

Hypokalemic periodic paralysis: 2 novel causes

M Gupta, S Lehl, R Singh, A Sachdev

Citation

M Gupta, S Lehl, R Singh, A Sachdev. *Hypokalemic periodic paralysis: 2 novel causes*. The Internet Journal of Neurology. 2008 Volume 12 Number 1.

Abstract

INTRODUCTION

Hypokalemic periodic paralysis is a rare disorder of muscle characterized by episodes of mild to severe muscle weakness associated with hypokalemia. The defect lies in the voltage-gated calcium or sodium ion channels which are mutated, resulting in abnormal sarcolemmal excitation. Upper respiratory tract infections both viral and bacterial are known to trigger a bout of hypokalemic periodic paralysis, but there is paucity of literature on chickenpox (varicella) and dengue fever precipitating such an attack. The following cases highlight this unusual presentation.

CASE REPORTS

CASE 1

A 26 year old male presented to our emergency with two day history of high grade fever, generalized body pain and one day history of gradually progressively increasing weakness of both the lower legs. Over the past few hours he noticed the weakness spreading and involving both the upper limbs also. There was no history of recent vigorous exercise or a high carbohydrate meal. He was competently bedridden when seen in the emergency department with power in the upper and lower limbs grade 1 and generalized areflexia. There was no sensory deficit and no evidence of any bowel or bladder involvement. There was no respiratory distress. There was no evidence of diplopia, ptosis or difficult deglutition.

He reported having two such episodes previously in last six months which had improved after hospitalization and intravenous fluids. On review of the records he was found to have hypokalemic periodic paralysis in the past and was not on any medication. His renal and thyroid functions were within normal limits. The serum sodium was normal but potassium was 1mEq/l. Arterial blood gases revealed a non-anion gap acidosis. Urine analysis showed a pH of 5.9, a specific gravity of 1.005 and no proteinuria or glycosuria.

Over the next day the patient developed vesicular lesions on the face and the trunk suggestive of chickenpox (Figure1). Patient's neurological symptoms resolved with potassium and bicarbonate supplementation in normal saline. On the fifth day he was discharged with the advice to continue taking potassium and alkali supplements.

Figure 1

Figure 1: Vesicular lesions on the trunk suggestive of chickenpox



CASE 2

A 24 year old male presented to emergency with a three day history of high grade fever associated with headache, severe myalgias and one day history of petechial skin rash on the lower extremities and progressively increasing weakness of legs. On examination he was conscious and oriented, had conjunctival suffusion, petechial rash on the lower legs and forearms. His vitals were stable. Neurological examination revealed proximal muscle weakness and generalized hyporeflexia. There was no sensory abnormality and bowel or bladder involvement. Other systemic examination was unremarkable except for mild hepatomegaly. There was no history of vomiting or diarrhoea. He had one episode of similar symptomatology an year back following gastroenteritis. Laboratory investigations revealed a raised hematocrit and a platelet count of 20,000/ul. IgM dengue serology was positive. Patient had normal renal functions

with a potassium value at admission of 1.5mEq/l. Arterial blood gases was suggestive of normal anion gap acidosis. Urine analysis showed a pH of 5.5 without proteinuria or glycosuria. He was started on oral and intravenous potassium supplementation in normal saline. The clinical finding of hypokalemia improved over next 2 days. His thyroid functions were within normal range.

DISCUSSION

Hypokalemic periodic paralysis can be primary or secondary¹. Secondary hypokalemic periodic paralysis is a sporadic disease with individuals having their first attack in adulthood with some identifiable underlying cause or trigger such as thyrotoxicosis, barium or licorice ingestion, primary hyperaldosteronism and potassium wasting disorders. Potassium wasting may be gastrointestinal or renal. Commonly renal tubular acidosis is the underlying etiology in secondary hypokalemic periodic paralysis.

Hypokalemic periodic paralysis can be induced by a variety of triggers in susceptible individuals. The list of triggers varies from individual to individual. Most consistent of these are rest after exercise and a high carbohydrate meal. High Sodium intake, lack of sleep, menstrual cycle and pregnancy, changes in humidity, alcohol intake and dehydration may also induce attacks; these different triggers need to be identified and individualized for every patient. Certain medications like insulin, beta-agonists and corticosteroids have also been incriminated as triggers.

Pathological conditions that can precipitate hypokalemic periodic paralyzes include upper respiratory infections (both viral and bacterial) including influenza, fever, diarrhea and vomiting, intercurrent illness or surgery and Type 1 diabetes mellitus. However there is not a single case report of primary varicella infection or dengue fever precipitating an attack of hypokalemic paralysis as of date.

Many neurological complications are known to occur with varicella infection, estimated incidence being 0.01–0.03%². The most common manifestations are cerebellar ataxia, encephalitis, transverse myelitis, aseptic meningitis,

Guillain-Barre syndrome, delayed contralateral hemiparesis, optic neuritis, motor neuropathy and facial palsy. Acute flaccid paralysis secondary to hypokalemic periodic paralysis as a complication of varicella infection has not been reported in medical literature.

Dengue fever is also known to be complicated by neurological syndromes which include encephalitis, meningitis, seizures, myelitis and acute flaccid paralysis due to Guillain-Barre syndrome³. Recently there have been case reports of quadriplegia caused by dengue myositis, acute transverse myelitis^{4,5}. However hypokalemic periodic paralysis as a manifestation of dengue fever has not been previously described.

Treatment of hypokalemic periodic paralysis includes correction of life threatening hypokalemia and underlying disease. Maintenance of serum potassium levels within normal limits with potassium supplementation is imperative to decrease attack frequency. It is also necessary to identify and avoid the specific triggers.

CONCLUSION

Both our cases highlight novel and unusual presentation of acute viral diseases namely chickenpox and dengue fever. These diseases are increasingly presenting with neurological symptomatology and thus clinicians need to have a high index of suspicion for above viral illnesses while dealing with neurological cases.

References

1. Gutmann L. Periodic paralyzes. *Neurol Clin* 2000;18:195-202
2. Guess HA. Population-based studies of varicella complications. *Pediatrics* 1986;78:723–7.
3. Jackson ST, Mullings A, Bennett F, et al. Dengue infection in patients presenting with neurological manifestations in a dengue endemic population. *West Indian Med J*. 2008 Sep;57(4):373-6.
4. Kalita J, Misra UK, Mahadevan A, Shankar SK. Acute pure motor quadriplegia: is it dengue myositis? *Electromyogr Clin Neurophysiol*. 2005 Sep-Oct;45(6):357-61
5. Seet RC, Lim EC, Wilder-Smith EP. Acute transverse myelitis following dengue virus infection. *J Clin Virol*. 2006 Mar;35(3):310-2.

Author Information

M Gupta

Department of Medicine, GMCH, Chandigarh, India

SS Lehl

Department of Medicine, GMCH, Chandigarh, India

R Singh

Department of Medicine, GMCH, Chandigarh, India

A Sachdev

Department of Medicine, GMCH, Chandigarh, India