Protean Neurological Manifestations in Chikungunya

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Abstract

Unplanned urbanization and secondary migration has caused increased spurt in arboviral diseases especially Dengue and Chikungunya. With this exponential rise in these illness, now we are beginning to notice uncommon presentations of these common illnesses. Here we present two interesting cases: one of paraparesis and another of quadriparesis with respiratory involvement secondary to Chikungunya, although the mechanism in one is hypokalemia and the other is GBS secondary to Chikungunya. Just the magnitude of cases presenting in metros and major cities of our country warrant sensitizing the physicians about these uncommon manifestations.

Introduction

Chikungunya, an arboviral disease transmitted by Aedes mosquitoes is gaining significant public health proportions due to rapid migration, unplanned urban settlements and poor sanitation. Usually the disease passes off harmlessly in 5 to 7 days; with few patients suffering from fatigue and post fever arthralgia. Delhi this year experienced an exponential rise in the number of Chikungunya cases withsome patients presenting with unusual clinical manifestations. Neurological syndromes are rarely known to occur with chikungunya barring few reported cases of encephalitis, encephalopathy or myeloneuropathy. We present two cases who presented with sudden onset limb weakness. The attributable aetiology for both was chikungunya, but the underlying causative mechanisms were eclectic.

Case 1

A 40-year-old male with no previous co morbidities presented with complaints of fever since three days, which was continuous and high grade with associated chills. Fever was accompanied by multiple joint pains. This was followed two days later by bilateral lower limb weakness with subsequent involvement of both upper limbs. He, however did not give any history of loose stools, vomiting, altered sensorium, rash, breathlessness, hoarseness of voice, dysphagia, difficulty in closing his eyes or band like sensation around his waist. Also, patient did not give any history of sensory complaints. He did not have any similar complaints in past. Patient had been residing in a slum for past 03 years which had ubiquitous mosquito breeding sites.

At presentation, his vitals were stable. Power in bilateral lower limb and upper limb were 2/5 and 3/5 respectively. Deep tendon reflexes were absent in both upper and lower limbs and bilateral plantars were flexor. No sensory deficit was noted. All other systemic examination was normal. Lab investigations revealed a Hemoglobin of 12.0gm%, TLC: 8000/mm3, PLATELET: 1.8L/mm3, PCV: 33.6%. Kidney and liver function tests were normal. Hypokalemia with a Potassium of 2.3meq/L was documented. His ECG also showed prominent U waves in precordial leads NCCT head and CSF evaluation was normal. Patient was started on oral and parenteral potassium supplements. Recovery of power was seen in all the 4 limbs within two days of initiating treatment.

Further, investigations were done to find the underlying association for Hypokalemia. S. magnesium levels were normal. ABG was normal with a pH of 7.41. 24-hour urinary potassium was 15mmol/L. Thyroid function with a TSH of 1.553micro IU/L, FT3 of 2.74pg/mL ad FT4 of 1.21ng/dL was also normal. CPK and CK-MB were also in normal range. In view of acute febrile illness with polyarthralgia, malarial, dengue and chikungunya serologies (IgM) were sent. In addition, a malaria antigen kit was also done, the result of which was negative. Later on, chikungunya serology came out to be positive. NCV and EMG were planned but couldn't be undertaken as power along with reflexes had recovered in the affected limbs.

Patient was managed conservatively with antipyretics and continued potassium supplementation. At the time of discharge (post admission: day 4), power in all four limbs was 5/5 and serum potassium was 4.9 meq/L.

Case 2

A 45 years old male presented with a history of progressive ascending quadriparesis involving both lower limbs simultaneously followed by upper limb involvement in a span of 2 days. There was an associated history of hoarseness of voice which was not associated with visual changes, facial deviation, slurring of speech or nasal regurgitation.

Bowel and bladder were not involved and no seizure, headache or vomiting was present and there were no sensory complaints. No back pain or band like sensation or pain during flexion of neck was present. Patient however had a history of fever for 3 days associated with multiple joint pains from 10 days with no associated GI or respiratory symptoms.

On examination, patient was conscious oriented having BP of 130/80 mmHg, pulse 100/min with respiratory rate of 20/min and single breath count of 20. On neurological examination there was no facial deviation or loss of wrinkling of forehead and bilateral uvula moved normally with vocalization however gag reflex was absent. Tone was flaccid in all four limbs and power in lower limb and upper limbs were 2/5 and 3/5

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respectively. Deep tendon reflexes in all four limbs were absent. Other systemic examination was unrevealing.

NCV was done which showed acute motor axonal and demyelinating neuropathy. Blood studies showed Hb 12g/dl with 8000/cumm of TLC and 1.5 lac/cumm of platelet count with other biochemical test within normal range including serum potassium of 4.1 meq/L.

Work up for fever showed negative results for dengue and malaria but chikungunya IgM serology in blood was positive. CSF examinationdoneshowed albumin-cytological dissociation with sugar of 40 mg/dl and protein being 2 g/L with absence of cells. Chikungunya serology in CSF was negative. ANA and anti-ganglioside antibodies were negative.

Based on all the above findings a diagnosis of post- chikungunya Guillain Barre Syndrome was made and patient was started onintravenous immunoglobulins in standard dose for 5 days however the illness progressed with absent movement of all four limbs and respiratory involvement. Thus, patient was intubated on 3rd day of admission and put on ventilator. Currently the patient is tracheostomized and on SIMV mode of ventilation with no progression since past week.

Discussion

Chikungunya fever is an arboviral disease that usually manifests as a self-liming disease with high fever, severe arthralgia, myalgia, and maculopapular rashes. Rare but severe complications may occur, such as myocarditis, hepatitis and neurological manifestations.[2] Unlike our patients, most of these complications are seen in elderlypatients or those with underlying medical illnesses[2].

Various neurological sequelae have been known to occur with chikungunya fever, with peripheral neuropathy having predominant sensory component being the most common. Neurological tropism of chikungunya virus seems to be lower than of other arboviruses such as Dengue, West Nile or Yellow fever viruses, yet several studies have described, (especially during epidemics) neurological manifestations.[3] Just the magnitude of cases presenting in metros and major cities of our country warrant sensitizing the physicians about these uncommon manifestations.

Most common diagnostic dilemma remains whether neurological symptoms are due to persistence of the virus or an inappropriate immune response. In some cases, IgM antibodies against chikungunya virus have been found in CSF of patients with meningitis, supporting the theory of neuroinvasion.[4,5] However, in our view a negative CSF serology and albumin-cytological dissociation we made a diagnosis of post- chikungunya Guillain Barre Syndrome and treated accordingly.

Hypokalemic paralysis is known to be precipitated by infections. Other precipitating causes include high carbohydrate diet, exposure to cold and strenuous exercise. Viral fever, particularly dengue infection has been extensively found to be associated with hypokalemic paralysis. Considering the existing similarities between dengue and chikungunya, the latter may also be an important cause for hypokalemic paralysis. The exact mechanism however is not known. Possible mechanism could be viral infection causing catecholamine release which causes redistribution of potassium within the cells. Another possible mechanism can be inadequate potassium intake precipitated by fever and dehydration.

There is no recommendation of supplementing potassium (post correction) on a long term basis after having ruled out other causes of hypokalemia

In both the cases no sensory involvement was noted. It is to be noted that though the patients were middle aged and no associated comorbities were present; still they presented with atypical complications. Association between hypokalemic paralysis and chikungunya is not commonly seen as in the presentation of GBS with bulbar involvement secondary to chikungunya. Thus, we present these cases of Chikungunya with rare neurological manifestations.

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