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Original Research Paper

Paediatrics

DENGUE AND CHIKUNGUNYA WITH GBS:A RARE ASSOCIATION.

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ABSTRACT Guillain-Barre Syndrome (GBS) is an autoimmune inflammatory polyneuropathy, which usually develops following infectious diseases. It is a rare complication of dengue fever, in particular, GBS following combined Dengue and Chikungunya infection is extremely rare. We hereby report a patient with combined Dengue and Chikungunya fever in association of GBS.The main concern in this case was the sudden development of paresis, which was responsive to immunoglobulin therapy .

KEYWORDS : Arboviral disease,Dengue fever, Chickungunya fever,Guillain-Barre syndrome.

INTRODUCTION:

During epidemics, Combined Dengue and Chikungunya infections can occur. Both of the viruses are transmitted by same *Aedes* mosquito, and both are acute febrile illnesses characterised by fever, myalgia, lethargy, headache and rash[2]

Incidence: The incidence of dengue infection associated with neurological manifestations is 1 to 5%.

Etiopathogenesis:

Dengue and chickengunya are mosquito-borne infections caused by an arbovirus and transmitted by Aedes Mosquitoes.

Four serotypes of dengue viruses (DEN1, DEN2, DEN3, DEN4). At present, DEN1 and DEN2 serotypes are widespread in India.

Chikungunya virus is an RNA alphavirus belonging to the *Togaviridae* family (group A arbovirus).

Guillain–Barré syndrome (GBS) is an immune-mediated disorder characterized by acute areflexia, paralysis with albuminocytologic dissociation in cerebrospinal fluid[3].

Dengue fever is an uncommon antecedent of GBS and only a few cases have been reported[5,7]

We are reporting a case of Guillian barre syndrome which occurred with dengue and chickengunya infection, as a neurological complication which is not commonly seen in routine practice. There are only few case reports available documenting GBS after dengue and chickengunya infections[5]

CASE SCENARIO

Presentation:

9 year male child presented with multiple joint pain for 2 to 3 days and fever for 2 days with no any complaints. On examination, findings in large joints s/o inflammatory changes with no any abnormal findings found.

Meanwhile, Routine investigations were sent which was found normal.chickengunia and dengue serology were sent which was positive and showing high titre of IgM.

Progress:

On 2nd day of admission, patient developed sudden onset of hypotonia (power in both lower limbs was 2/5) of lower limb with areflexia and respiratory difficulty. There was no complaints of sensory abnormality ,no bladder and bowel dysfunction.

Urgent cerebrospinal fluid study was sent and nerve conduction study done.

Within 10 hours of hypotonia ,patient developed laboured breathing and put on ventilator care.

Meanwhile, CSF study and NCV results were come.

CSF examination showed protein= 110 mg/dl; sugar 63= mg/dl; TLC= 5 cells/cumm; all lymphocytes which was suggestive of albuminocytological dissociation.

Nerve conduction study was done that showed reduced conduction velocity, increased latency, conduction blocks and prolonged F waves. NCS report was suggestive of demyelinating neuropathy.

So the diagnosis of GBS was entertained with antecedent dengue and chickengunya infection as a possible most likely aetiology. Intravenous immunoglobulin (IVIg) was given for 5 days. Patient responded well with the treatment and power of the limbs was improved up to 4/5 on the 16th day of admission.

Timeline:

First 2 days:fever and arthalgia
3rd day: lower limb hypotonia and areflexia
4th day:respiratory paralysis
6th day: wean off from ventilator care and shifted to oxygen therapy
8th day: shifted to room air, power in all 4 limbs became 3/5
9th day: started rt feeding
11th day: gag reflex improve,can stand with support
13th day: shifted to oral feeding,can walk with support
14th day:discharged on oral feeds

Follow up:

After 6 week patient improved in form of no neurological deficits and going to school.

DISCUSSION:

DENGUE[3]

Dengue infection is a leading cause of illness and death in tropical and subtropical regions of the world. Over 40% of the world's population are currently at risk from dengue. The clinical picture

resulting from dengue infection can range from relatively minor to catastrophic hemorrhagic fever. Because dengue infection can be asymptomatic, the actual number of cases of dengue infection has been underestimated. Various neurological manifestations have been reported with dengue infection. The incidence of infection associated with neurological manifestations is 1 to 5%. These are encephalitis, encephalopathy, aseptic meningitis, mononeuropathies, myelitis Guillain-Barre syndrome (GBS) and intracranial haemorrhage.

CHIKUNGUNYA[3]

Chikungunya virus is an RNA alphavirus (group A arbovirus) in the family *Togaviridae*.Chikungunya infection, after an incubation period of 2–10 days, has the main clinical manifestations of fever, polyarthralgia, and rash. Treatment consists of rest and medication for pain. Outcome is marked by incapacitating arthralgia, which can persist for several weeks or months. Complications are rare and consist of mild hemorrhage, myocarditis, and hepatitis[8]. Neurologic manifestations are less well known[9]. Infection is confirmed by the identification of genomic products in acute-phase blood specimens, (reverse transcription–PCR [RT-PCR]) or, more recently, by serum immunoglobulin (Ig) M or a 4-fold increase in other antibodies.

GBS[3]:

GBS is an acute, frequently severe and fulminant polyradiculoneuropathy that is autoimmune in nature. GBS manifests as a rapidly evolving areflexic motor paralysis with or without sensory disturbance. Weakness typically evolves over hours to a few days and is frequently accompanied by tingling dysesthesias in the extremities.

ASSOCIATIONS:

Both of the viruses are transmitted by the Aedes mosquitoes (mainly *Aedesaegypti* and less commonly *Aedesalbopictus*)[5] Hence, their infections are related epidemiologically. Both the viruses can also co-circulate in endemic areas, so that combined Chikungunya and Dengue virus infections can occur during an epidemic or in travellers to endemic regions. Neurological complications of Dengue virus infection occur in 0.5–0.7% of symptomatic cases[6.] Moreover, several immune-mediated neurological syndromes can occur following Dengue infection.

However, dengue infection may have been underestimated as a causative agent of GBS.5 It is suggested that the clinical manifestations of GBS are the result of cell-mediated immunological responses to non-self-antigens (infectious agents, vaccines) that misdirect to host nerve tissue through a resemblance of epitope (molecular mimicry) mechanism.3,6 The activated T cells cross the vascular endothelium (bloodbrain barrier) and recognize an antigen in the endoneural compartment. T cells produce cytokines and chemokines which open the blood-brain barrier allowing antibodies to enter and Schwann cells to attack.6

Dengue virus would initiate this immunological event, leading to the disease. Myelin or axons could be the target of this immune response. Diagnosis of GBS is mainly based on the clinical and lab findings. Clinically diagnosis is made by recognising the pattern of rapidly evolving ascending paralysis with are flexia, absence of fever or other systemic symptoms, and characteristic antecedent events as in our case. Investigations helpful in making diagnosis are CSF findings and nerve conduction study.

The CSF findings are distinctive, consisting of an elevated CSF protein level (100-1000 mg/dl) without accompanying pleocytosis i.e. albuminocytological dissociation.3 NCS suggests pattern of demyelinating neuropathy i.e. slowing of conduction velocity, conduction block, prolonged distal latency, prolonged F wave latencies and reduced amplitude of compound muscle action potentials (CMAPs). Treatment should be initiated as soon after diagnosis as possible and it is same for GBS due to any aetiology. Either high dose intravenous immunoglobulin (IVIg 2 gm/kg body weight divided in 5 daily doses) or plasmapheresis can be initiated, as they are equally effective for typical GBS. Glucocorticoids have not been found to be effective in GBS. We have given IVIg to our patient and he responded well to the therapy.

CONCLUSION

Dengue fever is very common in endemic areas but GBS is a rare neurological complication of it which is generally underestimated. It should always be considered if a patient of dengue fever develops progressive weakness of the limbs and treatment should be initiated as early after diagnosis as possible.

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