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A CASE OF WEST NILE ENCEPHALITIS FROM CENTRAL INDIA.

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ABSTRACT

There have been isolated cases of West Nile virus (WNV) infection from Kerala, Gujarat and Maharashtra but it has never been previously reported in Central India. We report a rare case of West Nile encephalitis from central India in a middle aged female presented with fever, headache, vomiting, pain abdomen, and followed by acute onset flaccid paralysis (AFP). To the best of our knowledge it is first report of West Nile Virus from Central India.

INTRODUCTION:

West Nile Virus (WNV), a member of the Japanese encephalitis serocomplex belonging to the genus Flavivirus and family Flaviviridae, is transmitted by Culex spp. Of mosquitoes [1–3]. Up to 20% of WNV-infected persons are symptomatic ranging from mild to severe neuro-invasive diseases. West Nile virus neuro-invasive disease (WNVND) may manifest as meningitis, encephalitis, or acute flaccid paralysis and comprises less than 1% of the total number of cases [4, 5].West Nile Virus (WNV) could be of significant morbidity and mortality; high clinical suspicion should be present to diagnose this disease. WNVND might be severe enough to require critical care. We report first laboratory confirmed case of neuro-invasive infection by West Nile virus from central India.

Case:

A 47 year old female patient admitted with history of low to moderate grade fever, abdominal pain for 25 days, Headache and vomiting for 20 days and Weakness of all extremities for 4-5 days. Previously she was hospitalized in other hospital for evaluation of fever and headache and remained conscious oriented and was able to do her activities unassisted. She was found to have mild splenomegaly and thrombocytopenia, and managed for viral fever with symptomatic treatment.

She developed progressive weakness of both upper and lower limbs and started having difficulty in getting up from sitting position and needed assistance to get up, four days before presenting to us. Progressively she became bed bound and developed respiratory distress over next 4 days. She was intubated and started on mechanical ventilator support. She also developed hypotension and required inotropic support.

At presentation she was conscious, and was following motor commands. She was able to make eye to eye contact, ocular motor functions were normal and pupillary examination was normal. She had pure motor quadriparesis (MRC grade 1/5) with preserved all deep tendon reflexes except ankle jerk which was absent. Her bilateral planter response was extensor. She had signs of meningeal irritation in form of neck rigidity and positive kerning's sign.

On the basis of above clinical presentation, she was evaluated for possibility of infectious myelo-arachnoiditis.

Her initial work up including haematological, biochemical tests were normal except for elevated transaminases. Lumbar puncture revealed increased opening cerebro spinal fluid (CSF) pressure of 29cm H_2O. Further CSF analysis showed lymphocytic pleocytosis, elevated proteins and sugar was one third of corresponding blood sugar.

Her nerve conduction studies (NCS) revealed decreased amplitudes of compound motor action potentials, normal sensory nerve action potentials, non-recordable F waves and H Reflex, suggesting symmetric axonal radiculoneuropathy.

2D-echocardiograhy showed Global Hypokinesia, LV systolic dysfunction with ejection fraction of 25-30%, along with elevated cardiac enzymes.

Her computed tomography of head were normal. Patient was managed with ventilatory support and inotropes and planned to get MRI Brain and Cervical spine but due to hemodynamic instability it could not be done.

Her ANA profile and Antiganglioside antibody profile was negative.

On the basis of initial work up infectious myeloradiculo-arachnoiditis with myocarditis of Viral etiology was likely diagnosis; she was further worked up for possibilities of Rabies, West Nile, Japenese Encephalitis viruses.

Finally CSF serology for West Nile virus (IgG, IgM) was strongly positive- IgG level by ELISA was 166.12 RU(upper normal limit >16RU) and IgM –ratio by ELISA was highly positive i.e. 8.39(upper normal limit<0.8).

She was managed with supportive care, intravenous immunoglobulins, and broad spectrum antibiotics. Despite of all efforts, patient died on seventh day of hospitalization.

DISCUSSION:

WNV has been recognized in India for many decades. WNV is a mosquito-borne RNA flavivirus and human neuropathogen, was first isolated from a febrile woman in the West Nile region of Uganda, Africa in 1937 that is spread by Culex mosquitoes. WNV is single stranded ribonucleic acid virus of the family Flaviviridae, related to JE, Murray valley and St. Louis encephalitis viruses. WNV infection is primarily maintained

in nature in a cycle between birds and mosquitoes (usually *Culex*). WNV is transmitted during blood meals by mosquitoes to man and horses (dead end hosts). Cases of man to man transmission have been documented only after solid organ transplantation or blood transfusions [6, 7].

Before 1996, WNV was known to cause high grade fever, chills, malaise, headache, backache, arthralgia, myalgias, retro-orbital pain, and a maculopapular rash, but neurological symptoms were uncommonly reported. However, since the New York City outbreak, severe neurological illness, including encephalitis and meningitis, has been reported much more frequently, together with neuromuscular manifestations.

The diagnosis of WNV infection should be considered in any patient with an unexplained acute febrile or neurological illness during the summer months, particularly if recently exposed to mosquitoes. In such cases, serum should be tested for class M immunoglobulin (IgM) antibody to WNV, which indicates a recent infection. If there are signs of CNS involvement, cerebrospinal fluid (CSF) should be analyzed and also tested for WNV IgM antibody. CSF findings typically show increased leukocytes (usually >200 cells/mm3), increased protein, and normal glucose. Almost half of WNV meningitis patients may have at least 50% neutrophils in their initial CSF specimen [8], followed by a shift to lymphocytosis. WNV infection is usually asymptomatic or results in a non-specific viral fever.

The most common neuromuscular manifestation of WNV infection is a poliomyelitis syndrome with asymmetric paralysis variably involving one (monoparesis) to all four limbs (quadriparesis), with or without brainstem involvement and respiratory failure .This syndrome of acute flaccid paralysis may occur without overt fever or meningoenephalitis. Although involvement of anterior horn cells in the spinal cord and motor neurons in the brainstem are the major sites of pathology responsible for neuromuscular signs, inflammation so may involve skeletal or cardiac muscle (myositis, myocarditis), motor axons (polyradiculitis), and peripheral nerves (Guillain–Barré syndrome, brachial plexopathy). In addition, involvement of spinal sympathetic neurons and ganglia provides an explanation for autonomic instability seen in some patients.

At present, no specific therapy has been approved for human use in WNV infection. Promising therapies include the use of interferon and interferon inducers, which have been shown to reduce mortality in mice infected by subcutaneous injection of WNV [9]. The role of corticosteroids in WNV neuroinvasive disease is controversial, with concern that immunosuppressive effects may worsen outcome. However, high-dose steroids have been used to successfully treat a patient with WNV-associated acute flaccid paralysis [10]. There also has been great interest in passive immunization with intravenous immune globulin (IVIG) for the treatment of patients with acute WNV infection.

Present case is the West Nile virus neuroinvasive infection first laboratory confirmed case from Central India. Abdominal pain as an early symptom was an unusual presentation in our case.

One should keep a high index of suspicion in patients presenting with encephalitis and meningitis with flaccid weakness.

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