



Expanded Dengue Syndrome: A case Series

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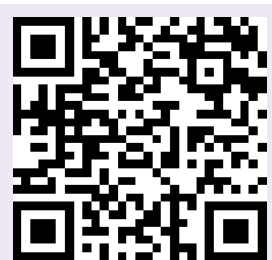
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Abstract

Dengue fever is an alarming arboviral infection of utmost global importance with many serotypes. Expanded dengue syndrome is a term first introduced in 2011, which refers to unusual and severe involvement of the liver, kidney, brain or heart in association with dengue. The following series of six expanded dengue syndrome cases fills the lacunae of knowledge gaps in this field. All the six cases of age groups between one month to 18 years presented to our hospital. The patients had life-threatening complications including acute liver failure, acute pancreatitis, myocarditis, pericardial effusion, encephalitis, pulmonary edema, acute kidney injury, and acute liver failure. The children were diagnosed as expanded dengue syndrome and managed thus. Early detection and intervention of children with expanded dengue syndrome can be very beneficial in decreasing the mortality and morbidity of dengue.

Introduction

Dengue fever is a viral illness caused by RNA viruses transmitted by *Aedes aegypti* mosquito. It is prevalent in most Indian states, peaking around monsoon season. As per the recent data, more than one lakh dengue cases were documented in India between January and October, 2022.^{1,2} Prevalence of expanded dengue syndrome among children (1 month - 18 year) hospitalized for dengue fever in India is up to 18.5% as per Mahajan V et al.³ As per national centre for vector borne disease control program, Haryana reported 6468 cases of dengue in 2024.⁴

Atypical manifestations are now often being seen due to escalating disease burden. Healthcare professionals often overlook these unusual presentations. Lack of awareness poses challenges in treatment, leading to more complications, morbidity, and mortality. There is dearth of literature on children with dengue expanded syndrome, especially in our parts of the world.

In this series of cases, we present individuals with confirmed dengue through serological testing using the immunochromatographic dengue card test, showcasing expanded dengue syndrome. These findings not only aid in prompt diagnosis but also help in reducing morbidity and mortality risks.

Case 1- Dengue encephalitis with right-sided hemiparesis

A 5 years toddler boy with normal anthropometric indices came with complaints of fever since 10 days, drowsiness, and decreased movement of the right upper



limb and lower limb for two days. On admission the child was sick with confused mental status and GCS of E4V4M4 with normal vital parameters. In CNS examination deep tendon reflexes were exaggerated with decreased tone and power of right upper limb and lower limb (2 / 5 as per MRC scale), extensor plantar right lower limb and normal response in left lower limb with no other CNS signs. In the respiratory system examination air entry was slightly reduced on right side. Investigations revealed raised TLC with neutrophilic predominance, thrombocytopenia and positive dengue NS1Ag (Table 1). Chest x ray showed blunting of CP angle on right side with mild pleural effusion. CSF study was acellular with normal sugar and protein content. MRI showed focal nonspecific white matter changes in the right parietal lobe. The child was started on antibiotics empirically with supportive care. Hemiparesis gradually resolved and GCS improved, and he was discharged after 10 days of hospitalisation.

Case 2- Severe dengue with empyema and pericardial effusion

A 4-year-old toddler boy presented with complaints of fever for a week, loose stool, vomiting, and cough for four days. Child was sick with a pulse rate of 110 / min, good volume and respiratory rate of 44 / min with distress, BP = 100 / 62 (Systolic and diastolic BP > 50th centile for his height), CRT < 3 sec and SpO₂ of 88% on 2 liters oxygen support by nasal cannula. Hence he was shifted to non-invasive mechanical ventilation. Child was moderately underweight as per WHO growth charts. Respiratory system examination revealed bilateral bronchial breathing, crepitations and reduced air entry with sub-costal and intercostal retractions. Per abdomen examination showed tender hepatomegaly with a liver span of 7.5 cm. In cardiovascular system examination heart sounds were slightly muffled with no murmur. Investigations revealed metabolic acidosis, polymorphic counts, thrombocytopenia, elevated CRP, ESR and positive dengue NS1Ag (Table 1). CXR showed left-sided gross pleural effusion with consolidation (Figure 1). Pleural tap was done which showed exudative effusion. IV antibiotics were started immediately and ICD was inserted. ECG showed low voltage complexes, prompting an echocardiography which revealed pericardial effusion. Child was referred for pericardiocentesis after 48 hours of admission. On follow up, the child was recovering well and completed four weeks of IV antibiotics followed by oral medication.

Case 3 - Severe dengue with myocarditis and type 1 respiratory failure

A 9 year old school girl with normal nutritional status came with complaints of fever and vomiting for three days and lethargy since 1 day. On admission, she was sick, irritable but oriented. She had respiratory distress, tachycardia with feeble pulses, CRT < 3 sec and hypotension. The recorded

SpO₂ on 5 litres of oxygen was 90 - 92% and hence child was shifted to non-invasive mechanical ventilation. She also had periorbital puffiness. She had tender hepatomegaly with positive fluid thrill. Respiratory system examination revealed bilateral reduced air entry. Investigations revealed positive NS1Ag with leucopenia, raised D-dimer, and deranged LFT (Table 1). CXR showed bilateral pleural effusion (Figure 2). ECG showed ST segment elevation with T wave inversion and raised CK-MB suggestive of myocarditis and child was managed conservatively. She was discharged in stable condition after seven days of hospitalization.

Case 4- Severe dengue with pancreatitis with type 2 respiratory failure

A 14 year old female teenager with normal nutritional status presented with history of fever, abdominal pain, loose stool, and fast breathing since three days. On admission, she was sick with GCS of E3V3M4. She was tachycardic with feeble pulses, hypotensive and having labored breathing pattern with respiratory rate of 48 / min. She also had generalized anasarca and positive tourniquet test. On chest auscultation, there was reduced air entry bilaterally. She had epigastric tenderness and hepatomegaly. She had slightly low GCS of E3V3M4 with neurological status. Her investigations showed thrombocytopenia, positive dengue NS1Ag, elevated amylase and lipase (Table 1). X-ray showed pleural effusion and USG abdomen was suggestive of fluid in perihepatic, peri-splenic, and inter-bowel regions. MRI brain showed non specific white matter changes in right parietal lobe (Figure 3). She was intubated immediately in view of respiratory failure and started on ventilatory support. Supportive management with colloids and ionotropic support was given. Unfortunately, she could not be revived and succumbed after four hours.

Case 5 - Severe dengue with acute liver failure with pulmonary oedema

A 3 year old female toddler was brought with complaints of fever and cough since five days, and abdominal pain for one day. General condition of the child was very sick. She was irritable with a pulse rate of 132 / min with feeble pulses, prolonged CRT, hypotensive and respiratory rate of 62 / min. She did not maintain saturation 5 liters of oxygen and hence was put on NIV. She had hepatomegaly with positive shifting dullness. In respiratory system examination she had bilaterally reduced air entry. Investigations revealed metabolic acidosis, leukocytosis, Positive NS1Ag, thrombocytopenia, deranged SGOT / PT with increased INR with bilateral pleural effusion and ascites (Table 1). She was managed with boluses, N-acetyl cysteine, antibiotics along with FFP. On third day, her distress worsened. She was started on diuretics for pulmonary oedema. She was gradually weaned off ventilator and subsequently discharged with normal liver function tests after 14 days.

Case 6 - Dengue shock syndrome with MODS

An 8 year old school boy with a normal BMI status presented to the emergency department with complaints of abdominal distension for 10 days, fast breathing, and facial puffiness for past two days. The boy was being managed with NSAIDs, antibiotics and steroids in the previous hospital. On admission, the boy was sick, lethargic, tachycardic with feeble pulses, hypotensive and under respiratory distress. The boy was hypoxemic with bilaterally reduced air entry and coarse crepitations on chest. He has massive hepatomegaly with liver span of 12 cm and positive shifting dullness. He was intubated immediately in view of impending respiratory failure. Investigations were suggestive of secondary sepsis with polymorphic count and a strongly positive dengue NS1Ag by ELISA (Table 1). Chest x-ray showed bilateral prominent broncho vascular markings with radio opacity and pleural effusion. Ultrasound was suggestive of heterogeneous echo texture with dilated portal vein, GB wall oedema, and ascites, suggestive of pancreatitis (Figure 4). ECG showed ventricular tachycardia with wide QRS complex and echo showed findings of myocarditis. Child was immediately started on crystalloids, inotropic support and antibiotics. As SGOT and SGPT levels increased over the next 24 hours, along with a deranged INR, N-acetyl cysteine and FFP transfusion was given but child continued to deteriorate. Over next 24 hours, child had bleeding from multiple sites and repeated transfusions were done. A probability of DSS with MODS with severe sepsis was kept. In spite of all resuscitative measures, child succumbed after 60 hours of admission.

Discussion

In our case series, various manifestations of expanded dengue syndrome were seen. The predominant presentation was acute respiratory failure, followed by fluid overload and shock. Acute respiratory failure developed in five out of six cases. Of these two were intubated, two required NIMV support and one needed oxygen support.

It is assumed that fluid overload of more than 10% in initial 24 hours is a risk factor for prolonged ventilator use and PICU stay.⁵ Careful assessment of input output status of PICU patients is utmost important to reduce complications. However, a retrospective cohort study in 154 DSS children showed no differences with liberal or restrictive fluid therapy. It showed that infection and inflammatory mediators causes organ dysfunction prolonging PICU stay.⁶

Fluid overload is a known complication in dengue. Vascular leakage in dengue promotes aggressive fluid therapy which could be prevented with colloids. Hourly observations with immediate access to hematocrit testing and careful fluid titration forms the basis of management of critically ill dengue patients.⁷

Next common manifestation in our case series was that of acute liver failure (ALF) and MODS. Liver failure is the terminal pathway in treating critical dengue patients which may occur due to prolonged shock and fluid overload.⁸ Our patients were treated with IV N-acetyl cysteine based on the evidence of paracetamol poisoning and a Sri Lankan study which showed improvement with N-acetylcysteine.⁹ Wijewickrama et al noted that excessive NSAIDs during illness heightens the susceptibility to bleeding and hepatic enzyme abnormalities.¹⁰ The WHO guidelines advise against the use of NSAIDs such as ibuprofen or antipyretic in dengue fever.¹¹ A paediatric study done on 190 children with dengue showed deranged liver enzymes in more than 50% patients.¹² Four out of six cases in the present series developed MODS. Early targeted and timely therapy with appropriate antimicrobial will certainly be the mainstay of therapy.¹³ Dengue serotypes also contribute to disease severity with DEN2 being more severe.^{14,15}

Dengue with CNS manifestations was an unusual presentation found in one patient with right sided hemiparesis. So, severe dengue can present with central nervous system symptoms without shock or hemorrhage.¹⁶ Therefore, healthcare professionals in dengue-endemic areas should remain more vigilant for neurological signs.¹⁷ Dengue with myocarditis was seen in two out of six cases. Cristodulo et al found 4.2% myocarditis in hospitalized dengue patients.¹⁸ Early detection of these complications through close monitoring and detailed cardiac evaluation is crucial to reduce mortality in dengue patients. Pancreatitis is one of the rare complications in dengue and very few case reports have been published. This could be resulted due to direct viral infiltration leading to inflammation and destruction of pancreatic acinar cells or an autoimmune reaction.^{19,20} Hence, if a child presents with acute abdomen following dengue fever, the rare complication of acute pancreatitis should be considered.

Conclusions

Dengue can have multi-system, unrecognized and atypical presentations. Hence medical professionals should have a high index of suspicion for timely diagnosis of the varied manifestations. Only appropriate timely intervention with spurious monitoring of hemodynamics can reduce morbidity and mortality

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Table 1: Haematological and biochemical parameters of the cases

| Investigations | Case 1 | Case 2 | Case 3 | Case 4 | Case 5 | Case 6 | Reference |
|--|--------------------------------------|---|--------------------------------------|-----------------------------|--------------------------------------|---|----------------------------------|
| Hb (g / dl) | 8.8 | 11.3 | 11.4 | 11.2 | 11.0→10.6 | 11.2 | 12.0-15.0 |
| PCV (%) | 27.5 | 35.0 | 35.2 | 34 | 33.4 | 30.2 | 35-45 |
| PC (Lakh / cumm) | 1.34 | 1.50 → 1.2 → 0.9 | 4.33 → 1.50 → 0.75 to 0.45 | 50k | 80k → 55K → 1L → 1.5 | 1.50 → 2.6 | |
| TLC (Cells / cumm) | 18790 | 32100 → 29600 | 3100 → 4330 | 3200 | 36920 → 26k → 8800 | 28400 | 4000 -10000 |
| DLC | N = 77; L = 17; E = 01; M = 05 | N = 81; L = = 12; E = 01; M = 01 | N = 85; L = 11; E = 02; M = 02 | N = 88L = 8E = 2M = 2 | N = 77; L = 17; E = 01; M = 05 | N = 90; L= 7; E = 1; M = 02 | |
| CRP (mg / L) | 43.2 → 6.5 | 205 → 94.1 | 26.8 → 27.7 | 50 | 19.9 | 24 | 0 - 5 |
| D-Dimer (ng / ml) | - | 887.0 | - | - | - | 1130 | 0- 250 |
| SGOT (IU / L) | 33.7 | 52.9 → 105.8 | 626 → 1140 | 485 | 4500 → 128 | 92.6 → 1045 | 0 - 35 |
| SGPT (IU / L) | 17 | 14.1 → 41.3 | 253 → 740 | 316 | 4600 → 873 | 58.6 → 5060 | 0 - 45 |
| GGT (U / L) | 27.8 | 22.8 | 85.7 | 110 | 61.5 | 115 | < 24 |
| PT / aPTT / INR | 12.4 / 33.1 / 1.06 | 12.3 / 33 / 1.05 | 15.8 / 61.2 / 1.4 | 11.6 / 29 / 1.1 | 14 / 41 / 1.9 → 12.8 / 33.61.1 | 31 / 61.5 / 2.6 → 34.3 / 58.5 / 2.6 | PT: 10 - 14 INR: 0.8 - 1.2 |
| Serum Albumin (g / dl) | 2.34 | 2.21 → 1.93 | 3.51 → 1.97 | 3.5 | 2.68 | 2.57 → 2.45 | |
| Serum Lipase (IU / L) | - | - | - | 624 | - | 34.8 | |
| Serum Amylase | - | - | - | 410 | - | 55.9 | |
| Urea / Creatinine (mg / dl) | 31.7/0.65 | 9.2/0.13 | 41.2/0.61 | 40/0.8 | 26.6/0.53 | 46.7 / 1.04 → 53.2 / 1.88 | U:18 - 55 Cr: 0.6 - 1.1 |
| Sodium / Potassium (mmol / L) | 132 / 4.2 | 132 / 3.8 | 134 / 4.6 | 135 / 4.3 | 131 / 4.3 | 143 / 4.4 | Na:136 -145 K: 3.5 - 4.5 |
| NS1 Antigen | + | + | + | Outside + | Outside + | + | > 11 |
| Dengue IgM | + | - | - | - | - | + | > 11 |
| CK - MB (U / L) | - | - | 54.3 Raised | - | 151.7 | - | 0- 16 |
| Fluid cytology | 5 cells, all mononuclear | Pleural fluid-6000 cells,all neutrophils | - | - | - | - | |
| Fluid biochemistry | Sugar-69.1; Protein -92.1 | Sugar-29, protein-6 | - | - | - | - | |
| Hb - Haemoglobin; PCV - Packed cell volume; PC - Platelet count; TLC - Total leucocyte count; DLC - Differential leucocyte count; CRP - C Reactive Protein; PT - Prothrombin time; GGT - Gamma glutamyl transferase; CK MB - Creatinine kinase | | | | | | | |

Case 2



Figure 1 : Chest X Ray showing U/L pleural effusion

Case 3

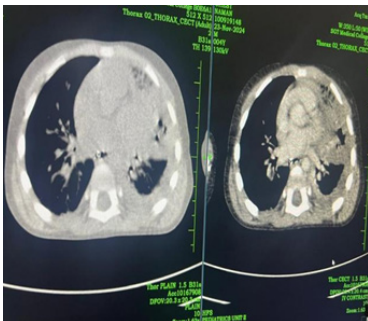


Figure 2 : CT Scan showing massive consolidation

Case 4

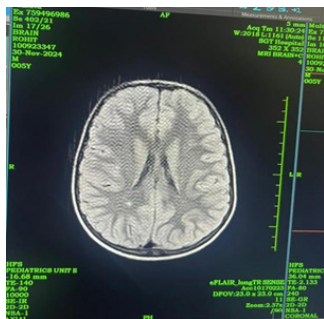


Figure 3 : Focal non specific white matter changes in right parietal lobe

Case 6

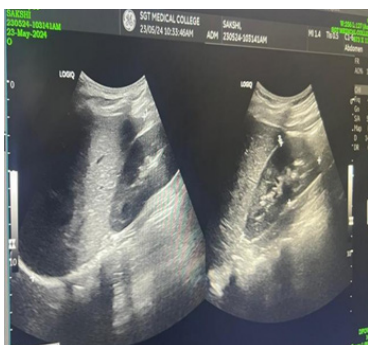


Figure 4 : Fluid in the perihepatic, perisplenic and interbowel region with dilated main pancreatic duct s/o pancreatitis

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