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Intracranial Hemorrhage in Dengue Fever: A Case Series

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ABSTRACT

Background- Recently it is seen that clinical profile of atypical presentations in dengue fever is expending and these are being reported more frequently during last decade. Although there are many causes of Intracranial hemorrhage but dengue can also complicated to this fatal illness. We hereby reported two cases of dengue hemorrhagic fever, one of them was 45-year-old male farmer who was diagnosed as intracerebral hemorrhage and improved on conservative treatment. Other case was 30 years old male clerk and unfortunately he died because of acute subdural hematoma.

Key Words: Dengue fever, Intracerebral hemorrhage, Subdural hematoma, Aedes aegypti, Atypical manifestation

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INTRODUCTION

Dengue is an arboviral infection found in tropical and subtropical countries. It is transmitted by infected female aedes aegypti mosquito bite. Dengue virus has four distinct strains (dengue 1-4) and all have similar clinical syndrome. The typical presentation includes fever with macular rashes, headache, retro orbital pain along with the sever myalgia that gave rise to colloquial designation "break-bone fever". In rare cases second infection with different serotype from primary infection leads to dengue DH with severe shock.^[1] The overall mortality in dengue infection, without treatment is 1-5% and with proper treatment less than 1% while in severe infection 26%.^[2] reaches up to Dengue mortality hemorrhagic fever is reported to cause many bleeding manifestations including life threatening intracranial hemorrhages, epidural spinal hemotoma, or hematemesis/melena.^[3] Several have described reviews many atypical manifestations of dengue.

CASE REPORT 1;

A 45 years old male farmer was presented to our emergency department with complaints of low to high grade fever with rashes over limbs for 9 days and one episode of generalised seizure followed by altered sensorium of 1 day. At the time of admission patient was in unconscious state with a Glasgow coma score (GCS) of 9 (E2M4V3), There was no sign of meningeal irritation. Patient had no pallor, icterus or signs of dehydration. He was febrile (101°F). His pulse rate- 86/ min normovolumic and regular, B.P- 108/80 mmHg and respiratory rate was-18/min. Cardiovascular, respiratory and abdominal examinations were normal. He had no history of hypertension, mellitus. cardiovascular diabetes disease. atherosclerosis, trauma, seizure, drug abuses, coagulopathy, alcoholism, or any similar episodes of unconsciousness in past. His relevant laboratory investigations are summarised in table [1] Dengue IgM antibody in serum was positive. Serology of enteric fever and smear examination for malaria parasite were negative. Electrocardiography, 2D Echocardiography, abdominal ultrasonography, chest X-ray and Urine analysis were normal. Computed tomography of head (CT) showed intracerebral bleed in left region. Cerebrospinal frontal fluid (CSF) examination was not done. On the basis of clinical features and investigations, final diagnosis of this case was settled as left sided intracerebral hemorrhage secondary to dengue fever. He was managed conservatively with adequate intravenous fluids. intravenous antibiotics, antiepileptic drug and transfusion of six units' platelets. Patient improved and got discharged in well conscious state. He was on antiepileptic drug and asymptomatic up to 4 months of follow up.

CASE REPORT 2;

A 30 years old man was referred to our emergency department in unconscious state. He had complaints of low to high grade fever with rashes all over body for 13 days and altered sensorium of 2 days. On enquiry, patient was admitted in a private hospital since last 2 days. At the time of admission patient was unconscious with a

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Glasgow coma score (GCS) of 4 (E1M2V1) with hyperventilatory chest. He was febrile (100°F). He had mild pallor and no icterus. His pulse rate- 104/ min hypovolumic, Systolic blood pressure- 80 mmHg. Chest auscultation found minimal creptations in right side of chest. Abdominal examination was normal. He had no history of trauma, headache, hypertension, diabetes mellitus, cardiovascular disease, atherosclerosis, seizure, drug abuses, coagulopathy, alcoholism, or any similar episodes of altered sensorium in past. Available laboratory parameters during other hospital and KGMU are given in table1. Clinical **TABLE 1;** presentation of fever with rashes. thrombocytopenia and endemic area were suggestive of dengue fever. Positive serology of dengue was also evident of dengue infection. The patient was managed with intravenous fluid, vasopressors and broad spectrum antibiotics. He was put on mechanical ventilation and eight units platelets were transfused. Vitals were of monitored closely but blood pressure did not pick up even after 4 hours of therapy. Unfortunately patient could not be saved despite our best effort.

Laboratory	Case 1						Case 2	
parameters with	1 st Day	2 nd Day	3 rd Day	5 th Day	6 th Day	10 th Day	In Other	1 st Day
duration							Hostital	
Hb(g/dl)	14.0	13.5	13.8	14.2	13.6	14.2	11.5	10.0
TLC (103/µL)	6.2	8.80	11.0	11.0	9.2	8.2	12.6	13.0
DLC (%)	N60L12	N66L18	N67L20	N62L15	N64L18	N64L16	N72L14	N76L14
PC (103/µL)	17.0	18.0	30.0	70.0	100.0	135.0	20.0	18.0
HCT (%)	40.2	32.3	31.3	31.1	34.5	33.2	-	33.4
S.Na+(mmol/L)	134	-	136	140	-	141	132	130
S.k+(mmol/L)	3.8	-	4.3	4.0	-	3.8	4.0	4.3
S.Urea(mgdl)	44.4	-	40.8	-	-	32.0	50.4	54.0
S.Creat (mgdl)	0.95	-	0.82	-	-	0.80	1.10	1.20
RBS(mg/dl)	110	120	118	122	100	-	100	98
PT(in seconds,	14.8	-	13.4	-	-	13.0	-	14.2
control 12.1 s)								
INR (seconds)	1.32	-	1.22	-	-	1.2	-	1.3
ALT (IU/L)	80.	-	-	-	74	-	-	82
AST (IU/L)	70	-	-	-	68	-	-	80
S.ALP	90	-	-	-	60	-	-	90
S.Protein(g/dl)	7.2	-	-	7.0	-	-	-	6.4
S.Albumin(g/dl)	4.1	-	-	4.0	-	-	-	3.2
S.Lipase(U/L)	-	-	-	-	-	-	-	80
S.Amylase(U/L)	_	-	_	-	-	-	-	100

ALT, alanine transaminase; AST, aspartate transaminase; S.ALP, serum alkaline phosphatise;

RBS, random blood sugar; PT, prothrombin time;

INR, international normalised ratio

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Figure 1 (A and B)

Figure 1 (A and B)- Ill defined heterogeneous lesion of haemorrhagic attenuation with adjacent

area of hypo density /clot retraction noted in left frontal region s/o bleed.



Figure 2 (A and B)

Figure 2 (A and B)- Ill defined extra axial concavo-convex collection of haemorrhagic attenuation s/o subdural haematoma(red arrow) in

right fronto-temporo-parietal region with subarachnoid extension(white arrow).

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DISCUSSION

Dengue fever is a common mosquito borne disease in north India. The common presentations of dengue are high grade fever, myalgia, arthralgia, patechie, epigastric pain, nausea and Less common presentations vomiting. are epistaxis, hematurea, hemetemesis and other bleeding manifestation.^[4] Only few cases of Intracranial hemorrhage in dengue fever are reported in literatures.^[3,5] 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 incorporate variety of system to atypical presentations of dengue infection affecting various organ systems including hepatic, neurological, gastrointestinal, pulmonary and renal.^[6] 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) postinfection 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.^[7] The exact pathogenesis of hemorrhagic complications is not well understood. Impairment of platelet function can

also increases the risk of vascular fragility which may lead to hemorrhage. However possible mechanism of bleeding diathesis in dengue be due to vasculopathy, infection may thrombocytopenia and platelet dysfunction.^[8] Low platelets count in dengue fever are due to both from decreased production and increased destruction which correlates with the clinical severity of DHF.^[9] Hemorrhagic complications and circulatory collapse are the major causes of mortality in these patients. Therefore, a high index of suspicion needs to be maintained in febrile patients with atypical manifestations, especially in dengue endemic areas.

CONCLUSION

Hemorrhagic complications and circulatory collapse in dengue fever remains major causes of mortality in epidemics. Our case report highlights the potential danger of dengue fever. Guidelines of managing such complications are not mentioned in dengue literature so treating physician must be aware of the development of atypical neurological manifestations.

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