pain also subsided. Regular clinical monitoring was done and the patient was shifted to the ward. On the 4th day the patient had completely gained strength in all the 4 limbs and the neurological examination was found to be normal and was planned for discharge in stable condition. The repeated Serum Potassium level was found to be 4.6mg / dl and TLC count settled to 7000. He completely improved and was discharged with the following medications and follow up in the OPD.

Discharge Medications

1. Potklor Liquid: 60ml Thrice Daily with Lime Juice.
2. Tab Shelcal 500mg Twice Daily After Food.
3. Tab Pantocid 40mg before Breakfast.
4. Tab Zifi 200mg Twice Daily After Food.
5. Tab Zincovit after Lunch.

Conclusion

This above case report shows how confusing the clinical presentation can be. The gross neurological deficit in this patient was initially thought to be due to CVA and in view of that a MRI imaging was also done. But a electrolyte level changed the whole diagnosis . Usually Hypokalemia is accompanied by Hypomagnesemia, but in our patient the serum Magnesium Level was normal. Although the patient improved but such patient can again return back or land up with similar issues. So it is mandatory to counsel such patient and take necessary actions and follow a potassium rich diet. The best source will be to drink coconut water and banana, which has a very rich source of potassium. From the perspective of Emergency Medicine , in such cases we should keep our mind broad and consider a wide variety of differential diagnosis. We can then rule out the major causes and reach a final diagnosis. Although the case was was managed proficiently but the clinical neurological finding do mimick an acute cerebral / neurological insult.

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