Arch Argent Pediatr. 2026;124(5):e202611082

Congenital dengue with detection of viral RNA in cerebrospinal fluid: A case report

Felipe Cabezón Jarvis[1], *Valentina Medrano Paz*[1], *Mariana Rodríguez Braumüller*[2]

ABSTRACT

Dengue virus infection is the most common arbovirus disease worldwide, and its spread has increased the relevance of less common forms, such as vertical transmission. Congenital dengue poses a diagnostic challenge due to its nonspecific presentation and overlap with late-onset sepsis. Transmission occurs during maternal viremia, especially when the infection occurs in the days leading up to delivery, and may present with fever and hematological abnormalities.

We present the case of a 35-week preterm newborn, the daughter of a mother with confirmed dengue before delivery, who developed a fever and hyporesponsiveness on the seventh day. Detection of viral RNA via polymerase chain reaction in serum and cerebrospinal fluid confirmed the infection. She developed thrombocytopenia and coagulation abnormalities, with normal neurological findings and a favorable clinical course.

This case highlights the importance of considering congenital dengue in neonatal sepsis when appropriate clinical settings are present.

Keywords: *congenital dengue; neonatal sepsis; vertical transmission of infectious disease; newborn.*

doi: http://dx.doi.org/10.5546/aap.2026-11082.eng

To cite: Cabezón Jarvis F, Medrano Paz V, Rodríguez Braumüller M. Congenital dengue with detection of viral RNA in cerebrospinal fluid: A case report. *Arch Argent Pediatr.* 2026;124(5):e202611082.

[1] *Department of Pediatrics;* [2] *Department of Neonatology; Hospital Británico de Buenos Aires, Autonomous City of Buenos Aires, Argentina.*

Correspondence to *Felipe Cabezón Jarvis: fcabezonjarvis@gmail.com*

Funding*: None.*

Conflict of interest: *None.*

Received*: 3-5-2026*
Accepted*: 4-6-2026*

This is an open access article under the Creative Commons Attribution–Noncommercial–Noderivatives license 4.0 International. Attribution - Allows reusers to copy and distribute the material in any medium or format so long as attribution is given to the creator. Noncommercial – Only noncommercial uses of the work are permitted. Noderivatives - No derivatives or adaptations of the work are permitted.

INTRODUCTION

Dengue virus infection is a disease transmitted primarily through the bite of female mosquitoes of the genus *Aedes* (*Aedes aegypti* and *Aedes albopictus*); in rare cases, transmission via blood transfusion and vertical transmission during pregnancy have also been reported.[1] It is caused by a virus of the genus *Flavivirus*, family *Flaviviridae*, which comprises four antigenically distinct serotypes (DENV-1 to DENV-4).[2]

With widespread distribution in tropical and subtropical regions,[3,4] it is the most common arbovirus disease worldwide. The annual incidence is estimated at nearly 390 million infections, of which approximately 96 million are symptomatic.[3] In recent decades, its spread has intensified steadily,[4] a phenomenon attributed to climate change, unplanned urbanization, limitations in vector control, and increased population mobility.[4]

Clinically, it is a dynamic condition and, in most cases, self-limiting. It begins with a febrile period that may be accompanied by abdominal pain, vomiting, diarrhea, rash, headache, joint pain, and muscle pain. It can present asymptomatically or with few symptoms in all age groups, including pregnant women. In this group, the infection has been associated with an increased risk of preterm birth, low birth weight for gestational age, spontaneous abortion, and stillbirth.[5-10]

Vertical transmission has been documented,[6-9] especially when maternal infection occurs in the early stages of pregnancy or within 10–15 days before delivery.[6] Transplacental passage occurs during the period of maternal viremia, even in asymptomatic women.[6-9] Congenital dengue occurs when maternal infection occurs in the days before delivery (within 10–15 days), leaving no time for the transfer of protective antibodies.[6] In endemic areas, it should be suspected in newborns with fever, maculopapular rash, and thrombocytopenia, particularly if there is a compatible maternal history.[7-8,11] In these cases, both mother and child should be evaluated simultaneously using antigen detection and serological tests.[7,8]

Neonatal complications include low birth weight, hematological abnormalities such as thrombocytopenia and coagulopathy, and neurological involvement.[7,8] The involvement of the central nervous system is a potentially serious complication.[12,13] The virus exhibits neural tropism and can cross the blood-brain barrier, replicate in the brain parenchyma, and cause neuronal damage.[12,13]

Given this epidemiological situation, it is essential to consider congenital dengue in the neonatal differential diagnosis.

CLINICAL CASE

We present the case of a female preterm newborn at 35 weeks' gestation, with a birth weight of 2540 g, born to a 31-year-old primiparous mother. The pregnancy had proceeded without complications until week 34, at which point the mother developed a fever accompanied by an erythematous rash, pruritus, and low back pain. Following clinical evaluation and laboratory tests, the diagnosis of dengue was confirmed. Four days after the onset of symptoms, she presented with premature rupture of membranes, persistent fever, and hypokalemia. In this context, and given the suspicion of chorioamnionitis, an emergency cesarean section was performed.

At birth, the patient had an Apgar score of 8/9 and respiratory distress that required continuous positive airway pressure (CPAP) during delivery; she did not require supplemental oxygen thereafter. She was admitted to the neonatal intensive care unit, where laboratory tests and blood cultures were performed. Empirical treatment with ampicillin and gentamicin was initiated but discontinued after 48 hours due to negative cultures and favorable clinical progress.

On the seventh day of life, the infant developed a fever, mild transient hyporesponsiveness, pallor, and generalized reticulations. There was no irritability or impaired sucking, and the fontanelle was normal. Given the suspicion of late-onset neonatal sepsis, blood cultures, a urine culture, and a lumbar puncture were performed.

The cerebrospinal fluid was clear, xanthochromic, with 1 leukocyte, glucose of 51 mg/dL, protein of 0.79 g/L, and chloride of 127 mEq/L; no bacterial growth was observed. Given the maternal history, virological testing was expanded to include serum and cerebrospinal fluid, and dengue virus infection was confirmed by polymerase chain reaction in both samples. Empiric intravenous antibiotic therapy with meropenem and vancomycin was initiated but discontinued after 72 hours due to a negative bacterial culture. The patient did not receive glucocorticoids and responded well, with clinical improvement within 24-48 hours and no signs of neurological involvement during hospitalization.

On the ninth day of life, laboratory tests revealed abnormalities in the coagulation profile.

The patient received intravenous vitamin K for 3 days, with a gradual return to normal levels. At 11 days, the patient presented with thrombocytopenia but did not require a transfusion, as there was no active bleeding. A slight decrease in white blood cell count was observed, without leukopenia, and a mild increase in hematocrit. The serial changes in hematological and coagulation parameters are detailed in *Table 1*.

Additional tests were performed, including funduscopy, brain ultrasound, echocardiogram, brain MRI, and electroencephalogram, all of which were normal. Given the favorable clinical course and the resolution of the laboratory abnormalities, the patient was discharged from the hospital at 19 days of age. Neurological follow-up was conducted until 6 months of age, with neurological monitoring in accordance with age-appropriate neurodevelopmental guidelines.

DISCUSSION

In the current epidemiological context of sustained dengue spread,[14] vertical transmission takes on clinical significance that outweighs its low frequency. Although congenital dengue has been documented in various series,[7-9] it remains underdiagnosed, partly due to its nonspecific presentation and overlap with symptoms of neonatal sepsis, which leads to delays in etiological confirmation. In this context, it is worth comparing this case with a national report on perinatal dengue, which describes newborns with nonspecific symptoms—such as fever and changes in skin color—that mimic neonatal sepsis. In both scenarios, the mother's medical history during the peripartum period is a key factor in guiding the clinical suspicion.[11]

The risk of transmission increases when maternal infection occurs close to delivery, preventing adequate antibody transfer. IgG transfer begins in the second trimester and peaks in the third;[15] transmission is more common when infection occurs within 10-15 days before delivery and is rare if it occurs weeks or months earlier.[15] In the case presented, the proximity between maternal infection and birth reinforces this pathophysiological mechanism.[15]

Acute infection during pregnancy has been associated with an increased risk of spontaneous abortion and stillbirth, particularly in severe cases.[5] In this case, the timeline between maternal infection and the onset of symptoms on the seventh day of life is consistent with the intervals described in the literature,[6,7] supporting transplacental transmission during viremia.

From a clinical standpoint, neonatal case series consistently describe fever, thrombocytopenia, and leukopenia as the predominant findings.[8,9] A key pathophysiological aspect is that, unlike what is observed in older children, hemoconcentration is not a reliable parameter in the neonatal period due to physiological polycythemia.[8] In the presented patient, thrombocytopenia and coagulation abnormalities were the key developmental markers, whereas hematocrit proved to be of little use as an indicator of severity. This highlights the need to adapt the monitoring criteria used in other age groups to the neonatal setting, avoiding extrapolations that could lead to misinterpretations of risk.

Although rare, central nervous system involvement is recognized in dengue infection.[12,13] Encephalopathy has been documented in approximately 0.5% of cases in prospective cohorts.[12] The virus exhibits neural tropism and can cross the blood-brain barrier via inflammatory

Table 1. Changes in laboratory parameters

Parameter	Days of life								
	1	7	8	10	11	13	15	16	20
White blood cells (/mm^3)	10 400	9500	5700	6500	5700	14 800	19 900	16 900	12 500
Hematocrit (%)	62	61	59	62	55	54	50	52	46
Hemoglobin (g/dL)	21.6	21.5	20.6	20.7	19	19.1	17.4	17.9	15.8
Platelets (/mm^3)	29 000	165 000	213 000	172 000	150 000	46 000	152 000	198 000	346 000
aPTT (s)	-	-	68	89	85	-	48	-	45
PT (%)	-	-	52	33	43	-	84	-	94
Fibrinogen (mg/dL)	-	-	-	172	-	-	367	-	-
C-reactive protein (mg/dL)	<0.1	-	0.1	<0.1	-	-	-	0.1	-
Serum PCR for dengue	-	-	Positive	-	-	-	-	-	-

aPTT: activated partial thromboplastin time; PCR: polymerase chain reactio; PT: prothrombin time.

and immune-mediated mechanisms, with damage mediated by both direct viral replication and the inflammatory response.[12,13] In neonates, evidence is limited; however, detection of viral RNA in cerebrospinal fluid, as in this patient, supports the possibility of central nervous system invasion even in the absence of neuroimaging findings. This finding demonstrates that normal structural studies do not rule out neurological involvement in the early stages.

Furthermore, viral identification in cerebrospinal fluid helped guide clinical management, shorten the duration of empirical antibiotic therapy, and focus monitoring on potential hemorrhagic and neurological complications. In endemic or epidemic settings, dengue should be systematically included in the differential diagnosis of neonatal sepsis when there is a compatible maternal history. Simultaneous mother-child evaluation using virological and serological tests[7-9] is essential for timely diagnosis and to optimize therapeutic decision-making.

In a particularly vulnerable population such as newborns, recognizing these unusual presentations and establishing standardized protocols for their management is a key strategy for improving prognosis and strengthening clinical surveillance in the face of a spreading disease. ■

REFERENCES

1. ANLIS-INVH, Argentine Association of Microbiology, Muñiz Hospital, Ministry of Health of Argentina, PAHO/WHO, Argentine Society of Infectious Diseases, et al. Infectious Diseases: Dengue. A Guide for Healthcare Providers. 4th ed. Buenos Aires: Ministry of Health; 2015.
2. Velandia ML, Castellanos JE. Dengue virus: structure and viral cycle. *Infect*. 2011;15(1):33-43.
3. Bhatt S, Gething PW, Brady OJ, Messina JP, Farlow AW, Moyes CL, et al. The global distribution and burden of dengue. *Nature*. 2013;496(7446):504-7. doi: 10.1038/nature12060.
4. Guzman MG, Gubler DJ, Izquierdo A, Martínez E, Halstead SB. Dengue infection. *Nat Rev Dis Primers*. 2016;2:16055. doi: 10.1038/nrdp.2016.55.
5. Paixão ES, Teixeira MG, Costa MCN, Rodrigues LC. Dengue during pregnancy and adverse fetal outcomes: a systematic review and meta-analysis. *Lancet Infect Dis*. 2016;16(7):857-65. doi: 10.1016/S1473-3099(16)00088-8.
6. Mulik V, Dad N, Buhmaid S. Dengue in pregnancy: Review article. *Eur J Obstet Gynecol Reprod Biol*. 2021;261:205-10. doi: 10.1016/j.ejogrb.2021.04.035.
7. Britez S, Mir R, Lacarruba J, Mendieta E, Céspedes E, Genes L. Vertically transmitted dengue: a case series review. *Pediatr (Asunción)*. 2014;41(1):25-31.
8. Salgado DM, Rodríguez JA, Lozano LDP, Zabaleta TE. Perinatal dengue. *Biomédica*. 2013;33(Suppl. 1):14-21. doi: 10.7705/biomedica.v33i0.1449.
9. Nguyen TM, Huan VT, Reda A, Morsy S, Giang HTN, Tri VD, et al. Clinical features and outcomes of neonatal dengue at Children's Hospital 1, Ho Chi Minh City, Vietnam. *J Clin Virol*. 2021;138:104758. doi: 10.1016/j.jcv.2021.104758.
10. Goldenberg RL, McClure EM, Saleem S, Reddy UM. Infection-related stillbirths. *Lancet*. 2010;375(9717):1482-90. doi: 10.1016/S0140-6736(09)61712-8.
11. Berberian G, Fariña D, Rosanova MT, Hidalgo S, Enría D, Mitchenko A, et al. Perinatal dengue. *Arch Argent Pediatr*. 2011;109(3):232-6. doi: 10.1590/S0325-00752011000300008.
12. Trivedi S, Chakravarty A. Neurological complications of dengue fever. *Curr Neurol Neurosci Rep*. 2022;22(8):515-29. doi: 10.1007/s11910-022-01213-7.
13. Singh S, Alallah J, Amrit A, Maheshwari A, Boppana S. Neurological manifestations of perinatal dengue. *Newborn (Clarksville)*. 2023;2(2):158-72. doi: 10.5005/jp-journals-11002-0066.
14. Pan American Health Organization, World Health Organization. Epidemiological Update: Dengue in the Region of the Americas. Washington, DC: PAHO/WHO; 2026. [Accessed on February 27, 2026]. Available at: https://www.paho.org/sites/default/files/2026/02/2026-18feb-phe-actualizacion-dengue-es-final1.pdf
15. Basurko C, Matheus S, Hildéral H, Everhard S, Restrepo M, Cuadro-Alvarez E, et al. Estimating the Risk of Vertical Transmission of Dengue: A Prospective Study. *Am J Trop Med Hyg*. 2018;98(6):1826-32. doi:10.4269/ajtmh.16-0794.