International Journal of Science and Research (IJSR)
ISSN (Online): 2319-7064
Impact Factor (2012): 3.358

Dengue Encephalitis with Acute Intracerebral Infarction and Facial Palsy; A Rare Presentation

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Abstract: ***Dengue fever is known to manifest a variety of atypical complications which may progress to self-limiting to life-threatening conditions. Recently it is seen that clinical profile of dengue is expending and atypical systemic manifestations are being reported more frequently. Encephalitis with intracerebral infarction is one of the atypical neurological manifestation which is reported very rarely. Hereby we reported a 26-year old female of dengue fever who was diagnosed Encephalitis with left sided hemiparesis with left side facial palsy cause right sided intracerebral infarction secondary to dengue fever. She recovered partially on conservative treatment.***

Keywords: Dengue fever, Intracerebral infarction, Facial palsy, Encephalitis, *Aedes aegypti*

1. Introduction

Dengue is a arboviral infection found in tropical and subtropical countries. It is transmitted by infected female *aedes aegypti* mosquito bite. The spectrum of neurological manifestations due to dengue is not well clear. Dengue encephalitis with hemiparesis with facial palsy is an uncommon manifestation of central nervous system in dengue fever and is not yet widely recognised. Awareness of this condition will help physician to make early diagnosis and effective management.

2. Case Report

A 26 years old nonpregnant housewife was referred to our emergency department in unconscious state. According to her relatives, she had complaints of low to high grade fever with rashes over limbs for 12 days and one episode of generalised seizure followed by altered sensorium of 2 days. On enquiry her relatives told that initially patient was admitted in a private hospital. Her condition was deteriorating so patient was referred to our king George medical university. At the time of admission patient was in unconscious state with a Glasgow coma score (GCS) of 8 (E2M4V2) with decreased left sided body movements. There was no sign of meningeal irritation and her cranial nerve examination revealed left sided facial palsy. Patient had no pallor, icterus or dehydration. She was febrile (101°F). Her pulse rate- 88/ min normovolumic and regular, B.P- 110/80 mmHg and respiratory rate was-20/min. All deep tendon reflexes were normal, plantar reflex was extensor of left side and flexor of right side. Cardiovascular, respiratory and abdominal examinations were within normal limit. She had two previous normal vaginal deliveries without any history of abortions. She had no history of hypertension, diabetes mellitus, cardiovascular disease, dehydration, seizure, drug abuse or oral contraception pills, hypercoagulable disorders, vasculitis, or any similar episode of hemiparesis in past. Her laboratory investigations are given in table [1]. Routine and microscopic urine examination showed trace proteinuria. Serum Protein C and protein S level, serum homocystein level and ANA titre were within normal limit. Enzyme-linked immunosorbent assay for human immunodeficiency virus, Australia antigen for Hepatitis B and antibody against Hepatitis C virus were negative. Serology of enteric fever and smear examination for malaria parasite were negative. Dengue NS1 Antigen was positive in blood while dengue IgM antibody was negative. After one week, serology (IgM antibody) for dengue infection was repeated which was positive. Cerebrospinal fluid (CSF) examination was normal and CSF dengue IgM antibody was negative. Electrocardiography, 2D Echocardiography, abdominal ultrasonography and chest X-ray were within normal limit. MRI brain showed acute infarct in right corona radiata and central semiovale, right fronto-temporo-parieto-occipital region and left occipital area. On the basis of clinical features and investigations, final diagnosis of left sided hemiparesis with upper motor neuron type facial palsy cause right sided intracerebral infarction secondary to dengue fever was made.

3. Treatment and Follow Up

Patient was managed conservatively with adequate intravenous fluids, intravenous antibiotics and antiepileptic drug. Limbs physiotherapy with general care was done daily during hospitalisation. The patient showed gradual improvement and got discharged on 16th day of admission in fully conscious state with power 4/5 in left upper and 3/5 in left lower limb. Limbs physiotherapy with general supportive care was advised at the time of discharge. After four months of follow up, she recovered so much that she was able to do her daily routine work.

4. Discussion

According to WHO guideline revised in 2011, dengue was divided into dengue fever (DF), dengue hemorrhagic fever (DHF) without shock or with shock (DSS) and expanded dengue syndrome. Expanded dengue syndrome is a new entity to the classification system to incorporate variety of atypical presentations of dengue infection affecting various organ systems including hepatic, neurological, gastrointestinal, pulmonary and renal.[1] The relationship between DHF and atypical neurological disturbances was first described in 1976.[2] On the basis of last few years

studies, neurological complications occur in 0.5–6% of the cases with dengue fever.[3] Various neurological manifestations reported in dengue fever are summarised into three categories: (I) manifestations due to neurotrophic effect of virus: rhabdomyolysis, myositis, myelitis, meningitis and acute encephalitis; (II) systemic manifestations of dengue fever: hypokalemic paralysis, papilledema, encephalopathy and stroke (hemorrhagic and ischemic) and (III) post-infection manifestations: fatigue syndrome, acute disseminated encephalitis (ADEM), encephalomyelitis, myelitis, neuromyeltis optica, optic neuritis, Guillain-Barre syndrome, phrenic neuropathy, long thoracic neuropathy, oculomotor palsy and maculopathy.[4] The common causes of ischemic stroke are thrombosis and embolic occlusion while uncommon causes includes hypercoagulable disorders, venous sinus thrombosis, fibromuscular dysplasia, vasculitis, subarachnoid hemorrhage vasospasm, drugs, moyamoya disease and eclampsia.[5] The major mechanisms that underlie ischemic stroke are occlusion or in situ thrombosis of intracranial vessels and hypoperfusion caused by limiting stenosis of cranial vessels often producing “watershed” ischemia. We excluded above causes of cerebral infarction by clinical examinations and laboratorical parameters. The pathogenesis of central nervous system involvement in dengue fever is largely due to vasculitis and leaky capillary syndrome with resultant fluid extravasations, cerebral oedema, hypoperfusion, hyponatremia, liver faluire and renal falure.[6] However, virus isolation from brain tissue and CSF of patients with neurological symptoms are reported which suggest direct virus invasion of the CNS and this may be related to dengue encephalitis.[7] There was no signs of capillary leak in our patient. Thus, encephalitis with acute intracerebral infarction in this case may have been related to vasculitis and direct virus invasion of the CNS.

5. Conclusion

It is clear that dengue fever can present a wide spectrum of atypical multisystem manifestations. Early diagnosis and management with prompt supportive care can reduce the morbidity and mortality of these patients by preventing neurological disabilities.

Table 1:

Laboratory parameters	Investigations during KGMU hospitalisation with date						
	18.11.12	20.11.12	22.11.12	26.11.12	28.11.12	01.11.12	03.11.12
Hb(g/dl)	12	11.4	11.0	11.4	10.0	9.8	10.2
TLC (103/µL)	14.7	13.8	11.5	9.4	8.5	8.5	8.2
DLC (%)	N78L18	N70L24	N68L10	N64L16	N70L15	N73L14	N74L20
PC (103/µL)	90.0	98.0	112	130	150.0	200	200
HCT (%)	38.1	34.8	36.0	36.4	38.4	35.8	36.4
S.Na+(mmol/L)	134	138	138	137	134	135	140
S.k+(mmol/L)	4.5	4.2	3.4	4.6	4.5	3.8	4.4
S.Urea(mgdl)	45.4	52.0	33.2	35.1	43.8	34.4	48.4
S.Creat (mgdl)	0.95	1.2	1.3	1.1	0.9	1.0	0.96
RBS(mg/dl)	114	102	98	201	180	112	108
PT(in seconds, control 12.1 s)	14.4			14.8		14.0	
INR (seconds)	1.2			1.0		1.08	
S.Bilirubin(mg/dl)	1.0		0.8			0.9	
ALT (IU/L)	100		37			40	
AST (IU/L)	112		52			44	
S.ALP	402		393			200	
S.Protein(g/dl)	5.8		5.4			6.0	
S.Albumin(g/dl)	3.6		3.2			3.0	

6. Abbreviations of Table

ALT, alanine transaminase; AST, aspartate transaminase; S.ALP, serum alkaline phosphatise; RBS, random blood sugar; PT, prithrombin time; INR, international normalised ratio

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Legend of Figures

Figure:1(a,b and c)- MRI brain showing signal intensity alterations s/o acute infarct in right corona radiata and central semiovale, right fronto-temporo-parieto-occipital region and left occipital area.

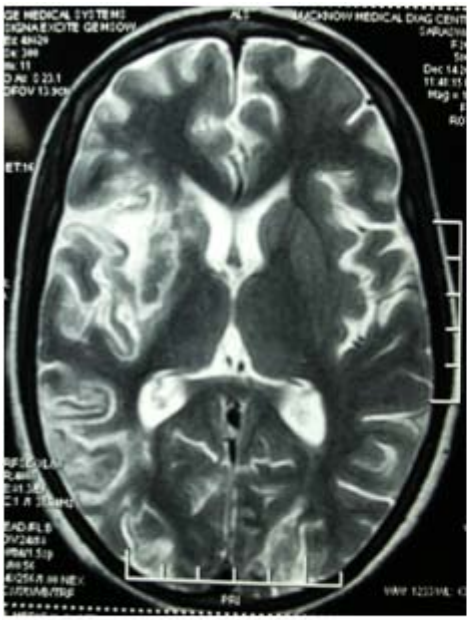

Figure 1(a): T2 image

T2F

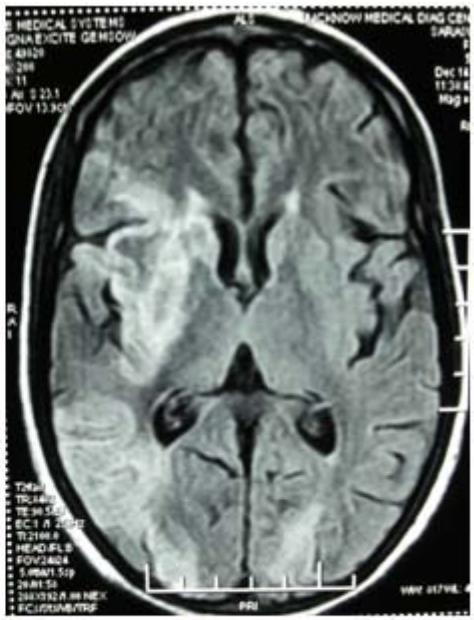

Figure 1(b): T2 Flair image

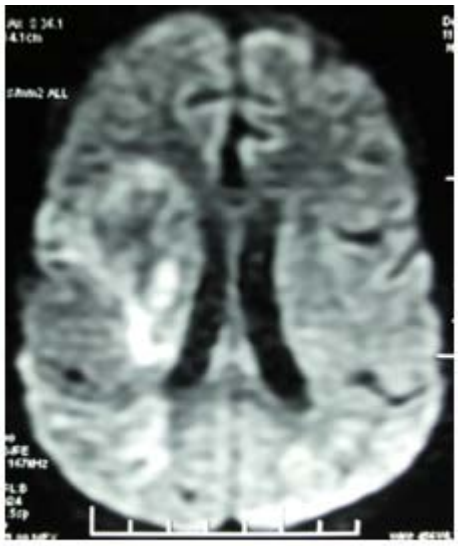

Figure 1(c): DWI image