PEDIATRIC ONCALL
CHILD HEALTH CARE

CASE REPORTS

ATYPICAL SEVERE ORGAN INVOLVEMENT IN PAEDIATRIC DENGUE: A CASE SERIES FROM A TERTIARY INTENSIVE CARE UNIT IN SOUTH INDIA

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ABSTRACT

Background: Dengue is a major cause of paediatric hospitalization in endemic regions, presenting with atypical organ-specific complications that are easily overlooked. This series describes children with dengue and uncommon cardiac, neurological, and hepatobiliary involvement managed in a tertiary paediatric intensive care unit.

Case presentation: 11 children with serologically confirmed dengue infection were admitted to PICU of Narayana Health City, Bengaluru, during August–October 2024. The cohort manifested life-threatening complications, including myocarditis with cardiogenic shock requiring inotropes, levosimendan, and, in one case, VAECMO, ANEC; and hepatobiliary dysfunction with features suggestive of secondary HLH. All patients received protocol-based dengue management, individualized organ support, and, where indicated, immunomodulatory therapy with corticosteroids, IVIG, and biologic agents such as Anakinra. 3 had single-organ involvement, while others showed multisystem disease with significant plasma leakage. 7 out of 11 children to hospital discharge with some requiring prolonged ICU care.

Conclusions: Atypical manifestations of dengue involving the heart, liver, and central nervous system are not uncommon. Early recognition, appropriate referral to intensive care, meticulous fluid and haemodynamic management, and judicious use of immunomodulation are essential to improving outcomes in such cases.

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ABBREVIATIONS

HLH - Hemophagocytic lymphocytic histiocytosis

IVIG - Intravenous Immunoglobulin

PLEX - Plasma Exchange

CRRT - Continuous Renal Replacement Therapy

ANEC - Acute Necrotising Encephalitis

SGOT - Serum glutamic oxaloacetic transaminase

SGPT - Serum glutamic pyruvic transaminase

IVMPS - Intravenous Methylprednisolone

VA ECMO - Venoarterial Extra corporeal membrane Oxygenation

POCUS - Point of Care Ultrasound

Introduction

Dengue, with a mortality rate of around 2.5%, it continues to be a major public health concern with children particularly at risk.[1] The clinical spectrum ranges from mild, self-limiting illness to severe disease characterized by haemorrhagic manifestations, plasma leakage, and organ failure. The exact mechanism of severe manifestations of dengue is unknown but increasing evidence indicating that they are largely immunopathologically mediated affecting multiple organs leading of death.[2] No specific therapy for dengue is currently available; therefore, management remains supportive and focuses on meticulous fluid therapy, haemodynamic optimization, and organ support. This case series describes 11 children with serologically confirmed dengue who presented to a single tertiary-care centre during the same epidemic season with varied cardiac, hepatic, and neurological complications requiring individualized intensive care management.

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Case Report

Group A: Myocardial dysfunction

Four children aged 6–12 years with dengue were referred with persistent shock despite guideline-directed fluid resuscitation. One child was intubated while the others were receiving supplemental oxygen, and inotropic support viz epinephrine

POCUS demonstrated a plethoric inferior vena cava, depressed left ventricular systolic function (ejection fraction ≤25%), and bilateral pulmonary edema with confluent B-lines. ABG revealing severe metabolic acidosis with hyperlactataemia, hypoxia, and hypercapnia, reflecting impaired peripheral perfusion and cardiogenic pulmonary edema. Inflammatory markers, including C-reactive protein and ferritin, were modestly elevated.

Challenge was fluid titration to keep macrocirculation intact with support of Inotrope and ventilation. All patients received levosimendan infusion and immunomodulation

with IVMPS (10–30 mg/kg) and IVIG (2 g/kg). Three children demonstrated progressive improvement in cardiac function and peripheral perfusion, but one went on to VAECMO due to refractory hypotension with persistently poor ventricular function despite maximal medical therapy. All 4 of them survived.

Myocarditis has been reported at approximately 11.3% dengue patients.[3] Management is primarily supportive and aims to maintain adequate organ perfusion while the acute inflammatory phase resolves. Although data for corticosteroids versus IVIG in dengue myocarditis are lacking, immunomodulation is often considered in fulminant cases given the suspected immune-mediated pathophysiology.[4,5]

Group B: HLH

Six children aged 9–14 years presented with fever, myalgia, vomiting, abdominal distension, and varying degrees of encephalopathy having deranged liver function, elevated ferritin, metabolic acidosis, and hyperlactataemia; hyperammonaemia requiring CRRT and PLEX suggestive of secondary HLH.

Immunomodulation included intravenous methyl-prednisolone (IVMPS), anakinra, and PLEX, with dosing of anakinra and steroids decided in consultation with paediatric rheumatology. CRRT and PLEX were initiated according to severity of organ dysfunction. Despite efforts, this subgroup had highest mortality with 4 of 6 succumbing. This observation is consistent with reports of Bhat et al[6] who demonstrated that extreme hyperferritinemia (ferritin >10,000 ng/mL) in children with severe dengue is associated with worse outcomes. Severe dengue is characterized by an inflammatory response with elevated levels of cytokines which contribute to endothelial injury, increased vascular permeability, and MODS. Anakinra, IL-1 receptor antagonist can attenuate the downstream inflammatory cascade, limit hepatocellular necrosis, and help stabilize endothelial function. High mortality associated with hyperferritinemia and HLH-like presentations indicates early recognition, rapid referral to tertiary care, and timely initiation of advanced organ support and immunomodulation.[6]

Group C: Acute necrotizing encephalopathy of childhood (ANEC)

A 12-year-old girl with dengue NS1 antigen positive presented with a 4-day history of fever, altered sensorium, and seizures was comatose and required immediate intubation. MRI demonstrated bilateral thalamic hyperintensities on T2-weighted and FLAIR sequences, characteristic of ANEC. She was treated with IVMPS at 30 mg/kg/day for 5 days and IVIG at 2 g/kg. The patient was successfully extubated after 3 days of ventilation but was discharged with residual neurological deficits.

Immunomodulatory treatment is central component of management in ANEC, although guidelines are lacking. In the present case, a combination of pulse-dose corticosteroids and IVIG was used, consistent with previous case reports and series like Okumura et al.[8]

Figure 1: MRI finding of the patient with ANEC showing T2 Flair image with bilateral thalamic hyper intensities.

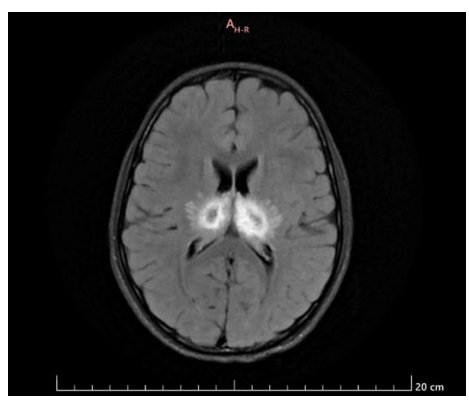

Table 1: Details of patient admitted with myocardial dysfunction

Parameters(at admission)	patient 1	patient 2	patient 3	patient 4
age (in years)	6	9	13	8
Gender	Male	female	Female	male
day of illness	4	4	5	5
Hb (at admission)(g/dl)	14	13.1	13.6	12.1
PCV (%)	42.7	43	42.3	37
platelet count (/cumm)	50000	35000	150000	60000
CRP(mg/dl)	8.3	1.8	0.725	1.8
Ferritin (ng/ml)	211	2100	554	4080
SGOT/SGPT	129/97	86/90	107/83	301/111
Creatinine(mg/dl)	0.65	0.79	0.86	0.69
pH	7.3	7	7.29	7.11
Base excess	-7	-19.7	-17.5	-14.3
Lactate(mmol/l)	3.9	8.3	13.5	6.9
Pao2 (mmhg)	55	53	53	56
Pco2(mmhg)	35	47	19	48
MPS	30 mg/kg (3 doses)	10 mg/kg (3 doses)	10 mg/kg (3 doses)	10 mg/kg 1 dose
IVIG	2 g/kg	2 g/kg	1 g/kg	2 g/kg

ECMO	No	no	No	Yes
levosimendan (0.1 mcg/kg/min for 48 hrs	Yes	yes	Yes	no
invasive mechanical ventilation	Yes	yes	Yes	Yes
CRRT	no	no	No	no
Outcome	discharge	discharge	discharge	discharge

Table 2: Details of patients admitted with hepatobiliary dysfunction/HLH.

Parameters(at admission)	patient 1	patient 2	patient 3	patient 4	patient 5	patient 6
age (in years)	14	12	10	9	5	6
gender	male	female	female	Female	Male	male
day of illness	5	4	4	4	3	4
Hb (at admission)(g/dl)	14.9	14.8	14	14.4	13	13.2
PCV (%)	45.2	46.2	42.4	44	38.6	43.1
platelet count (/cumm)	25000	35000	50000	10000	25000	30000
CRP(mg/dl)	113	3.09	2.9	9.4	3.29	50
Ferritin (ng/ml)	23900	94200	24100	31200	18100	45500
SGOT/SGPT	2809/951	7295/1925	2518/1249	4991/2237	4536/1280	5000/2300
INR	1.92	2.59	2.7	1.87	2.24	2.73
Creatinine(mg/dl)	0.65	0.74	0.83	0.78	0.43	0.92
pH	7.06	7.14	7.28	0.31	7.25	7.1
Base excess	-25	-21	-13.1	-13	-11.4	-17.3
Lactate(mmol/l)	11	9.5	4.4	5	6.4	7
Pao2 (mmhg)	55	100	49	194	332	54
Pco2(mmhg)	18	23	29	26	36	38
MPS	YES	YES	NO	YES	YES	NO
IVIG	NO	NO	NO	1G/KG	1G/KG	NO
ECMO	NO	NO	NO	NO	NO	NO
levosimendan (0.1 mcg/kg/min for 48 hrs	NO	NO	NO	NO	NO	NO
invasive mechanical ventilation	YES	YES	YES	YES	YES	YES
CRRT	YES	YES	NO	YES	NO	NO

Conclusions

Atypical manifestations of paediatric dengue, including myocardial dysfunction, HLH-like hyperinflammatory hepatobiliary disease, and ANEC, were observed in this case series from a single tertiary-care centre during one epidemic season. These phenotypes are associated with high risk of organ failure and mortality, especially in children with hyperferritinemia and hepatic dysfunction. Early recognition of these complications, prompt referral to centres with paediatric intensive care capability, meticulous hemodynamic and organ support, and judicious use of immunomodulatory therapies—such as corticosteroids, IVIG, anakinra and PLEX tailored to the organ system involved and disease severity—are essential to optimizing outcomes.

Compliance with Ethical Standards

Funding: None

Conflict of Interest: None

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