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When Dengue Goes Neurological: Encephalopathy in An Adolescent Male

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ABSTRACT

Background: Neurological involvement in dengue virus infection has increased significantly in recent years. Dengue encephalopathy, the most frequent neurological manifestation of dengue infection, occurs in 0.5% to 21% of cases. Among pediatric patients, neurological complications are associated with a mortality rate of up to 10%. Early recognition and timely treatment are crucial to prevent severe outcomes and long-term sequelae. ***Objective:*** To highlight the importance of considering dengue infection as a potential cause of encephalopathy in febrile patients. ***Case:*** A 15-year-old male presented with altered consciousness, cold extremities, and a weak pulse. He had experienced a fever for three days prior to admission. His consciousness gradually improved following fluid resuscitation. Subsequently, he developed abdominal pain, nausea, vomiting with bloody mucus, and spontaneous gum bleeding. Physical examination revealed epigastric tenderness. Laboratory findings showed leukopenia, thrombocytopenia, elevated hematocrit, hypoalbuminemia, compensated metabolic acidosis, ketonuria, hematuria, and a normal head CT scan. He was diagnosed with dengue encephalopathy and managed with fluid resuscitation, anticonvulsants, antibiotics, Fresh Frozen Plasma (FFP) transfusion, and supportive treatment. The patient showed gradual improvement and fully recovered after seven days of hospitalization, four days in intensive care, and three days in a regular ward. ***Conclusion:*** Early identification of dengue encephalopathy is needed to enable prompt and appropriate treatment. Despite a severe clinical presentation, full recovery is possible with timely and comprehensive management.

Keywords: Dengue encephalopathy; adolescent; altered consciousness; neurological complication; dengue fever

BACKGROUND

Dengue is the second most common mosquito-borne disease affecting human beings after malaria, caused by the dengue virus (DENV). Aedes mosquitoes, namely *Aedes aegypti, Aedes albopictus, Aedes scutellaris*, and *Aedes polynesiensis*, are the recognized vectors for the transmission of dengue infection [1]. Approximately 50 million dengue infections occur worldwide annually. Dengue Hemorrhagic Fever (DHF) is more prevalent in children less than 15 years who live in hyperendemic areas that have recurrent dengue infection. An estimated 500,000 DHF patients require hospitalization each year, 90% of whom are children less than five years old, and about 2.5% of those affected die. The Ministry of Health reported 68,407 DHF cases with an Incidence Rate (IR) of 26.12 in 100,000 population in Indonesia. East Java is the province with the highest mortality of DHF in 2017, with 105 deaths. Mortality due to shock accompanied by severe gastrointestinal bleeding and encephalopathy remains high. Meanwhile, the prevalence of DHF with shock in various hospitals in Indonesia varies between 11.2–42%[2].

Dengue has a broad spectrum of clinical symptoms, ranging from mild dengue fever to severe dengue, with the classification of severe hemorrhage, severe plasma leakage, and severe organ involvement[3]. DENV1-4, along with St. Louis encephalitis virus, Japanese encephalitis, West Nile encephalitis virus, tick-borne encephalitis virus, and yellow fever, belongs to the family of Flaviviridae known to cause neurotropism. Dengue is considered non-neurotropic, but recent trends have shown DENV 2 and 3 to be frequently implicated in causing neurological manifestations [4].

High body temperature, elevated hematocrit, thrombocytopenia, skin rash, and liver dysfunction are independent risk factors for the neurological complications of DENV infection[1].

Encephalopathy is a general term for brain dysfunction with altered consciousness level[5],[6]. The incidence of these symptoms ranges from 0.5% to 21% of cases [3]. Various theories have been postulated for dengue encephalopathy: 1. Prolonged DHF with fluid extravasation and hyponatremia causing cerebral oedema. 2. Direct invasion of the virus into the central nervous system. 3. Autoimmune reactions and metabolic variations worsening neurological infirmity[4], [7], [8].

Here, we report a case of dengue encephalopathy in a 15-year-old male adolescent who initially presented with a 3-day history of fluctuating fever accompanied by headache, reduced appetite, nausea, and vomiting, before developing acute altered mental status with agitation and impaired consciousness. Subsequent clinical assessment and supportive investigations established the diagnosis, and the patient demonstrated steady neurological recovery with timely supportive management and close monitoring. This case report aims to emphasize the diagnostic challenge of recognizing neurological involvement in dengue when early manifestations are nonspecific and neuropsychiatric features emerge abruptly.

CASE

A 15-year-old male presented to the Emergency Department (ED) with reduced consciousness. The patient exhibited incoherent self-talking, disorganized speech, absent eye contact, and minimal interaction. Fever began 3 days prior to admission, fluctuating after antipyretic use, although temperature was not recorded at home. Along with the fever, the patient experienced headache, loss of appetite, nausea, and vomiting. Shortly before admission, he developed restlessness and a sensation of coldness. The patient had never experienced such symptoms before. There was no past medical history and no prior history of dengue infection.

The patient was born at term via spontaneous vaginal delivery, with immediate crying at birth. His birth weight was 3000 g, and his birth length was 48 cm. There was no history of allergies. He had complete immunization according to age, and developmental milestones were met appropriately.

Initial assessment in the ED revealed a Glasgow Coma Scale (GCS) score of E3V3M5, with blood pressure 154/75 mmHg, pulse 90/min, temperature 39.6°C, and respiratory rate 20/min. Anthropometric measurements showed a weight of 56 kg and height of 165 cm, consistent with good nutritional status. Physical examination revealed cool acral regions of both upper and lower extremities.

Complete blood count showed leukocytes 6.10 × 10^3/µL, hemoglobin 13.3 g/dL, hematocrit 39.7%, and platelets 80 × 10^3/µL. Random blood glucose was 96 mg/dL. Serum electrolytes were as follows: sodium 130 mmol/L, potassium 3.0 mmol/L, and chloride 97 mmol/L. Blood Gas Analysis (BGA) revealed pH 7.43, $PaCO_2$ 28 mmHg, PaO_2 84 mmHg, $cHCO_3^-$ 18 mmol/L, ABE −6 mmol/L, SBC 19 mmol/L, and SaO_2 97%. Serum albumin was 4.1 g/dL. Non-contrast head computed tomography showed no intracranial hemorrhage, infarction, or Space-occupying Lesion (SOL). Rhinitis with nasal septal deviation was noted.

The clinical presentation and diagnostic workup were consistent with dengue encephalopathy. Initial management included fluid resuscitation with Ringer's lactate at 10 mL/kg over 1 hour. Persistent acral coolness prompted a second RL bolus of 10 mL/kg over 30 minutes. Significant agitation was managed with a single dose of Intravenous (IV) diazepam 5 mg. Ongoing agitation prompted phenobarbital 200 mg twice daily. Empiric antibiotic therapy with cefotaxime 1 g three times daily IV was started. Symptomatic treatment included paracetamol 1,000 mg four times daily IV, ranitidine 25 mg twice daily IV, and ondansetron 4 mg twice daily IV. Intensive care monitoring was initiated, and a urinary catheter was placed.

By the second day of hospitalization, the patient had regained full alertness, although persistent generalized weakness was noted. Fever continued, with one episode of vomiting containing blood-tinged mucus. Reassessment showed GCS E4V5M6, blood pressure 99/57 mmHg, pulse 80/min, temperature 37.7°C, and respiratory rate 24/min. Extremities were warm, and complete blood count demonstrated leukocytes 4.17 × 10^3/µL, hemoglobin 15.6 g/dL, hematocrit 42.8%, and platelets 27 × 10^3/µL. Maintenance RL was continued based on an estimated daily fluid requirement of 2220 mL, adjusted for oral intake of 500 mL, and administered at 24 Drops per Minute (dpm). Phenobarbital was discontinued after the patient recovered consciousness, while other treatments were maintained.

On the third day of hospitalization, the patient was fully alert but continued to experience generalized weakness. Additionally, spontaneous gingival bleeding, epigastric pain, and dark, reddish, concentrated urine were reported. Vital signs remained stable with GCS E4V5M6, blood pressure 93/64 mmHg, pulse 75/min, temperature 36.7°C, and respiratory rate 24/min. Epigastric tenderness was noted on examination. Laboratory results showed leukocytes 3.30 × 10^3/µL, hemoglobin 16.4 g/dL, hematocrit 44.2%, and platelets 11 × 10^3/µL. Serum albumin had decreased to 3.3 g/dL. Urinalysis revealed cloudy yellow urine with ketone 1+, blood 3+, erythrocytes 25–30 per field, leukocytes 1–2 per field, squamous epithelial cells 2–4 per field, and bacteria present. Additional management included an RL bolus of 500 mL, followed by continued fluid infusion at 24 dpm. Fresh Frozen Plasma (FFP) transfusion was given as 1 unit every 24 hours for 3 doses, with premedication using furosemide (Lasix) 1 ampoule.

Cefotaxime and ranitidine were continued, and paracetamol was provided as needed for fever. Ondansetron was discontinued.

The patient exhibited ongoing weakness and epigastric pain and developed a diffuse pruritic erythematous rash that involved nearly the entire body on the fourth day of hospitalization. Clinical re-evaluation showed GCS E4V5M6, blood pressure 105/60 mmHg, pulse 82/min, temperature 36.7°C, and respiratory rate 22/min. Physical examination confirmed generalized urticaria and epigastric tenderness. Complete blood count showed leukocytes 5.12 × 10^3/µL, hemoglobin 15.4 g/dL, hematocrit 41.3%, and platelets 17 × 10^3/µL. Diphenhydramine was administered at one-half ampoule twice daily IV. Cefotaxime and ranitidine were continued, while paracetamol remained as needed for fever. The third planned FFP transfusion was withheld due to an allergic reaction. Transfer to the general ward was permitted.

By the fifth day of hospitalization, weakness, nausea, epigastric pain, and abdominal bloating persisted, although pruritus and erythema had improved. Vital signs remained stable with GCS E4V5M6, blood pressure 100/70 mmHg, pulse 88/min, temperature 36.1°C, and respiratory rate 22/min. Examination revealed warm extremities, reduced urticaria, and persistent epigastric tenderness. Complete blood count showed leukocytes 4.08 × 10^3/µL, hemoglobin 13.5 g/dL, hematocrit 36.4%, and platelets 23 × 10^3/µL. Diphenhydramine was used for recurrent allergic manifestations. Sucralfate 10 mL three times daily was added, and the urinary catheter was removed.

On the sixth day of hospitalization, the patient showed further improvement, with reduced weakness and diminished epigastric pain and abdominal bloating. Reassessment revealed stable vital signs, GCS E4V5M6, blood pressure 110/70 mmHg, pulse 86/min, temperature 36.5°C, and respiratory rate 21/min. Minimal epigastric tenderness remained. Complete blood count demonstrated leukocytes 4.84 × 10^3/µL, hemoglobin 12.9 g/dL, hematocrit 34.9%, and platelets 41 × 10^3/µL. Ongoing management was continued as before.

By the seventh day of hospitalization, the patient was completely asymptomatic. Vital signs were stable with GCS E4V5M6, blood pressure 100/70 mmHg, pulse 88/min, temperature 36.5°C, and respiratory rate 22/min. Physical examination was unremarkable. Complete blood count showed leukocytes 5.51 × 10^3/µL, hemoglobin 13.6 g/dL, hematocrit 37.3%, and platelets 130 × 10^3/µL. The patient was discharged.

DISSCUSION

The new classification of the World Health Organization classifies dengue infections into three categories, which is: (1) Dengue Fever Without Warning Signs, (2) Dengue Fever with warning signs, (3) Severe dengue. CNS involvement is one of several criteria of severe dengue fever[9].

Murthy divided the neurological involvement in dengue infection into three categories in his review of dengue-associated neurological complications. The first category was due to the neurotropism of the dengue virus, which results in encephalitis, meningitis, myelitis, myositis, and rhabdomyolysis. The second category included neurological complications due to systemic effects of dengue infection leading to encephalopathy, haemorrhagic or ischemic stroke, hypokalaemic paralysis, and papilledema. The third category comprised post-infectious immune-mediated complications, which included Guillain-Barré syndrome, ADEM, encephalomyelitis, optic neuritis, neuromyelitis optica, and other neuropathies[7].

The patient in this case was a 15-year-old male who presented with a febrile illness that began 3 days prior to admission. The fever was reported to fluctuate despite antipyretic administration, which aligns with the typical presentation of dengue fever. In addition to fever, the patient exhibited headache, anorexia, nausea, and vomiting, which are common systemic manifestations of dengue. However, the patient developed altered consciousness, incoherent speech, and absent eye contact, which prompted further evaluation and led to a diagnosis of dengue encephalopathy. These neurological signs are in line with the second category of neurological complications, which is due to systemic effects of the infection leading to encephalopathy.

Most cases of encephalopathy are found in DHF during or after the critical phase, but may occur in the febrile phase. Few cases are found among DF patients[9]. This patient, who was initially diagnosed with a febrile illness and then developed neurological symptoms, fits this atypical progression, where encephalopathy manifested even before entering the critical phase of dengue. Diagnosis of dengue encephalopathy is based on clinically diagnosed DHF according to the World Health Organization (WHO) criteria, with CNS manifestations including abrupt onset of hyperpyrexia, non-transient alteration of consciousness, headache, vomiting with or without seizures, and normal cerebrospinal fluid (CSF)[9].

Recent research suggests that in dengue virus infection, cytokine overproduction results in immune-mediated endothelial cell damage, which contributes to many of the CNS manifestations. Cytokines like IL-1β, TNF, IL6, IL8, IL10, enzymes like MMP2, and chemotactic proteins like IP2 and RANTES (regulated upon activation, normal T cell expressed, and secreted; also known as CCL5) play an upper hand in causing endothelial injury and dysfunction, leading to increased vascular permeability and fluid leakage. This leakage may result in generalized cerebral edema[1].

Burst suppression, electrographic seizures, focal patterns, or Epilepsia partialis continua may be observed on EEGs in these patients[1]. From CSF parameters, we could find a normal cell count or

pleocytosis, and a normal or high level of protein[9]. Suggestive of extensive involvement of the bilateral cerebellar region, brainstem, and thalamus along with peculiar rim enhancement, can be found in MRI. Encephalopathy caused by dengue fever can manifest as reduced sensitivity, cognitive impairment, convulsions, and personality and behavior disorders, such as acute mania, depression, emotional lability, anxiety, psychosis, and agoraphobia. Brain edema, anoxia, hemorrhage, intense hyponatremia, liver or kidney failure, release of toxic substances, metabolic acidosis, and direct organ invasion are commonly reported precursors of encephalopathy in patients with severe dengue[7], [9].

In this case, the patient's altered consciousness and incoherent speech were significant indicators of encephalopathy, which were confirmed with normal brain CT scans, ruling out other potential causes like intracranial hemorrhage, infarction, or space-occupying lesions. Additionally, the patient exhibited dark reddish concentrated urine, which could indicate renal involvement or dehydration, a common complication in severe dengue.

Encephalopathy in dengue can result from a range of factors, including cerebral edema, hyponatremia, and systemic hemorrhage. The patient's blood gas analysis revealed mild hyponatremia with sodium levels of 130 mmol/L, aligning with the theory that electrolyte imbalances contribute to CNS dysfunction in severe dengue. In this case, the patient had a progressive decrease in platelet count which dropped to $11 \times 10^3/\mu L$ by the third day of hospitalization, reflecting severe thrombocytopenia and confirming the severe nature of the disease.

Currently there is no specific treatment of dengue encephalopathy due to its unclear and complex pathogenesis. Treatment is based on emergency conditions presented in patients. Treatment in Intensive Care Unit (ICU) with a multidisciplinary team is required given the decreased level of consciousness, complication of airway, breathing, circulation (ABC) problems, the presence of comorbidities, and considerations of specific etiology [9].

First and foremost, clinicians must maintain adequate airway and oxygenation with oxygen therapy. Intubation may be needed in patients with respiratory failure or semi-coma/coma. Secondly, intracranial pressure (ICP) increase should be prevented by: (1) Minimalize IV fluid to maintain adequate intravascular volume, ideally the total IV fluid should not exceed 80% maintenance; (2) Switch to colloidal solution earlier if hematocrit continues to rise or a large volume of IV is needed in cases with severe plasma leakage; (3) Administer diuretic if indicated in cases with signs and symptoms of fluid overload; and (4) Consider the use of steroids to reduce ICP, dexamethasone 0.5 mg/kg/day intravenously every 6- 8 hours is recommended [9]. The immune-mediated manifestations generally respond well to immunomodulators like high doses of corticosteroids or intravenous immunoglobulin therapy. There is no proven value of antiviral agents [1].

The patient's recovery followed a supportive management protocol that included fluid resuscitation with Ringer's lactate (RL), which is a key component of managing severe dengue with encephalopathy. The patient required two boluses of RL, totaling 20 mL/kg due to persistent cool extremities, which indicated compromised circulation and shock.

Due to involvement of hepatic failure in dengue encephalopathy, clinicians should decrease ammonia production with the use of lactulose 5-10 ml every 6 hours for induction of osmotic diarrhea. Local or systemic antibiotics should be administered to eliminate bowel flora. Avoid unnecessary drugs because most drugs have to be metabolized by the liver [9].

Laboratory parameters should be kept in normal ranges. Blood sugar should be maintained. If hypoglycemia occurs, glucose infusion should be given at infusion rate between 4-6 mg/kg/hour. The presence of acid-base and electrolyte imbalance such as hypo/hypernatremia, hypo/hyperkalemia, hypocalcemia and acidosis could be found as one of the causes of encephalopathy. Thus, must be corrected promptly [9].

Since intracranial hemorrhage could be the cause of encephalopathy in severe dengue, intravenous Vitamin K1 could be given as prophylaxis. The dosage is 3 mg for <1 years old, 5 mg for <5 years old and 10 mg for >5 years old and adults. Platelets and fresh frozen plasma are quite routinely used as prophylaxis. However, numerous studies showed that there was no significant decrease of bleeding incidence between dengue patients who were given platelets or fresh frozen plasma and those who were not. Moreover, transfusion of blood products increases the risk of fluid overload and increased ICP in dengue patients. Therefore, transfuse blood only in hemorrhagic conditions and with caution, preferably packed red cells [9].

Other treatments such as anticonvulsants for control of seizures, H2-blockers or proton pump inhibitors may be given as indicated. Clinicians should consider plasmapheresis or hemodialysis or renal replacement therapy in cases of clinical deterioration. The use of renal replacement such as continuous veno-venous hemodialysis is important in managing fluid states in the recovery phase of dengue hemorrhagic fever in those with renal impairment [9].

Besides altered consciousness, the patient also had an episode of vomiting containing blood mucus, suggesting gastrointestinal involvement, a well-known complication of severe dengue. The patient was also developed spontaneous gingival bleeding, epigastric pain, and dark reddish concentrated urine, further indicating the severity of the disease and multi-organ involvement.

These findings support the theory that severe dengue with encephalopathy can lead to significant systemic disturbances that require intensive monitoring and supportive care.

The patient showed improvement, suggest that early and aggressive management including fluid therapy and supportive care, can result in favourable outcomes. By the seventh day of hospitalization, the patient was asymptomatic, with stable vital signs and no significant abnormalities in his laboratory parameters, confirming a positive outcome.

Treatment outcome for patients with dengue encephalopathy is variable and depend on causal/precipitating factors as well as on the extent of medical care[1]. Several prognostic factors were reported to be correlated with the outcome of severe dengue, such as platelet count less than 100,000 cells/mm^3, serum albumin <35 g/L, Alanine aminotransferase (ALT) > 400 U/L, and total bilirubin >17 µmol/L [11]. In Indonesian children, prognostic factors of severe dengue were overweight/obesity, vomiting, hepatomegaly, and prolonged activated partial thromboplastin time (APTT)[12]. Study in India showed that mortality was observed in 10% cases of neurological complications due to dengue fever in pediatric patients [13].

CONCLUSION

We should consider dengue infection as a potential cause of encephalopathy in febrile patients. Early identification of dengue encephalopathy needed to enable prompt and appropriate treatment. Although the initial presentation may be severe, complete neurological recovery can be achieved with timely and comprehensive treatment.

Conflict of Interest

The author declares that there is no conflict of interest related to the publication of this research article.

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Ethics

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

REFFERENCES

[1] Trivedi, S., & Chakravarty, A. (2022). Neurological complications of dengue fever. Current Neurology and Neuroscience Reports, 22(8), 515–529. https://doi.org/10.1007/s11910-022-01213-7

[2] Fadilla, A. N., Husada, D., Utomo, B., & Penelitian, A. (2020). Epidemiology of children with severe dengue infection in Dr. Soetomo General Hospital (Gambaran epidemiologi anak dengan infeksi dengue berat di RSUD Dr. Soetomo). Journal of the Indonesian Medical Association, 70(4), 41–48.

[3] Leng, X., et al. (2024). Dengue encephalopathy in an adult due to dengue virus type 1 infection. BMC Infectious Diseases, 24(1), Article 280. https://doi.org/10.1186/s12879-024-09198-z

[4] Khosla, S., Chauhan, R., Aggarwal, A., & Patel, N. B. (2024). Dengue encephalitis – An unusual case series. Journal of Family Medicine and Primary Care, 13(8), 3420–3423. https://doi.org/10.4103/jfmpc.jfmpc_413_24

[5] Durkin, S. M., Da Silva, A. L., Davies, N. W. S., & Sriskandan, S. (2023). Dengue encephalopathy or dengue encephalitis? You decide. Open Forum Infectious Diseases, 10(10), ofad490. https://doi.org/10.1093/ofid/ofad490

[6] Pitton Rissardo, J., Byroju, V. V., & Fornari Caprara, A. L. (2024). A scoping review of neurological manifestations of dengue virus infection besides cerebrovascular disorders [Preprint]. MDPI. https://doi.org/10.20944/preprints202412.0936.v1

[7] Rangankar, V., Kumar, D., Kuber, R., & Kalekar, T. (2022). Imaging of the neurological manifestations of dengue: A case series. SA Journal of Radiology, 26(1), 2078. https://doi.org/10.4102/sajr.v26i1.2078

[8] Guzman, M. G., & Martinez, E. (2024). Central and peripheral nervous system manifestations associated with dengue illness. Viruses, 16(9), 1367. https://doi.org/10.3390/v16091367

[9] Setiorizaldi, I., Tedjaningrum, A. A., Panggabean, C. G., Nangin, E., Mahardhika, J. C., & Daryanani, C. P. (2022). Pediatric dengue encephalopathy: A review. Medical Clinical Update, 1(1), 5–7. https://doi.org/10.58376/mcu.v1i1.5

[10] Naderian, R., et al. (2025). Pathophysiology and clinical implications of dengue-associated neurological disorders. Frontiers in Microbiology, 16, 1536955. https://doi.org/10.3389/fmicb.2025.1536955

[11] Huy, B. V., & Toàn, N. V. (2022). Prognostic indicators associated with progresses of severe dengue. PLoS ONE, 17(1), e0262096. https://doi.org/10.1371/journal.pone.0262096

[12] Baiduri, S., Husada, D., Puspitasari, D., Kartina, L., & Basuki, P. S. (2020). Prognostic factors of severe dengue infections in children. Indonesian Journal of Tropical and Infectious Disease, 8(1), 43–54. https://doi.org/10.20474/ijtid.v8i1.10721

[13] Shokeen, P., Yadav, S., Verma, C. R., & Masand, R. (2018). Neurological complications in Dengue fever. International Journal of Contemporary Pediatrics, 5(3), 983–987. https://doi.org/10.18203/2349-3291.ijcp20181526