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Mimicry of Two Disease Guillain-Barre Syndrome and *Japanese encephalitis*: A Case Report

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

Guillian-Barre syndrome (GBS) is an acute immune-mediated inflammatory demyelinating polyneuropathy characterized by symmetrical limb weakness and areflexia preceded by an infection while *Japanese encephalitis* (JE) virus is a neurotropic RNA virus belonging to Flaviviridae family. GBS can have different clinical manifestations which overlaps JE. GBS secondary to JE has been reported earlier though in the incidence is low.Involvement of the peripheral nerve, areflexic paralysis, electrophysiological nerve examination and JE IgM in CSF were consistent with a diagnosis of GBS with JE. Prompt detection and diagnosis can reduce morbidity and mortality.

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1. INTRODUCTION

Guillain-Barre syndrome (GBS) is a hetrogenous autoimmune disease commonly seen in children which manifests as a acute inflammatory polyradiculoneuropathy with generalized weakness, areflexia with or without sensory loss that is often antecedent by either bacterial infections [1,2] like *Campylobacter jejuni*, *Mycoplasma pneumoniae*, and viral infections like Cytomegalovirus, Epstein–Barr virus, Arboviruses Dengue & West Nile [3]. *Japanese encephalitis*(JE) is a RNA neurotropic common mosquito borne flavivirus encephalitis in Asian subcontinent [4] and not commonly known to be associated with GBS. JE typically presents with fever followed by malaise, altered sensorium, convulsions, neck stiffness, mask-like facies, tremor, cogwheel rigidity, and choreoathetoid movements are clinical features. Apart from them acute flaccid paralysis like illness, bulbar palsy & cranial nerve involvement has been reported [5]. Herein we present a case GBS with JE in central India which is rare in this region.

2. CASE PRESENTRATION

An 8-year old male child admitted in pediatric intensive care unit with complaints of fever since seven days along with generalized loss of power and tone with numbness in all four limbs. He also had altered sensoriumsince last two days along with difficulty in breathing and one episode of seizure at the time of admission. On detailed history, the patient was known to have had a high grade fever with headache and myalgia one week back. On examination the patient had quadriparesis, which was more distally, with generalized hyporeflexia. Sensory disturbances like tingling, numbness, loss of pain and temperature sensation along with dysphagia, dysautonomia and respiratory distress was present. There was neck rigidity, weak gag reflex and absence of Babinski signon examination.. On admission, his GCS was 7/15 and the patient was in respiratory distress with heart rate 180 beats/min, respiratory rate 58 cycles/ min, oxygen saturation 80% on 5L of O2 , blood pressure 80/60 mmHg. The patient was intubated and was given ventilator care. Patient was sedated and started on intravenous broad-spectrum antibiotis, antivirals, inotropes, and other supportive medication. He was investigated, and showed a hemoglobin level of 10.8 g/dl and total leukocyte count of 11,900/cu mm with 65% neutrophils and 35% lymphocytes, erythrocyte sedimentation rate of 18 mm/h and platelet count of 254,000/µl. Urine microscopy was normal. MRI was done which was suggestive of Electroencephalography was normal. Magnetic resonance imaging of the brain revealed meningoencephalitis. Examination of the cerebrospinal fluid was clear with WBC count of 12 cell/µL with a lymphocytic predominance (85%). CSF protein was 72 mg/dL, glucose 76mg/dL, choride was low 104mEq/Land concomitant serum glucose level of the patient was 98mg/dL. CSF culture showed no growth. Nerve electrophysiology suggested an predominantly axonal motor polyradiculoneuropathy. Enzyme linked immunosorbent assay for JEV IgM was positive. He was diagnosed with GBS associated with a recent JEV infection. Intravenous immunoglobulin along with methylprednisolone was administered. The patient's condition improved and hemodynamically stable after 10 days and he was discharged later with complete neurological recovery was noted on further follow up.

3. DISCUSSION

Infectious disease remains the major cause of morbidity and mortality in humans. JE is a common encephalitis in Asia being particularly endemic in the eastern belt of the Indian subcontinent. GBS is an acute polyradiculoneuropathy of variable etiology and often precededby an unidentifiable pathological infection [6,7]. Pathogenesis of GBS is still not clear. It is understood to be a variety of acute neuropathies with multiple immune mediated pathogenic mechanisms.

The JEV is a mosquito borne flavivirus, and may manifest as a meningoencephalitis with typical fever, headache, neck rigidity and mental state. Unusual clinical features of JE seen in literature are respiratory failure, oromandibular dystonia, hemiplegia with dysarthria and acute flaccid paralysis [8]. These signs and symptoms may overlap those of GBS. GBS secondary to JE infection has been reportedin India and China [9]. JE infection can be associated with GBS in JE endemic areas but Madhya Pradesh (Bhopal) is not an endemic area.

JEV myelitis results in anterior horn cell damage [2]. The pathological changes in GBS are demyelination of nerve fibres and axonal degeneration. Such changes slow down the nerve conduction velocity, decreased compound muscle action potential amplitude and abnormal F wave on electromyography. Another immnopathologic mechanisms is endoneural inflammation in spinal nerves roots, nerve segments or around potential nerve entrapment sites. Cross reacting determinants with host tissue share viral proteins leading to detection of self proteins by antibodies as viral infection. Antibodies to host/self proteins are often formed post viral infections, this may be due to molecular mimicry between myelin basic protein of humans and viral proteins. Axon and myelin sheath have the common antigen and are thus affected. Such phenomenon results in autoimmune mediated tissue damage [10,11].

In our case though the patient is from rural area of Madhya Pradesh (Bhopal) which is not an endemic region and thus had low risk and not vaccinated for JE. Recently we had few case of JE in Bhopal. Involvement of the peripheral nerve, areflexic paralysis, electrophysiological nerve examination and JE IgM in CSF were consistent with a diagnosis of GBS with JE. The incidence of GBS and JE is low for both but possibility of both diseases occurring completely independently is rare.

4. CONCLUSION

The better prognosis of patients of GBS on treatment with immunoglobulins is more promising as compared to JEV patients with CNS manifestations, who will require supportive and symptom based care. Hence, the need to differentiate between the two. A suspicion of JE should be in consideration with acute flaccid paralysis. JE is spreading in affected area too so reporting of such case to health authorities for prevention by immunization and vector control respectively.

CONSENT

As per international standard or university standard, parental(s) written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

The study was approved by the Institutional Ethics Committee.(EC/20/04).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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