*Case Report*

# Neurological Complication of Chikungunya Fever: Guillain Barre Syndrome - A Case Report

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**Abstract:**

*Chikungunya fever has been known as reemerging disease since 2005. Its feature is more or less like dengue fever. Major outbreak occurred in Bangladesh in 2017. Lot of complications can occur in patient suffered from it. We report a Chikungunya case presenting with Guillain-Barré syndrome (GBS) with sensory involvement and bowel- bladder dysfunction who responded to plasma pharesis. Laboratory parameters and temporality support that GBS in the complication of Chikungunya.*

**Key words:** Chikungunya, Guillain-Barré syndrome

## INTRODUCTION

Chkungunya is an Alpha virus. It is transmitted to human by bite of Aedes mosquito[1]. It was first isolated in 1952 in Tanzania and has been known as a reemerging disease since 2005[2]. Major outbreak occurred in Reunion Island in 2004 which involved 38.2% of 785000 inhabitants[3]. Chikungunya strain was isolated in India during the 2004-2006 outbreak[4]. In Bangladesh major outbreak occurred in 2017 in Dhaka city. Chikungunya fever is characterized by an abrupt onset of fever and moderate to severe arthralgia, myalgia, headache, rash, fatigue and nausea[5]. Unusual manifestation includes cardiovascular, ocular, gastrointestinal, renal, neurological features. Among the neurological features acute transeverse myelitis, encephalitis, stroke, psychosis Guillain Barre Syndrome are reported [6, 7]. We report a case of Chikungunya presenting with GBS with bowel bladder involvement who improved with plasma pharesis.

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## CASE REPORT

A 50 year old hypertensive man was admitted in the department on September 2017. He had history of high grade fever and multiple small and large joint pain. Four days later fever subsided but joint pain persisted. Twelve days later of fever he developed weakness of both lower limbs along with constipation and urinary retention. Urinary catheter was given to relieve retention. Within another two days he noticed weakness of both upper limbs. He had no shortness of breath, dysphagia or nasal regurgitation. On examination vital signs were stable. Neurological examination revealed muscle power of lower limbs 1/5 and 2/5 on upper limbs bilaterally. All the jerks of lower limbs were absent whereas in upper limbs were diminished. Plantar reflex absent bilaterally. Sensory functions including pain, touch, position and vibration were impaired without any definite sensory level. Fundoscopy was normal. Other system examinations were normal. On investigation (Table– 1) anti Chikungunya Ab IgM was positive, CSF study showed albuminocytological dissociation, NCS of limbs showed acute motor and sensory axonal neuropathy (AMSAN). MRI (Magnetic Resonance Imaging) of brain and dorsolumbar spine were normal. He was started with plasma pharesis. Muscle power improved from 1 to 3 in lower limbs and 2 to 4 in upper limbs, bowel and bladder function was static at the time of discharge. He was discharged with advice for regular physiotherapy at home.

**Table-1: Investigations**

| CBC | Anti Chikungunya Ab(IgM+IgG) | S.electrolytes (mmol/L) | CSF study Physical- | NCS | MRI |
|---|---|---|---|---|---|
| Hb% 14.3g/dl<br>ESR 05mm<br>1st Hour<br><br>TC-<br>WBC- 15×109/L<br>RBC- 4.65×1012/L<br>Platelet- 250×109/L<br><br>DC-<br>N84%, L 13%,<br>M02%, E01%<br>Hct- 0.43l/l | ICT for IgM positive | $Na^+$ 134<br>$K^+$ 4<br>$Cl^-$ 102<br>TCO2 15.9 | Colour watery<br>Appearance clear<br>**Microscopic examination**<br>TC- no cell found<br>**Gram and ZN stain**<br>No organism found<br>**Glucose**<br>3mmol/L<br>Protein 287mg/dl<br>**ADA** 1.6U/L | Sensory motor polyneuroradiculopathy: Acute motor sensory axonal neuropathy (AMSAN) | **Dorsolumbar spine-** Normal<br>**Brain-**<br>Normal |

ADA- Adenosine Deaminase

## DISCUSSION

Chikungunya fever has a similar pattern of dengue fever. Some atypical cases were reported. Mortality in those cases were 10%.[6,7] Neurological complications have been identified early in 1960 and still needs special attention.[4,9] From the Reunion Island 12% Chikungunya cases had neurological manifestation in the form of GBS, seizure, cranial nerve deficit, altered consciousness and other neurological features.[9] Our case presented with Chikungunya fever later on developed GBS within 12 days with atypical features like sensory and autonomic involvement in the form of bowel and bladder dysfunction. Limb weakness was ascending with bizarre sensory loss. There was no respiratory weakness. CSF (Cerebro-spinal Fluid) protein was very high whereas cell count was zero. Nerve Conduction Study (NCS) revealed Acute Motor and Sensory Axonal Neuropathy (AMSAN). On serological test for Chikungunya IgM was positive that confirms recent Chikungunya viral infection and establishes a temporal relation with the onset of GBS. For this reason we conclude that the Chikungunya virus was probably responsible for the GBS.

## CONCLUSIONS

Finding Chikungunya case along with its complication such as GBS during major outbreak in Bangladesh is an epidemiological evidence that Chikungunya can cause GBS. Moreover laboratory parameters and temporality support that GBS is the complication of Chikungunya fever in this case. This case report would create awareness among physicians that Chikungunya can cause neurological complications like GBS.

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