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.

# Dengue Fever Presenting With Acute Cerebellitis : A Case Report

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## Abstract

The incidence of dengue fever is on the rise in tropical countries. It has become a endemic in many places in India. The mortality is gradually increasing with varied clinical manifestations. Classical dengue fever presents as a febrile illness with an uneventful recovery. A proportion of patients develop potentially life-threatening dengue haemorrhagic fever, which is associated with plasma leakage and shock [2]. Acute liver failure, acute kidney injury, and multi-organ failure are known complications [2]. However, many unusual manifestations have been reported with dengue, and there are many reports of neurological manifestations. With the increasing numbers of rare manifestations of dengue are occasionally encountered. With early diagnosis and treatment we can prevent the mortality. We report a patient with acute febrile illness who presented with bilateral cerebellar signs as the presenting feature of dengue with neurological manifestation of cerebellitis.

**Keywords**: dengue fever ,cerebellar signs, neurological complications

## Introduction

Dengue fever is the mosquito-transmitted, arboviral infection mainly found in tropical and subtropical countries. Around 2.5 billion population worldwide is at risk of dengue infection, and its endemic zone comprises more than 100 countries of the world. Dengue infection is caused by any of the four antigenically related distinct serotypes. The clinical presentation of dengue infection has a wide spectra, ranging from mild clinical febrile illness to severe life-threatening situations like dengue hemorrhagic fever and dengue shock syndrome. In recent years, the virological characteristics of dengue viruses have been changing, resulting in widespread neurological complications. We report a patient with acute febrile illness who presented with bilateral cerebellar signs as the presenting feature of dengue with neurological manifestation of cerebellitis.

## Case report

A 24 year old male patient came with history of high grade intermittent fever with chills &rigors for past one week ,with associated myalgia.Patient has history of headache since one week withpresenting illness of unsteadinesswhile standing and walking since three days. Patienthad difficulty in speaking since 3 days. No siginificant illness and comorbid disease in past history. With no personal history of alcohol intake and smoking previously.

Summarising the history patient with an acute febrile illness associated with unsteadiness of gait and difficulty to speak was admitted in zero delay ward of our hospital.On examination His pulse rate was 82 beats per minute; blood pressure was 120/80 with no postural hypotension; respiratory rate was 14/minute. Detailed neurological Clinical examination with a Glasgow coma scale (GCS) score of 15/15, revealed a dysarthriaand marked horizontal nystagmus of both eyes , with bilateral dysmetria, dysdiadokokinesia and incordination more prominent onthe right with unsteadiness in gait swaying to right side. His gait was wide-based and ataxic with a tendency to fall to the right side more than to the left. Other neurological examinations like spinomotor system with power ,toneand sensory system were normal. Patient was given general line of management. However, no evidence of plasma leakage or shock occurred. After the platelet count has improved in one week and clinically afebrile periods patient was given Intravenous methylprednisolone 1 gram per day for 5 days and tapered with oral steroids .

Investigations at the time of admission were done with CBC showed intial leucocyte count was 5400 ,platelet counts were decreased with initially day one around 11000 and gradually improved to 57000 after third day ,then 196000 at the end of one week of admission. ESR was 45 mm / hour. Peripheral smear

was seen with thromobocytopenia with no hemoparasites. RFT and LFT was within normal limits.Thyroid function test was normal. Serum vitamin B12 Assay was within normal limits. Malaria card test was negative. Leptospirosis IgM antibody was negative. Scrub typhus IgM antibody was negative .VCTC was non reactive. VDRL test was negative. Dengue nonstructural protein antigen 1 test and IgM antibodyelisatesting both became positive indicating acute dengue viral infection. CSF analysis for Viral PCR was positive for dengue virus and negative for other virus. Ultra sonogram of abdomen was normal study. Chest x-ray was normal study limits. He recovered from the febrile episode within 8 days since the onset of fever but cerebellar symptoms outlasted the fever by one week. The magnetic resonance imaging of brain was taken on third day of admission before starting steroids showed no focal lesion in cerebral hemispheres ,ventricles normal, brain stem normal,cerebellar hemispheres with no significant abnormalities, Sellar and parasellar regions are normal, Focal area of restriction of diffusion seen in splenium of corpus collsum.MRA and MRV is normal. Clinically cerebellar signs resolved gradually with treatmentby day 15 of the illness.

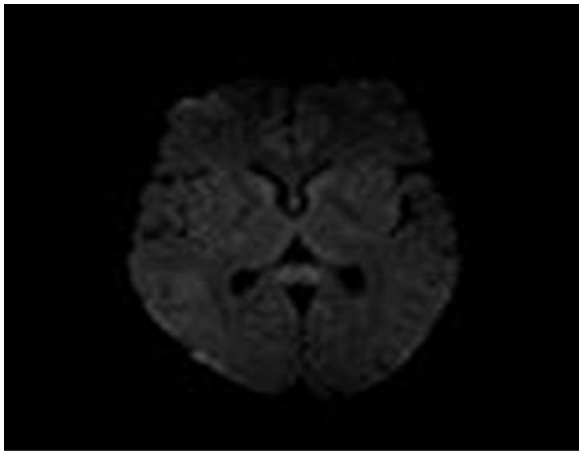

Fig-1

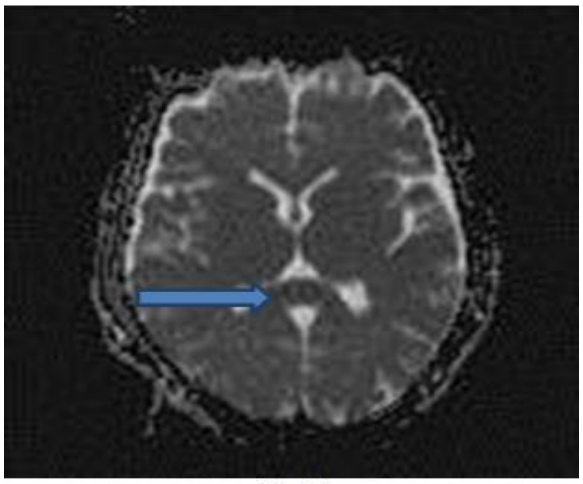

Fig -2

MRI showing no focal lesion in cerebral hemispheres, ventricles normal, brain stem normal, cerebellar hemispheres with no significant abnormalities, Sellar and parasellar regions are normal, Focal area of restriction of diffusion seen in splenium of corpus collsum. MRA and MRV is normal

## Discussion

Neurological complications occur in 0.5-6.0% of patients with dengue infection [4]. The neurological complication in dengue infection has been hypothesized through three pathogenic mechanisms: (1) concerned with neurotropism leading to encephalitis, meningitis, myositis and myelitis, (2) systemic complications resulting in encephalopathy, stroke and hypokalemic paralysis and (3) postinfectious immune-mediated acute disseminated encephalomyelitis, Guillain Barresyndrome and optic neuritis. Immune mediated mechanisms as well as direct tropic effects of the virus have been postulated to cause these neurological manifestations, and dengue antigen has been demonstrated in the brain in some patients with dengue encephalitis [5]. Dengue encephalopathy could be caused by cerebral edema, cerebral hemorrhage, hyponatremia, hypoxia, renal and hepatic insult. Immune-mediated disorders including Guillainbarre syndrome, acute disseminated encephalomyelitis and neuralgic amyotrophy, which is a new observation, are explained on the basis of autoimmunity, molecular mimicry or nonspecific activation of autoreactive T cell clones leading to destruction of the myelin sheath self-antigens.[11]. On the basis of the possible pathogenic mechanism: (1) neurotropic complications – encephalitis, myelitis, myosistis, (2) systemic complications – hypokalemic paralysis and (3) postinfectious immune mediated – acute disseminated encephalomyelitis, Guillain Barre syndrome and opsoclonus myoclonus syndrome.

Verma *et al*. recently reported a case of epilepsiapartialis continua in a young woman suffering from dengue encephalitis. She presented with encephalitic syndrome of headache, vomiting, altered sensorium and intractable seizures [13]. Opsoclonus can be defined as saccadic stability disorder consisting of involuntary arrhythmic multidirectional high-amplitude conjugate saccades. Opsoclonus is often accompanied by diffuse or local myoclonus and truncal ataxia along with other cerebellar signs. Opsoclonus myoclonus syndrome is a rare neurological condition of unknown cause that appears to be the result of an autoimmune process involving the nervous system.[13] It is one of the few paraneoplastic syndromes that occur in both children and adults. In children, it is often associated with neuroblastoma and in adults, it is presumed to be of viral infection, e.g. Epstein barr, Coxsackie B or enterovirus, etc.[14] All patients received primarily a symptomatic treatment with special care for

maintenance of fluid and electrolyte imbalance. Intravenous methylprednisolone and oral prednisolone were given to patients of myelitis, ADEM, brachial neuritis and myositis. Potassium supplementation in the standard regimen was given to patients of hypokalemic paralysis, IV Ig in standard dose of 0.4 gm/kg/day for 5 days for Guillainbarre syndrome patients and clonazepam was prescribed to patients having opsoclonus myoclonus. Antiepileptics and other supportive treatment were given to patients having seizures and encephalopathy.

Post-infectious cerebellar syndrome has been describedfollowing several viral infections, but the association with dengue has been reported in only four instances[6,7]. In these reports, the onset of cerebellar symptomsvaried between two days to two weeks after the onset offever [6,8]. Our patient is the second reported case of denguefever presenting with cerebellitis as the first manifestationof disease. Previously reported case study has similar presentation in Sri lanka.[10]. Interestingly, the cerebellar syndrome outlastedthe fever by almost two weeks. This report also highlightsthe self-limiting nature of this rare but severe neurological complication of dengue infection. In our case based on hypothesis of immune mediated reaction to infections ,steroids were given after platelet counts had improved in one week of admission and clinically afebrile with no evidence of shock and plasma leakage. MRI evidence of acute cerebellitis therefore may be transient and hence not seen in every patient depending on the timing of imaging.

The evaluation and investigations for dengue virus infection may involve detection of the virus, viral nucleic acid, antigens or antibodies (serology). In the initial 4–5 days, virus detection by culture, viral nucleic acid or antigen detection (NS 1) can be used to confirm dengue infection. However, after early acute phase, serology is preferred. Viral RNA nucleic acid detection by polymerase chain reaction assays are quite specific (100%) and sensitive (70%) in early acute phase, but they are more costly, more technical and not freely available everywhere. They may not be so useful in the later part of the illness when various neurological complications arise. As IgM antibody suggest recent infection, the detection of “dengue-specific” IgM antibody (serology) detection by MAC-ELISA (IgM antibody-capture enzyme-linked immunosorbent assay) is the preferred method for diagnosis after acute phase (after 5 days of fever onset),[12]. Dengue fever can result in various neurological manifestations. Dengue infection should be considered and properly investigated in patients presenting with various neurological disorders without obvious etiology, especially if preceded by a febrile illness compatible with dengue fever.

## Conclusions

Neurological complications of dengue infection are widespread and may involve almost all parts of the nervous system through various pathogenetic mechanisms. Along with various previously reported manifestations, in this study, we had described new neurological complications of dengue fever. Cerebellar syndrome in association with dengue fever has been reported in only few instances .This case report is intended to highlight the occurrence of acute cerebellitis as a presenting syndrome of the expanding list of unusual neurological manifestations of dengue infection. Furthermore, clinicians should familiarise with the wide spectrum presentation of post dengue neurological manifestations to prevent misdiagnosis. This case report has highlighted the importance of early recognition of the neurological complications of dengue. Thus, a close monitoring of clinical progress can be done to ensure appropriate management being given in timely manner. Furthermore, this early recognition could avoid unnecessary invasive investigation. However, some of the neurological complications are serious and life threatening, which require timely specific therapeutic interventions.

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