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Case Report

A Rare Pediatric Case of Dengue Hemorrhagic Fever with Encephalopathy and Suspected Subdural Hemorrhage

Aulia Cempakasari[1], Tiara Rahmawati[1], Alief Qobidh Al Bashor Arifin[1], Dewi Syahputri[1], Firdha Yekti Hutami[1], Pramita Anindya Nugraheni[2], Renata Prameswari[3], Ronald Pratama Adiwinoto[4*], I Made Dwi Mertha Adnyana[5,6]

[1] Faculty of Medicine, Hang Tuah University, East Java, Indonesia
[2] Department of Pediatric, Faculty of Medicine, Hang Tuah University, East Java, Indonesia
[3] Department of Dermatology, Venereology, and Aesthetics, Faculty of Medicine, Hang Tuah University, East Java, Indonesia
[4] Department of Community Medicine, Faculty of Medicine, Hang Tuah University, East Java, Indonesia
[5] Department of Medical Professions, Faculty of Medicine and Health Sciences, University of Jambi, Jambi, Indonesia
[6] Royal Society of Tropical Medicine and Hygiene, London, United Kingdom

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CORRESPONDING AUTHOR*
Ronald Pratama Adiwinoto
adiwinoto.ronald@hangtuah.ac.id
Department of of Community Medicine,
Faculty of Medicine, Hang Tuah University,
Surabaya, East Java, Indonesia

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ABSTRACT

Introduction: Dengue Hemorrhagic Fever (DHF) is a severe arboviral infection that rarely leads to neurological complications. Dengue encephalopathy, often secondary to metabolic disturbances or shock, presents a significant diagnostic and management challenge in the pediatric population.

Case Presentation: We report the case of a 4-year-3-month-old obese girl presenting with fever, gastrointestinal symptoms, and progressively decreasing consciousness (apathetic). Diagnosis of DHF was confirmed by a positive NS1 antigen test, severe thrombocytopenia (nadir 14,000/µL), and hemoconcentration with pleural effusion. Hepatic involvement was noted (SGOT 117.81 U/L). Neurological involvement was characterized by clinical encephalopathy, and a head CT scan revealed hyperdense lesions suspecting subdural hemorrhage (SDH). The patient required multidisciplinary management in the Intensive Care Unit (ICU) for supportive fluid therapy, inotropic support (Dobutamine), and platelet transfusion. The patient showed marked clinical improvement and was discharged on day nine. At a two-week post-discharge follow-up, she demonstrated full cognitive and motor recovery with no neurological sequelae

Conclusion: This case highlights the critical need for clinical vigilance for rare, simultaneous neurological complications (encephalopathy and suspected SDH) in pediatric DHF. It underscores that early diagnosis and aggressive, multidisciplinary supportive care can lead to complete recovery even in severe presentations.

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INTRODUCTION

Dengue Hemorrhagic Fever (DHF), caused by the dengue virus (DENV), is a major public health challenge in tropical regions, including Indonesia [1,2]. While typically characterized by fever, plasma leakage, thrombocytopenia, and bleeding manifestations[3,4], DHF can also lead to severe atypical complications [5–8].

Neurological involvement in dengue is rare, with an estimated global incidence of 0.5% to 6.2%.[9] These complications are particularly concerning in children, who are at higher risk for severe disease [9,10]. A primary diagnostic dilemma is distinguishing dengue encephalopathy from dengue encephalitis.

Encephalopathy is a syndrome of global brain dysfunction, often secondary to systemic metabolic insults (e.g., hepatic dysfunction, electrolyte imbalance, shock, hypoxia) or a systemic inflammatory 'cytokine storm' [11,12]. Conversely, encephalitis implies direct viral neuro-invasion.

While dengue-associated encephalopathy is documented, its co-presentation with significant intracranial hemorrhagic manifestations, such as subdural hemorrhage (SDH), in a toddler is infrequently reported. This case report describes the clinical course, diagnostic challenges, and successful multidisciplinary management of a 4-year-old girl with DHF, severe encephalopathy, and imaging suggestive of SDH. This report has been prepared in accordance with the CARE (CAse REport) guidelines.

CASE PRESENTATION

This study is a descriptive, retrospective analysis of a patient's medical records, including clinical findings, laboratory examinations, and radiological imaging. Ethical approval was obtained from the Institutional Review Board of Hang Tuah University, and written informed consent was provided by the patient's parents.

A 4-year-3-month-old girl (Body Weight: 33 kg, Height: 105 cm; indicating obesity) was brought to the Emergency Department (ED) of Citra Medika Hospital, Mojokerto. The patient's parents reported a 2-day history of intermittent fever (38.8°C), vomiting (3x/day), and diarrhea (4x/day). The patient also complained of abdominal pain and headache, appearing lethargic and refusing food. Past medical history was significant for broncho-pneumonia at 3 months of age.

On initial physical examination (Day 3 of illness), the patient was ill but conscious (compos mentis), with a temperature of 39°C, blood pressure of 101/68 mmHg, pulse rate of 136 bpm (strong), and respiratory rate of 28 breaths/minute. Petechiae were noted on the right hand.

On the third day of hospitalization (Day 5 of illness), the patient's condition deteriorated, necessitating transfer to the Intensive Care Unit (ICU) due to a decrease in consciousness (apathetic). She developed signs of compensated shock: T=35.9°C (hypothermia), BP=100/60 mmHg, pulse=130/min (weak and thready), RR=31 breaths/minute, and cold extremities. A summary of the clinical progression is provided in Table 1.

Investigation

Laboratory evaluation on Day 3 showed a platelet count of 183,000/µL (Normal Range: 150,000–450,000/µL). Following transfer to the ICU on Day 5, the NS1 test returned positive. Serial blood counts revealed progressive, severe thrombocytopenia, dropping from 55,000/µL to a nadir of 14,000/µL. Evidence of hemoconcentration (plasma leakage) was confirmed by a chest X-ray showing right-sided pleural effusion. Hepatic involvement was confirmed with an elevated SGOT of 117.81 U/L (Normal Range: <40 U/L) and an SGPT of 39.42 U/L (Normal Range: <40 U/L). Key laboratory findings are summarized in Table 2.

A head CT scan without contrast revealed a hyperdense lesion in the superior sagittal sinus and bilateral subtentorial region. These findings were suspicious for dengue encephalopathy complicated by subdural hemorrhage (SDH) (Fig.1A-C). Treatment was administered according to the patient's evolving condition. ICU care was managed by a multidisciplinary team, including supportive fluid therapy, antipyretics, platelet transfusion, intensive neurological monitoring, and hemodynamic observation. During the course of treatment, the patient showed significant clinical and laboratory improvement. She was transferred to a low-care inpatient unit and was later discharged in good condition, with no complaints, stable vital signs, and normal laboratory results (Fig.2,3).

Treatment

Management was adaptive and multidisciplinary, focusing on hemodynamic stabilization and supportive care. Initial ED management involved fluid resuscitation (IVFD KAEN 3B), antipyretics (Santagesik), and antiemetics (Ondansetron).

In the ICU, management was escalated. Oxygen therapy was provided (nasal cannula, 1–2 L/min). Due to signs of shock and poor perfusion, Dobutamine was initiated at 3 mcg/kg/min (later titrated to 7.5 mcg/kg/min) *to provide inotropic support and improve cardiac contractility*. In response to severe thrombocytopenia (14,000/µL) and the suspected SDH, a transfusion of four bags of thrombocyte concentrate was administered *to mitigate acute bleeding risk*.

Additional ICU interventions included Furosemide injections *to manage fluid overload secondary to capillary leakage, evidenced by the pleural effusion*, Omeprazole, and Tranexamic Acid. Supportive therapies included Metisoprinol as an immune-booster (though its specific efficacy in acute dengue is not established). As the patient's condition improved, inotropic support and oxygen were tapered.

Differential Diagnosis

The patient's presentation with fever, thrombocytopenia, plasma leakage, and altered consciousness required a broad differential. This included dengue-associated encephalopathy, dengue

Table 1. Summary of the patient's clinical progression.

Timeframe	Key Clinical Findings	Key Interventions
Day 1–2 (Home)	Intermittent fever (38.8°C), vomiting, diarrhea, abdominal pain, headache.	Oral intake.
Day 3 (ED Admission)	Conscious (*compos mentis*), T=39°C, HR=136 bpm (strong), petechiae. Platelets: 183,000/µL.	IV fluid resuscitation (KAEN 3B), antipyretics, antiemetics. Admitted.
Day 5 (ICU Transfer)	Decreased consciousness (Apathetic). Compensated shock (T=35.9°C, weak/thready pulse, cold extremities).	Oxygen therapy. Fluid escalation (D5 ½ NS). Dobutamine infusion initiated.
Day 5 (Critical Phase)	NS1 positive. Platelet nadir (14,000/µL). Elevated SGOT (117.81 U/L). Imaging: Pleural effusion & suspected SDH.	Platelet transfusion (4 bags). Furosemide.
Day 6–8 (ICU)	Gradual improvement in consciousness and hemodynamic status.	Weaning of dobutamine and oxygen.
Day 9 (Discharge)	Fully conscious (GCS 15), afebrile, hemodynamically stable. Laboratory results normalizing.	Discharged home.
2-Week Follow-up	Clinically well, normal cognitive and motor function.	No neurological sequelae.

Table 2. Summary of Key Laboratory Findings

Parameter	Day 3 (admission)	Day 5 (ICU)	Nadir/Peak	Normal Range
Platelet Count ($\times 10^3/\mu L$)	183	55	14 (Day 5)	150-450
SGOT (U/L)	N/A	117.81	117.81 (Day 5)	< 40
SGPT (U/L)	N/A	39.42	39.42 (Day 5)	< 40
NS1 Antigen	Not Done	Positive	N/A	Negative

encephalitis, other systemic infections involving the Central Nervous System (CNS) (e.g., bacterial sepsis, cerebral malaria), and non-dengue viral encephalitis. [6]

Dengue encephalopathy was strongly suspected given the positive NS1 test, profound thrombocytopenia, and decreased consciousness.[6,7,9] However, dengue encephalitis, involving direct viral invasion, remained a consideration. Due to the patient's critical condition and severe thrombocytopenia (nadir 14,000/µL), a lumbar puncture to obtain cerebrospinal fluid (CSF) was clinically contraindicated due to the high risk of iatrogenic bleeding. Therefore, the diagnosis of encephalopathy (a syndrome of neurological dysfunction) was favored over encephalitis (inflammation). Metabolic disturbances (e.g., hypoglycemia, hyponatremia) and hepatic dysfunction (evidenced by elevated SGOT) were also considered as primary drivers of the encephalopathy.

Outcomes and Follow-Up

The patient showed gradual clinical improvement following aggressive supportive therapy in the ICU. Her mental status progressively improved, and by day six of admission (Day 8 of illness), she was fully conscious (GCS 15), afebrile, and clinically stable. Platelet counts began to recover post-nadir.

The patient was discharged on day nine in good general condition. The head CT scan findings did not require neurosurgical intervention. At the *two-week post-discharge evaluation*, the patient had fully recovered, demonstrating *normal age-appropriate cognitive and motor function with no neurological sequelae*. Follow-up laboratory findings were within normal limits.

Ethical Considerations

Research ethics approval was obtained for this case report from the Hang Tuah University ethics committee (No. I/037/UHT.KEPK.03/VII/2025).

Patient confidentiality was strictly maintained. Written informed consent was obtained from the patient's mother for publication. This case report was prepared in accordance with the CARE (CAse REport) guidelines.

DISCUSSION

This case reports a rare and severe presentation of DHF in a 4-year-old child, characterized by the simultaneous occurrence of encephalopathy and suspected subdural hemorrhage. The patient's clinical course, marked by rapid neurological decline and

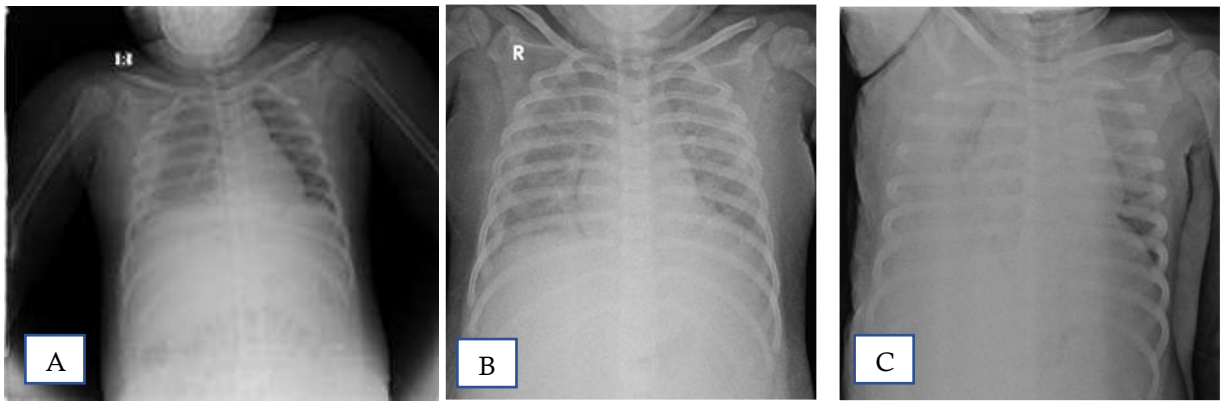


Fig.1A. AP Chest Radiograph (January 31st, 2025); **B and C.** AP/Lateral Chest Radiograph (February 1st, 2025)



Fig. 2. Head CT-scan examination showing a hyperdense lesion in the superior sagittal sinus supporting the diagnosis of dengue encephalopathy with possible subdural hemorrhage.

compensated shock, underscores the diagnostic and therapeutic challenges of atypical dengue.

The primary neurological challenge in this case was distinguishing encephalopathy from encephalitis [9,13]. Encephalopathy in DHF is often a secondary complication of the profound systemic insult [6,9,11]. This 'toxic' or metabolic encephalopathy can be driven by a 'cytokine storm,' leading to a breakdown of the blood-brain barrier, cerebral edema, or metabolic derangements such as hepatic dysfunction (seen in our patient with SGOT 117.81 U/L), electrolyte imbalances, and shock. The rapid and complete recovery of our patient's mental status following supportive care (fluid resuscitation, hemodynamic support) strongly suggests a metabolic or toxin-mediated pathogenesis rather than direct viral neuro-invasion (encephalitis), which might carry a worse prognosis.

The finding of a hyperdense lesion on CT, suspecting SDH, is a particularly severe complication. Intracranial hemorrhage in DHF is often linked to profound shock and severe thrombocytopenia. Our patient's platelet nadir of 14,000/µL placed her at extreme risk. This combination of findings highlights a dual pathology:

- Systemic capillary leakage and coagulopathy leading to hemorrhagic complications, and
- Systemic metabolic disturbance leading to encephalopathy.

Our case shares features with other reports of severe neurological dengue [11,14]. It also has unique aspects. Other studies have reported dengue encephalopathy in adults, highlighting the systemic inflammatory response and emphasizing the diagnostic ambiguity between encephalopathy and encephalitis, a key feature we faced. However, the co-occurrence of encephalopathy with suspected SDH in a young child, followed by a full recovery, is a crucial addition to the literature. This

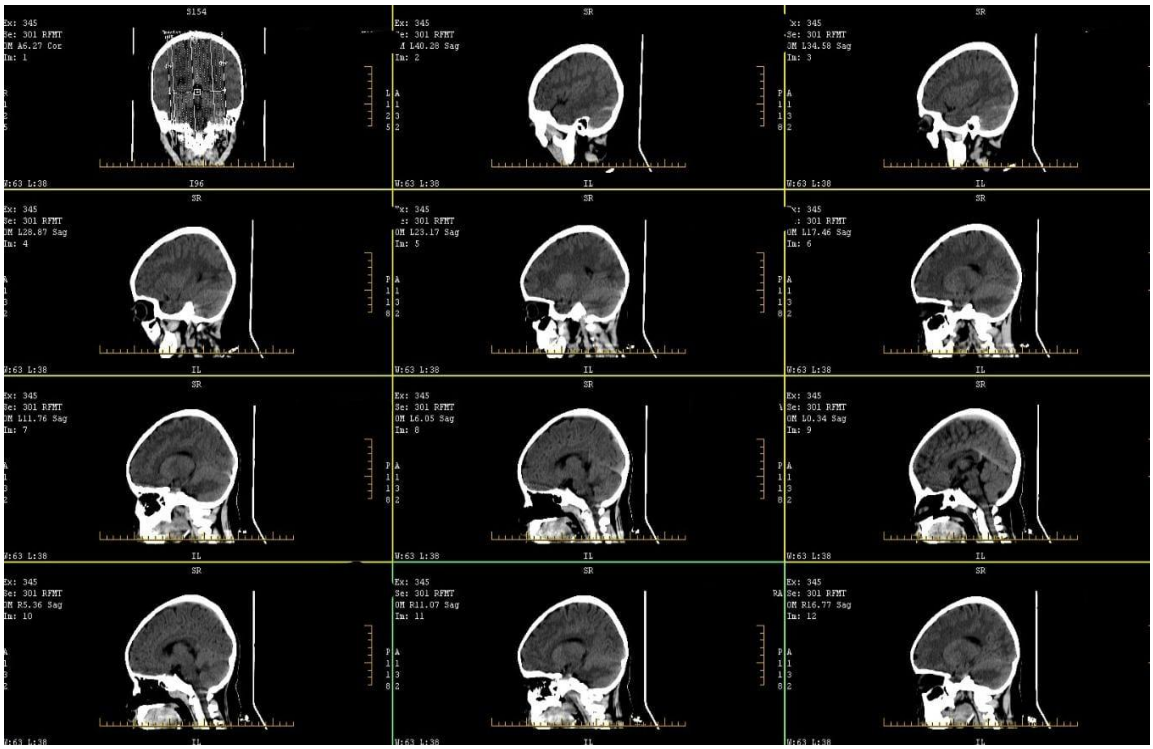


Fig. 3. Head CT-scan examination showing a hyperdense lesion in the bilateral subtentorial region, supporting the diagnosis of dengue encephalopathy with possible subdural hemorrhage**.**

successful outcome, despite the presence of obesity (a potential risk factor for severe dengue) [6,8,15], underscores the potential reversibility of dengue encephalopathy if shock and metabolic derangements are managed aggressively.

Study Limitations

A limitation of this report is the absence of CSF analysis, which could have definitively ruled out encephalitis. However, this procedure was clinically contraindicated by the patient's severe thrombocytopenia and high bleeding risk. Furthermore, DENV serotyping was not performed.

CONCLUSION

This case of pediatric DHF complicated by both encephalopathy and suspected subdural hemorrhage highlights the potential for severe, multifactorial neurological involvement in dengue infection.

The key clinical implications and take-home messages are:

1. High Index of Suspicion: Neurological symptoms (e.g., decreased consciousness, lethargy) in pediatric DHF must be treated as critical warning signs, prompting immediate neurological assessment, neuro-imaging, and consideration for intensive care.
2. Pathogenesis Matters: The pathogenesis of neurological symptoms is often metabolic (encephalopathy) rather than purely infective Fig. 1 AP Chest Radiograph (January 31st, 2025) (encephalitis). This reinforces that aggressive hemodynamic and metabolic supportive care is the cornerstone of management.
3. Multidisciplinary Approach: A multidisciplinary ICU team is essential for managing the complex interplay of plasma leakage, coagulopathy, and end-organ dysfunction.

Early recognition and prompt, comprehensive intervention can lead to excellent outcomes and prevent long-term neurological sequelae, even in critically ill children.

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CONFLICT OF INTEREST

The authors declare that there is no known conflict of interest that could influence the work reported in this paper.

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