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Case report

# Meningoencephalitis probably associated with dengue infection in an 84-year-old patient: A case report

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ABSTRACT

Dengue meningoencephalitis is a rare neurological complication of dengue infection, characterized by inflammation of the brain and leptomeninges. We present the case of an 81-year-old female patient with a five-day history of fever, accompanied by altered consciousness, seizures, and meningeal signs. Dengue infection was confirmed by a positive RT-PCR result in blood, and cerebrospinal fluid analysis revealed lymphocytic pleocytosis, suggestive of a viral infectious process. Additionally, an electroencephalogram showed focal epileptiform activity. This is the first reported case of dengue meningoencephalitis in Central America. This case highlights the importance of considering dengue virus as a potential etiological agent in patients presenting with fever and acute alterations in consciousness secondary to a viral infectious process. The publication of this case is relevant to enhance early recognition of rare neurological complications such as dengue meningoencephalitis, promoting timely intervention that may positively influence patient prognosis.

## Introduction

Dengue is an acute febrile illness caused by an arbovirus known as DENV, belonging to the *Flaviviridae* family and the *Flavivirus* genus. It is an enveloped, single-stranded RNA virus [1,2] with structural proteins—Envelope, Membrane, and Capsid—as well as non-structural proteins: NS1, NS2A, NS2B, NS3, NS4A, NS4B, and NS5 [3]. There are four serotypes of the virus, DENV-1 to DENV-4 [1,3]. Infection with one serotype provides permanent homotypic immunity, whereas heterotypic immunity against other serotypes is transient, lasting less than a year. A secondary infection with a different serotype increases the risk of severe complications, including neurological manifestations [1,2,4].

Historically, dengue has been documented for centuries. The first probable case of dengue symptoms was described in 992 AD in a Chinese medical encyclopedia from the Jin dynasty [1,5]. Currently, dengue virus infects approximately 390 million people annually across more than 100 countries [1]. It is endemic in Southeast Asia, the Pacific, Eastern and Western Africa, the Caribbean, and the Americas [1]. In Honduras, as of epidemiological week 42 of 2024, 167,808 suspected dengue cases have been reported, of which 2128 have been classified as severe dengue [6].

Neurological complications associated with dengue virus infection are uncommon. The first reported association between dengue infection and neurological abnormalities was described in 1976 by Sanguansermsri et al. in a patient with encephalopathy [5,7,8]. Currently, neurological complications due to dengue infection are estimated to occur in 0.5–20 % of cases [4,9].

Dengue infection may be asymptomatic or present with a wide range of clinical manifestations. The World Health Organization (WHO) classification of dengue severity, established in 2009, includes: dengue without warning signs, dengue with warning signs, and severe dengue [6]. Severe dengue includes central nervous system impairment [1,3,6]. Neurological complications of DENV infection can be categorized into three groups based on pathogenesis: those caused by metabolic disturbances (encephalopathy), those resulting from direct viral invasion—including encephalitis, meningitis, myositis, and myelitis—and those caused by autoimmune responses, such as acute disseminated encephalomyelitis, optic neuritis, myelitis, and Guillain-Barré syndrome [5,9].

Encephalitis is one of the neurological complications of dengue infection, characterized by seizures, altered consciousness, headache, and vomiting [2,5,7,9,10]. This report presents the first documented case of dengue meningoencephalitis in Central America. In our literature review, no other cases were found; however, three cases of encephalitis were reported in a case series from Guatemala [11]. The significance of this report lies in the widespread distribution and high incidence of dengue infection in the Americas. Given this epidemiological context, dengue virus should be considered a potential etiological agent in

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patients presenting with neurological alterations due to viral infections. Early recognition will facilitate appropriate therapeutic and supportive management.

## Case presentation

We present the case of an 84-year-old female patient with a medical history of hypertension and chronic obstructive pulmonary disease, with good adherence to treatment. She sought medical attention on the fifth day of illness after experiencing continuous fever for the first four days, accompanied by severe headache and nausea. Over the last 24 hours, she remained afebrile but developed altered consciousness, characterized by disorientation. Initially, she was evaluated at the *Santo Hermano Pedro Betancourt Hospital*, where she received outpatient management. However, due to persistent symptoms, she was brought to the emergency department at *Centro Medico El Buen Samaritano*.

Upon arrival, her vital signs were as follows: blood pressure 150/80 mmHg, heart rate 77 beats per minute (bpm), respiratory rate 17 breaths per minute, and temperature 36.8°C. Her Glasgow Coma Scale (GCS) score was 14/15, with eye response: 4, verbal response: 4 (disoriented in time, place, and person), and motor response: 6, following commands. Pupils were isocoric and reactive to light. Muscle strength was preserved in all four extremities, but she exhibited nuchal rigidity with positive Brudzinski and Kernig signs. Cardiac examination revealed an irregularly irregular rhythm. Pulmonary auscultation detected mild expiratory wheezing in all lung fields.

Laboratory findings included: white blood cell count of 5000/μL (neutrophils: 3000/μL, lymphocytes: 1700/μL), hemoglobin 11.6 g/dL, hematocrit 31.5 %, and platelet count of 68,000/μL. HIV testing was negative (Table 1). Dengue RT-PCR in serum was positive. Cerebrospinal fluid (CSF) analysis revealed increased cellularity (87 cells/μL), predominantly lymphocytic (93 % lymphocytes, 7 % neutrophils), with a CSF glucose level of 77 mg/dL and blood glucose of 125 mg/dL, yielding a CSF-to-blood glucose ratio of 0.61 (Table 2). Contrast-enhanced brain computed tomography showed no ischemic or hemorrhagic lesions but revealed moderate cortical volume loss in the bifronto-temporal region (Fig. 1).

**Table 1**
Laboratory test results.

| Variable | Result | Normal Adult Range |
|---|---|---|
| **White blood cells** | 5000 | 5000–10,000 |
| **Neutrophils** | 3800 | 2500–7500 |
| **Lymphocytes** | 800 | 1300–4000 |
| **Hemoglobin** | 12.8 g/dl | 12.0–17.4 g/dl |
| **Hematocrit** | 33.6 % | 36–52 % |
| **Platelets** | 68,000 | 150,000–500,000 |
| **Random glucose** | 128 mg/dl | Menor 140 mg/dl |
| **Creatinine** | 0.75 | 0.50–0.90 mg/dl |
| **Blood urea nitrogen (BUN)** | 18 mg/dl | 7.0–20 mg/dl |
| **Glutamic Oxaloacetic Transaminase (TGO)** | 13.9 UI/ml | 4.0–37.0 UI/ml |
| **Glutamic Pyruvic Transaminase (TGP)** | 12.5 UI/ml | 4.0–42.0 UI/ml |
| **Sodium** | 134.2 mEq/L | 135–150 mEq/L |
| **Potassium** | 3.32 mEq/L | 3.5–5.5 mEq/L |
| **Calcium** | 9.15 mEq/L | 8.6–10.0 mEq/L |
| **Albumin** | 3.81 mg/dl | 3.5–5.0 mg/dl |
| **Serum ammonium** | 30.25 Umol/l | 10–47 Umol/l |
| **Glycosylated Hemoglobin** | 4.92 % | 4.5–6.5 % |
| **Human Immunodeficiency Virus Serology (VIH)** | Negative | |
| **Procalcitonin** | < 0.10 ng/ml | < 0.5 ng/ml |
| **Thyroid stimulating hormone (TSH)** | 0.4 μU/ml | 0.34–5.6 μU/ml |
| **Fibrinogen** | 364.1 mg/dl | 200–500 mg/dl |
| **Prothrombin time** | 14.3 seg | 10.0–14.0 seg |
| **Partial thromboplastin time** | 29.54 seg | 22.0–34.0 seg |

**Table 2**
Cerebrospinal fluid examination results.

| Variable | Result | Normal adult range |
|---|---|---|
| **Leukocytes** | 87 cel/ul | 0–5 cel/ul |
| **Neutrophils** | 7 % | |
| **Lymphocytes** | 93 % | |
| **Glucose** | 77 mg/dl | 50–80 mg/dl |
| **Proteins** | 50 mg/dl | 15–45 mg/dl |
| **Potassium Hydroxide (KOH)** | No fungal structures are observed. | |
| **India Ink** | Negative for fungal structures | |
| **Adenosine Deaminase (ADA)** | 12.93 UI/L | Hasta 50 UI/L |
| **Ziehl Neelsen** | No BAAR observed in 100 fields | |
| **Molecular Test for MTB/RIF (Gen Xpert)** | Not detected | |
| **Gram Staining** | No bacteria observed | |
| **Bacterial culture** | No bacterial growth | |

On the second day of hospitalization, the patient experienced a generalized tonic-clonic seizure lasting one minute. An electroencephalogram (EEG) was performed, revealing frequent bilateral fronto-temporal epileptiform activity (Fig. 2). Supportive management was provided, including intravenous crystalloid hydration following the Pan American Health Organization guidelines, acetaminophen, aerosolized bronchodilators, and intravenous levetiracetam 500 mg every 12 hours, with no further seizure episodes.

After seven days of hospitalization, the patient was discharged. As a neurological sequela, she persisted with disorientation in time and place but was able to recognize individuals.

## Discussion

Neurological complications of dengue are uncommon [4,9,12]. These may manifest as encephalitis when there is inflammation of the brain parenchyma [12,13], meningitis when the leptomeninges are affected [13,14], and meningoencephalitis when both the meninges and brain parenchyma are involved [12,13]. Clinically, these conditions present with altered consciousness, headache, vomiting, nuchal rigidity, and seizures [2,9], consistent with the presentation of our patient, who developed fever, headache, neurological impairment, meningeal signs, and seizures during hospitalization. The case was managed as an acute altered consciousness syndrome secondary to meningoencephalitis.

The diagnosis of dengue infection in this case was based on the chronological progression of symptoms and the epidemiological context. During the initial phase of the disease, the virus is present in the bloodstream, making direct viral detection tests, such as RT-PCR, the preferred diagnostic method. In this case, RT-PCR in serum was positive. Given that the patient was in the first five days of illness—the viremic phase—molecular testing via RT-PCR was appropriate for confirming the infection. This test has been reported to have a sensitivity between 93 % and 100 % and a specificity of 100 % [5,9,15,16]. The confirmation of dengue infection via RT-PCR, combined with clinical findings, allowed for an accurate diagnosis.

Cerebrospinal fluid (CSF) analysis is essential in the evaluation of patients with acute altered consciousness syndrome. In this case, CSF studies revealed lymphocytic pleocytosis (87 cells/μL, with 93 % lymphocytes and 7 % neutrophils), mild hyperproteinorrachia, and normal CSF glucose levels (77 mg/dL), findings consistent with an acute viral infectious process and in agreement with reported data on dengue encephalitis and meningitis cases [5,16]. Additional CSF studies ruled out other potential causes of acute neurological deterioration. RT-PCR for dengue virus in CSF was negative; however, it is important to note that CSF RT-PCR has lower sensitivity compared to serum RT-PCR due to lower viral loads in the CSF [9,10,12].

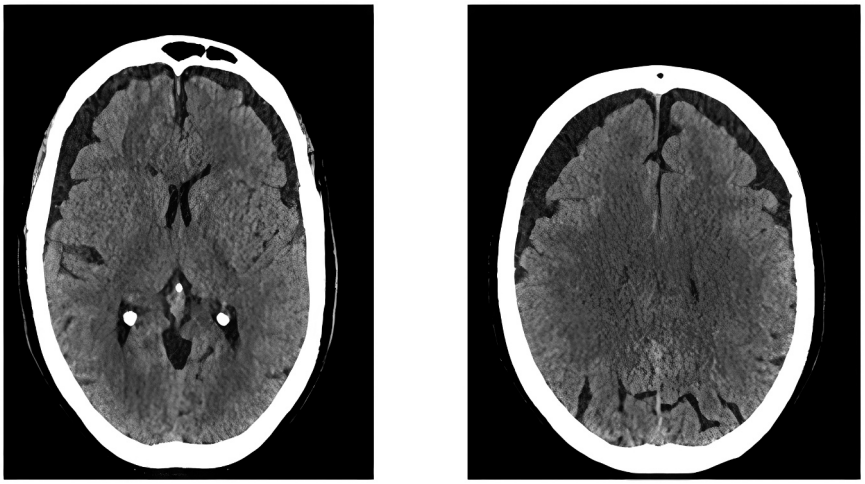

**Fig. 1.** The tomography. Contrast-enhanced brain computed tomography showing no ischemic or hemorrhagic lesions, with moderate volume loss in the bifronto-temporal cortex.

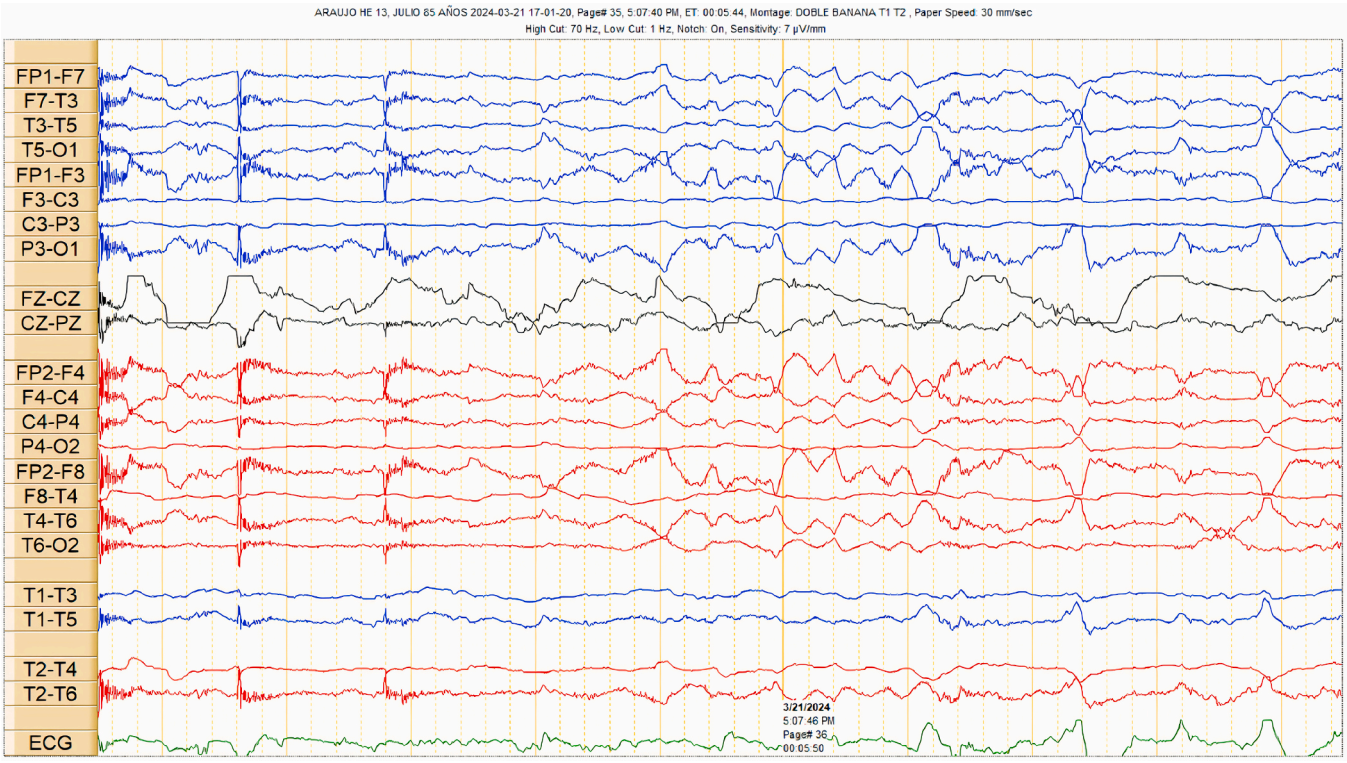


**Fig. 2.** The electroencephalogram. Digital electroencephalogram showing an electroencephalographic pattern consistent with a state of altered consciousness, characterized by a slow, asynchronous, asymmetric, and low-amplitude background rhythm (encephalopathy), along with frequent bilateral frontotemporal epileptiform activity.

Contrast-enhanced brain computed tomography (CT) showed no characteristic findings of encephalitis. However, literature reports indicate that both CT and magnetic resonance imaging (MRI) may be normal in cases of dengue meningitis and encephalitis [10,17]. Additionally, the electroencephalogram (EEG) in this case demonstrated encephalopathic dysfunction with frequent epileptiform activity, supporting brain involvement. These findings align with previously described EEG abnormalities in patients with dengue encephalitis [5,7,17].

The diagnosis of dengue meningoencephalitis in this case was established through the integration of clinical and laboratory findings, including meningeal signs, altered mental status, and seizures, along with a positive RT-PCR result for dengue virus in serum. CSF analysis was compatible with a viral process, and EEG findings demonstrated encephalopathic dysfunction with epileptiform activity, supporting brain involvement. This case met the diagnostic criteria outlined by the 2013 International Encephalitis Consortium, which include acute mental status alteration as a major criterion, accompanied by minor criteria such as fever, seizures, CSF pleocytosis, and EEG abnormalities [18]. Furthermore, the case fulfilled the WHO guidelines for diagnosing dengue-associated neurological disease, which require altered consciousness with a Glasgow Coma Score below 14, seizures, CSF lymphocytic pleocytosis, and virus detection in serum or CSF [5,7,10,12,13]. CSF studies ruled out other infectious causes of altered mental

status, consolidating the diagnosis of dengue meningoencephalitis.

Dengue encephalitis has a variable prognosis; while most cases resolve favorably, some patients may experience persistent neurological symptoms [6,14]. In this case, although the patient regained the ability to recognize individuals, she remained disoriented in time and place at discharge, significantly impacting her quality of life and leading to increased dependence on family caregivers due to impaired communication. This outcome underscores the importance of early identification of acute altered mental status and timely initiation of specific treatment and supportive care to improve recovery and minimize long-term sequelae.

In endemic regions, neurological complications of dengue should be considered in patients presenting with neurological deterioration, seizures, and meningeal signs during the acute phase of illness. In this case, early diagnosis through RT-PCR in serum, supportive CSF findings, and EEG abnormalities enabled the identification of a rare complication: meningoencephalitis. The prognosis varies, with most patients recovering; however, some may develop neurological sequelae such as disorientation and seizures. This report contributes to the recognition of these manifestations and their clinical implications.

A limitation of this case was the unavailability of IgM and IgG antibody testing for dengue virus in CSF due to laboratory constraints and financial restrictions that prevented testing at a private facility. Additionally, brain MRI, which has higher sensitivity than contrast-enhanced CT for detecting subtle early lesions associated with encephalitis, was not performed.

## Author Statement

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## CRediT authorship contribution statement

**Carlos Mejia Irias:** Writing – review & editing, Writing – original draft, Visualization, Validation, Resources, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization.

## Consent

“Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request”.

## Ethical approval

The study is a case report, only information from the patient́s file was used, no type of intervention was performed with the patient, so it does not have approval from the ethics committee.

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## Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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