CASE REPORT

Sudden bilateral sensorineural hearing loss associated with severe dengue virus infection: Case report

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Introduction: Dengue is an arboviral disease transmitted by Aedes aegypti mosquitoes, prevalent in tropical regions, particularly in Latin America. Clinical manifestations range from mild illness to severe forms with multiorgan involvement. Idiopathic sudden sensorineural hearing loss (ISSNHL) is a sudden hearing impairment that can have multiple causes, significantly affecting functionality and quality of life, with a poor prognosis for recovery.

Case presentation: A 39-year-old woman with severe dengue, characterized by multiorgan dysfunction, developed auditory symptoms during hospitalization consistent with sudden bilateral sensorineural hearing loss. Audiological studies after discharge confirmed profound, irreversible hearing loss. No prior history or other causes were identified.

Conclusions: This case provides evidence of a possible association between severe dengue and sudden sensorineural hearing loss, likely due to immunoinflammatory and vascular mechanisms. It underscores the need for clinical vigilance for auditory symptoms in severe dengue and early intervention to prevent permanent hearing sequelae. Further studies are required to establish associations and pathophysiological mechanisms.

Keywords: Dengue, sudden sensorineural hearing loss, sudden deafness, neurological complications

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Introduction

Dengue is the most important viral disease transmitted to humans by female mosquitoes, primarily of the species *Aedes aegypti*, and the second most common vector-borne disease after malaria [1]. This virus affects tropical and subtropical regions such as the Caribbean, South America, Central and Southeast Asia, being endemic in approximately 100 countries and putting 3.9 billion people at risk [2].

In 2023, the World Health Organization (WHO) documented an unprecedented global rise in incidence, reporting over five million infections and nearly 5,000 deaths; in the Region of the Americas, Colombia contributed the largest proportion of severe cases [3].

The disease is caused by any of the four dengue virus serotypes (DENV 1–4), resulting in a spectrum of pathological conditions ranging from asymptomatic cases or mild febrile illness to severe hemorrhage, multiorgan dysfunction, and shock. According to the revised WHO classification, these forms are categorized as dengue without warning signs, dengue with warning signs, and severe dengue, which typically progresses through three phases: febrile, critical, and recovery [4].

Diagnosis is based on clinical presentation and confirmed by laboratory tests such as NS1 antigen from day 0 of symptom onset (peaking around day 4), IgM from day 5, and IgG from day 10 [1]. Symptomatic management and fluid administration remain the cornerstone of treatment; other interventions depend on disease phase and severity [5]. Two WHO-authorized dengue vaccines exist: Dengvaxia (9–45 years) requires prior-infection screening, limiting use; Q-Denga (6–16 years) is recommended as a two-dose regimen in endemic settings [6].

Sudden idiopathic sensorineural hearing loss (SISNHL) is characterized by the abrupt onset of unexplained unilateral or bilateral hearing loss of at least 30 dB affecting three consecutive audiometric frequencies, occurring within ≤72 hours and in the absence of an identifiable pathogenic cause [7]. The estimated annual incidence is approximately 5 cases per 100,000 individuals; however, this figure is likely underestimated due to spontaneous recovery rates ranging from 32% to 65% prior to presentation at emergency services. In Colombia, 17,199 consultations related to SISNHL were reported in 2016. Although SISNHL

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may occur at any age, it most frequently affects individuals between 30 and 50 years of age [7].

Although SISNHL is often considered idiopathic, several etiological theories exist, including autoimmune, infectious, neoplastic, vascular, hematologic, endocrine, and traumatic causes [7,8]. Clinically, it manifests as an abrupt unilateral or bilateral hearing loss, often accompanied by aural fullness, tinnitus, vertigo, nausea, and vomiting. Diagnosis relies on a comprehensive clinical history, thorough physical examination, bedside tuning fork tests—most commonly demonstrating Weber lateralization to the unaffected ear—and prompt audiological; however, initiation of treatment should not be delayed when audiometric confirmation is unavailable [8].

Current guidelines support systemic or intratympanic corticosteroid therapy, showing favorable outcomes when initiated within the first two weeks [7–9]. Hyperbaric oxygen therapy (HBOT) may be considered as an alternative first-line approach, as well as prostaglandin E1 (PGE1) combined with systemic steroids. Prognosis remains poor: approximately 30% of patients recover completely, 30% achieve partial improvement, and the remainder shows no change in hearing [9].

The objective of this case report is to present a case of sudden bilateral sensorineural hearing loss associated with severe dengue virus infection as a potential neurological complication and to highlight the importance of timely diagnosis, management, and follow-up to reduce the impact on patients' functionality and quality of life.

Case Presentation

A 39-year-old patient with no known medical conditions or prior hearing disorders was admitted to a tertiary care hospital in the city of Ibagué, Tolima Department, Colombia, presenting with a 72-hour history of fever, headache, myalgia, chills, persistent vomiting, and diffuse abdominal pain. On admission, vital signs revealed blood pressure of 100/60 mmHg, heart rate of 120 bpm, respiratory rate of 40 breaths per minute, dry mucous membranes, and a distended abdomen.

During clinical observation, the patient experienced progressive deterioration, characterized by hypotension refractory to crystalloid resuscitation, elevated serum creatinine levels, and oliguria. Abdominal sepsis was suspected; blood cultures were obtained, and empirical antimicrobial therapy with piperacillin–tazobactam was initiated, along with vasopressor support using norepinephrine. Arterial blood gas analysis demonstrated severe metabolic acidosis, consistent with impending respiratory failure, prompting orotracheal intubation. Shortly thereafter, the patient developed cardiac arrest with asystole, achieving return of spontaneous circulation after two minutes of advanced cardiopulmonary resuscitation.

Initial and follow-up laboratory tests revealed leukocytosis with neutrophilia, progressive thrombocytopenia, direct hyperbilirubinemia, prolonged coagulation times, elevated transaminases, non-reactive serological tests for syphilis and HIV, a positive dengue NS1 antigen, and negative serologies for *Leptospira* spp. and hepatitis B virus (Table 1).

The clinical course was complicated by multi-organ dysfunction involving the cardiovascular, respiratory, renal, hepatic, and hematologic systems. The patient developed stage III acute kidney injury according to AKIN criteria, requiring continuous venovenous hemodiafiltration. Due to persistent severe metabolic acidosis and suspicion of a cytokine storm, extracorporeal hemoperfusion was initiated.

Imaging studies included a chest radiograph demonstrating bilateral alveolar infiltrates with pleural effusion, abdominal ultrasonography showing hepatic steatosis, and abdominal computed tomography without evidence of fluid collections or contrast extravasation. Despite the initial severity, gradual clinical improvement was observed, with hemodynamic stabilization, recovery of renal function, normalization of liver enzyme levels, increased platelet counts, and a decrease in inflammatory markers (Table 1). Vasopressor support and mechanical ventilation were subsequently discontinued.

During the intensive care unit stay, the patient reported sudden onset of hearing loss accompanied by non-pulsatile tinnitus, vertigo, and a sensation of aural fullness. Audiological evaluation and otolaryngology consultation could not be performed during hospitalization. Given overall

Table 1. Laboratory follow-up.

Parameter	Reference	Day 3	Day 5	Day 7	Day 14
WBC (x10³/mm³)	5–10	20500	13770	12030	5700
Hemoglobin (g/dL)	12–15	12.4	9	10.4	9.5
Hematocrit (%)	37–45	37.9	27.1	31	28.3
Platelets (x10³/mm³)	150–350	145	31	97	218
ALT (U/L)	<40	172	330	98	
AST (U/L)	<40	183	340	90	
Total bilirubin (mg/dL)	0.3–1.2	3.47	3.99	1.28	
Direct bilirubin (mg/dL)	<0.3	2.67	3.18	0.89	
LDH (U/L)	230–460	365	2020	428	
Creatinine (mg/dL)	0.5–1.3	4.72	1.66	0.45	0.44
PT (s)	12–14	13.2	12.2	11	
PTT (s)	<10 control	47.9	85	24.4	
NS1 antigen		Positive			

LDH: lactate dehydrogenase; PT: prothrombin time; PTT: partial thromboplastin time; AST: aspartate aminotransferase; ALT: alanine aminotransferase.

clinical improvement, the patient was discharged on day 14 with persistent hearing loss.

Two months later, in the outpatient otology clinic, physical examination revealed no abnormalities; however, tonal audiometry and speech audiometry confirmed profound bilateral sensorineural hearing loss, with no window for salvage therapy (Figures 1). Due to the irreversibility and severity of the hearing deficit, cochlear implantation was indicated as an auditory rehabilitation measure.

Discussion

Dengue virus infection is the most prevalent and globally significant arboviral disease transmitted to humans [1]. It presents a clinical spectrum ranging from asymptomatic and mild forms to severe presentations, with complication rates between 0.9% and 3%, including hemorrhage, multiorgan involvement, shock, and even death [3,4,10]. Although neurological manifestations are uncommon, they are increasingly recognized, including encephalopathy, encephalitis, meningitis, immune-mediated disorders, and cerebrovascular complications [10].

ISSNHL is clinically significant due to its impact on functional ability and quality of life, particularly if unrecognized and untreated. While most cases are idiopathic, etiologies may include autoimmune, infectious, vascular, and hematologic disorders [7,8].

The association between sudden deafness and *Dengue virus* infection is rare, and its causal relationship remains unclear. Some case reports suggest that such manifestations should be considered within the neurological spectrum of dengue. The first report was by Ribeiro B et al. (2014) in Brazil, describing an adult who developed bilateral serous otitis media and sudden hearing loss, requiring hearing aid adaptation. Other infectious, autoimmune, or structural causes were ruled out. Two pathophysiological mechanisms were proposed: a viral mechanism (direct invasion and cross-reactive immune response in the inner ear) and a vascular mechanism (occlusion of the labyrinthine artery in the context of dengue vascular leak syndrome) [11].

Additional studies from India, Iran, and Pakistan have also linked dengue with neuropathies and auditory dysfunctions, emphasizing that systemic severity increases the risk of neurological complications and sequelae, and that lack of early intervention leads to irreversible hearing loss [12]. This is consistent with our patient, who was evaluated by otolaryngology and otology services two months

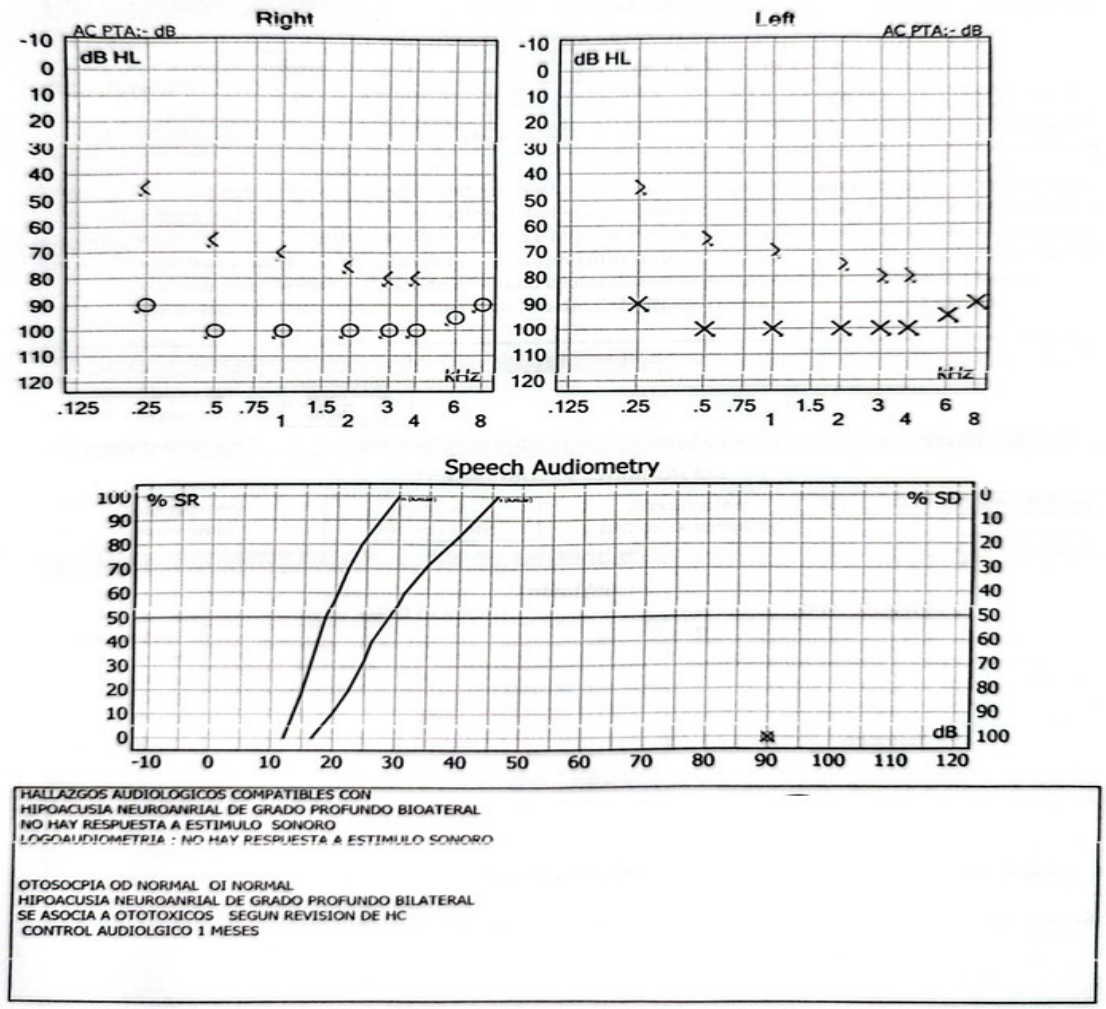

Fig. 1. Tonal audiometry and speech audiometry report: Profound bilateral sensorineural hearing loss; no response to auditory stimuli; speech audiometry shows no response to auditory stimuli

later, with no therapeutic window for rescue treatment.

Our analysis suggests that early diagnosis, prompt audiological evaluation, and timely treatment in patients with severe dengue are essential to prevent or minimize permanent sequelae [13]. However, the absence of specific protocols complicates adequate identification. These findings underscore the need for active clinical surveillance for auditory symptoms in patients with severe dengue.

Conclusion

This case contributes to the literature on rare complications of severe dengue by documenting irreversible bilateral sudden sensorineural hearing loss in a young patient. Given the secondary functional and psychosocial impact, it emphasizes the importance of raising awareness among healthcare providers regarding potential auditory involvement in dengue, especially in severe cases.

A multidisciplinary approach, early inclusion of audiological assessments, and timely initiation of therapies are key strategies to reduce permanent auditory disability. Further studies are needed to better establish the frequency, pathophysiological mechanisms, association between these conditions, and optimal therapeutic alternatives.

Authors'contributions

GFTP: study design, data acquisition and analysis, drafting of the manuscript, final approval.
MMAJ: study design, data acquisition and analysis, drafting of the manuscript, final approval.
CAMO: data acquisition and analysis, final approval.
JAPO: data analysis, drafting of the manuscript, final approval.

Ethical considerations

Ethical approval was obtained from a local institutional ethics committee, and written informed consent was obtained from the patient for publication.

Conflict of interest

None to declare.

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