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RESEARCH ARTICLE

CASE REPORTS OF JAPANESE ENCEPHALITIS: AN UNDERDIAGNOSED ENTITY IN AN ENDEMIC REGION OF UTTAR PRADESH, INDIA

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Abstract

Emerging viruses causing Acute Encephalitis Syndrome (AES) can be more damaging due to irreversible brain damage, irrespective of the identical medical characteristics created by all agents.

We report two cases of acute encephalitis syndrome caused by Japanese encephalitis virus from a usually under-reported geographic region of India. Both patients were managed conservatively with favourable outcome in one of them. There should be considerable effort to identify the particular causative agent that triggers AES, bearing in mind the various clinical manifestations of Japanese encephalitis virus. Although there is no significant impact on management, it is possible to prevent transmission to healthy contacts and the community through vector control and vaccination.

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Introduction:-

Japanese encephalitis (JE) virus, a mosquito-borne flavivirus, is the leading cause of vaccine preventable encephalitis in Asia. It mainly affects children < 15 years of age and is mostly asymptomatic.^[1] It usually resolves within weeks if symptomatic, and support for ventilators is not generally needed. Hospitalization is primarily due to neurological symptoms and may or may not occur due to respiratory involvement. An estimated 67,900 cases of JE are reported annually, with approximately 13,600–20,400 deaths, while in 24 WHO member countries, 2 billion people are at risk. India and China are experiencing 95% of JE's reported disease burden. According to the data, nearly two-thirds of the at-risk population for JE is in China and India only.^[2] An outbreak in India (2005) resulted in 1700 deaths reported mostly among children, exacerbating the ongoing disease burden in developing countries.^[3] Japanese B encephalitis virus (JEV) is an emerging pathogen in North India and has entrenched itself firmly in the eastern parts of Uttar Pradesh.

Case Reports:-

Case 1:

A 11 months old boy presented with fever, altered sensorium and un-coordination of movement in form of tonic clonic seizures of all four limbs for 2 days for which he was referred to our tertiary care centre. He did not have any recent travel history. On admission, he was febrile with a temperature of 103.9°F, pulse of 170/min blood pressure of 105/58.

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The patient was started on empirical antimicrobials and routine blood cultures were sent to bacteriology lab which was negative. The patient did not showed any sign of improvement on empirical therapy after 4 days of admission. On 4th day of admission the patient had 2 episodes of tonic clonic seizures. He developed aphasia and altered sensorium, unresponsive to oral commands, in view of which he was started on inj. acyclovir. His GCS was E4V2M5 11/15, aphasia was present, bilateral pupils were reacting to light. Magnetic resonance imaging (MRI) showed an acute small infarct in the left thalamus. Cerebrospinal fluid (CSF) was relatively clear in appearance and analysis revealed an elevated WBC count (78 cells/ μ L) with a lymphocytic predominance (80%). CSF protein was elevated (107 mg/dL), glucose was normal (78 mg/dL), chloride was normal (125 mEq/L); the concomitant serum glucose level of the patient was 123 mg/dL. CSF Gram stain showed few pus cells and no organisms; CSF for bacterial culture showed no growth. Herpes simplex virus 1 and 2 by ELISA on CSF and serum samples were negative. Dengue NS1 antigen, Dengue IgM antibodies by ELISA were negative by ELISA. Serum and CSF samples for IgM antibodies to Japanese encephalitis virus were sent to VRDL Lab and both were positive. The patient was treated conservatively with I.V. fluids, anticonvulsant drugs, diuretic therapy and supportive care. The patient was symptomatically better and hemodynamically stable after two weeks of management and he was discharged with cognitive impairment.

Case 2:

A 5 year old girl presented to our centre with history of fever, altered sensorium and abnormal movement for 3 days. There was history of travelling from Bihar district Gaya to UP district Aligarh 15 days back from the onset of fever. On examination the patient was febrile with temperature of 102.5⁰ F, pulse of 155/min blood pressure of 92/76. On pulse oximetry 98% saturation. On neurological examination her Glassgow Coma Scale (GCS) was E2V2M4 (8) with brisk deep tendon reflex, planter was extensor and deviation of mouth on left side. Bilateral Pupils were normal in size and reacting to light. There were no sign of meningism. The rest of systemic examinations were within normal limit. Treacheal intubation was done. The patient was started on empirical antibiotics. MRI (Figure 1) showed flair hyperintensities in bilateral thalami and left parietal lobe with diffusion restriction involving bilateral thalami with few areas of restriction in bilateral basifrontal lobe right temporal lobe. Serum and CSF showed fourfold increase in IgM antibodies against JE. Antibodies against herpes simplex were negative. CSF glucose (92mg/dl) was elevated, protein (34mg/dl) was normal. CSF gram stain showed few pus cells and no organism, CSF for bacterial culture showed no growth. The patient was treated conservatively with I.V fluid, steroid, diuretic therapy and supportive care. On 1th day of admission, patient developed exposure keratopathy and subsequently secondary infection to cornea. Organism isolated from corneal ulcer was multidrug resistant *Pseudomonas* species, sensitive to colistin only. the patient was on Ventilator support for 10 days but with no improvement. The patient left against medical advice and no further follow up could be done.

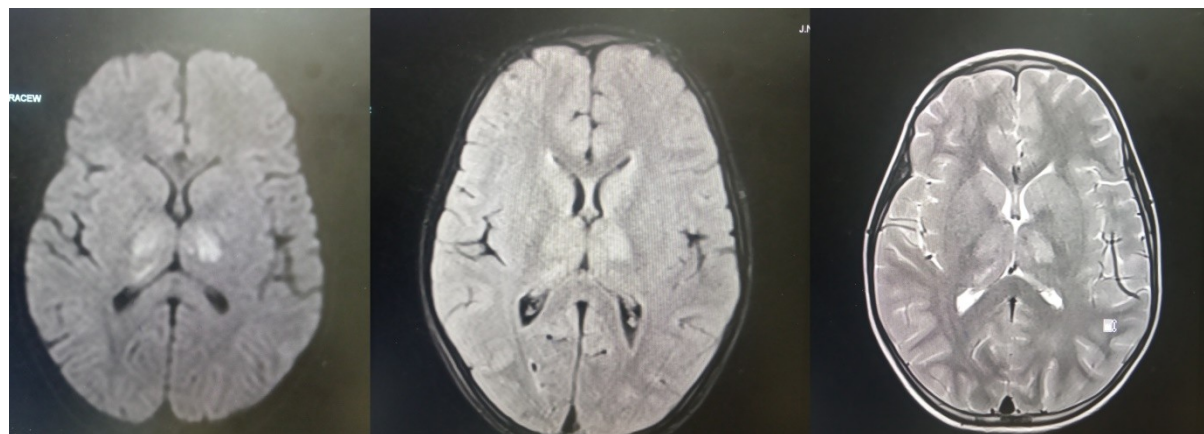


Figure 1:- MRI of Case 2 revealed T2 FLAIR hyperintensities in bilateral thalami and diffusion restriction involving bilateral thalami.

Discussion:-

JE is a viral zoonotic disease spread by a vector (mosquito), caused by an arbovirus (Flavivirus) of the Flaviviridae family. Five genotypes of the virus have been identified, which mainly affect the central nervous system.^[4]

In endemic regions, the disease is usually confined to the paediatric age group, especially below 15 years because subclinical infections provide acquired immunity till adulthood. Both of our reported cases are from paediatric age group but JE is a disease of all age groups and can affect adults in particular when the disease is introduced into non-endemic areas or regions lacking adequate coverage of JE virus (JEV) immunization in children. Most of the cases of JE outbreaks in temperate regions are reported in late summers or in the rainy season probably due to greater breeding of the Culex mosquito vector.^[5] Living in proximity to rice fields is also a risk factor for the transmission of JE as Culicine mosquitoes breed in abundance in paddy fields. Studies from different Indian states^[6,7,8] also showed higher JE positivity during the rainy season because the paddy fields covered with stagnant water serve as a healthy vector breeding environment. The present cases presented in the months of October, which again is a post rainy season in our area favouring the breeding of the involved vector. In our study, fever and change in mental status was the common presenting feature in both the cases. Others similar studies^[9] from India have also reported fever and change in mental status as the most common presenting symptoms of JE.

In a study from this part of Uttar Pradesh,^[10] JEV was not found in 87 cases of acute encephalitis Syndrome. Japanese encephalitis, although a big public health problem in eastern Uttar Pradesh, it has not yet made any inroads into our western Uttar Pradesh area till the appearance of these cases. Hence, we should be alarmed and extremely vigilant after the appearance of these two cases as majority of cases are asymptomatic in JE and these two cases may represent only the tip of iceberg. Vector control should be practiced stringently to prevent the onward march of JEV.

The criteria for laboratory evidence of Japanese encephalitis are positive IgM antibodies in a single sample of serum or CSF, according to the World Health Organization.^[11] Our both patients had positive IgM antibodies in serum and CSF thereby confirming the clinical suspicion of Japanese encephalitis. Few authors have described the usefulness of imaging studies to substantiate the diagnosis of JE. In JE patients, MRI imaging has showed lesions involving bilateral thalamus, pons, midbrain, basal ganglia, cerebral cortex, and edema of white matter.^[12] Our patients had lesions in thalami and parietal lobes. Both of our patients presented with seizures as it is a common manifestation in clinically symptomatic cases of JE.^[13]

Mortality or adverse complications, including permanent brain damage caused by Japanese encephalitis virus, are inevitable due to the neurotropical nature of JE virus and the lack of effective antivirals for JE therapy.^[12] Hence, despite the favourable outcome in one of our patient, he developed cognitive impairment.

It is difficult to diagnose acute encephalitis syndrome due to Japanese encephalitis virus unless there is experience of the disease's various presenting symptoms. JE concern should be raised when any patient in an endemic area such as ours present with AES. A combined use of all these will help to manage and contain Japanese encephalitis effectively.

Conclusion:-

JE should be considered in the differential diagnosis for any patient with an acute neurologic infection who recently has been in a JE-endemic area. In order to detect and contain possible outbreaks, prompt identification and reporting of cases is important. We need to strengthen the JE surveillance system and the vaccination program.

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