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Case Report

Intraparenchymal Hemorrhage in a patient with Post-Traumatic Craniectomy and Severe Thrombocytopenia Due to Fatal Acute Dengue: A Case Report

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Abstract: Intracranial complications in dengue fever are uncommon but have been increasingly reported in severe cases. Dengue fever is a mosquito-borne viral illness caused by the dengue virus, which usually presents mild symptoms such as fever, headache, and myalgia. In a minority of cases, the disease can progress to more severe forms characterized by plasma leakage, severe thrombocytopenia, and bleeding. Although neurological manifestations are not typical in dengue infections, factors such as severe thrombocytopenia and previous neurosurgical interventions may predispose patients to intracranial hemorrhagic events. This case report presents an 18-year-old female with a fatal intraparenchymal hemorrhage, occurring in the context of severe dengue and a history of prior post-traumatic craniectomy. This case illustrates how dengue-related thrombocytopenia and endothelial dysfunction can trigger catastrophic intracranial bleeding in areas of prior structural brain injury. Considering preexisting neurosurgical lesions as potential sites of secondary hemorrhage in dengue patients with severe thrombocytopenia.

Keywords: Dengue Fever; Intracranial Hemorrhage; Thrombocytopenia.

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1. Introduction

Dengue fever is caused by a flavivirus, which is a vector borne ribonucleic acid (RNA) virus with four antigenically distinct serotypes (Dengue 1–4) [1]. It is a widespread infection that occurs in tropical climate regions of the planet, representing an important health, economic and social impact in endemic areas [2, 3]. Dengue mosquitoes occur in urban areas with altitudes below 2,200 meters above sea level, they lay their eggs in water reservoirs, and it can affect people of any age [3]. The four dengue virus serotypes (DENV 1, DENV 2, DENV 3, and DENV 4) are circulating in the Americas and are circulating simultaneously in several countries. The number of countries or territories with simultaneous co-circulation of two or more dengue virus serotypes has increased in the past 20 years, which has increased the risk of severe dengue as well as outbreaks in the Americas Region [4].

World Health Organization has estimated that between 50 to 100 million cases of dengue occur in the world each year, being the main human arbovirus existent [4,5]. The areas most affected by the dengue virus are Southeast Asia, the West Pacific, and the Americas, for which more than 3 billion people in the world are at risk of contracting the infection. Colombia is one of the countries in Latin America with the most elevated annual

rates of dengue [5]. A significant increase in dengue cases has been registered in South America, especially in Brazil, Peru, Venezuela, Ecuador, Paraguay, and Colombia [5, 6]. Brazil is the country that registers the most cases. However, it is in Colombia where the greatest number of cases of severe and fatal dengue cases have been registered in recent years [5].

Colombia has registered between 1978 and 2008: 803,157 cases of dengue in the country. An average of 25,177 cases occur annually; It has been increasing from 5.2 cases per 100,000 inhabitants in the 1990s to 18.1 cases per 100,000 inhabitants in the last years. According to Sivigila, the National Institute of Health of Colombia, in 2021, the incidence of dengue according to classification, more than 22,000 cases were reported, which more than 50% were without warning signs, approximately 45% warning signs dengue and less than 2% corresponded to severe dengue [6]. Approximately 10% of dengue patients may present neurological alterations during infection, leading to the belief that the virus may encourage neurological dysfunction, either directly by invading and infecting nervous tissue or indirectly by affecting other Organs that affect nerve function [7, 8].

Neurological complications are infrequent in relation to other manifestations of the disease [7]. Usually, it results from multisystem dysfunction secondary to liver failure, cerebral hypo perfusion, electrolyte imbalance, shock, cerebral edema, and hemorrhage related to vascular leak. Encephalopathy, encephalitis, motor deficit, transverse myelitis, acute disseminated encephalomyelitis, or Guillain-Barré syndrome have been reported [9]. However, when preexisting neurological damage exists, such as prior neurosurgical interventions, the risk of secondary intracerebral hemorrhage may be substantially elevated [10].

We report a case of severe dengue fever complicated by intracranial hemorrhage and intraparenchymal hematoma in a patient with a history of prior craniectomy, who developed distributive shock and death.

2. Case Report

An 18-year-old female with a history of a traumatic brain injury following a motor vehicle accident in November 2018, which resulted in a subdural hematoma requiring craniotomy, presented to our facility. Since the incident, she had been left with residual left-sided hemiparesis. Eight days prior to admission, she developed intermittent, undocumented fever associated with myalgia, arthralgia, and diffuse headache. She sought care at a primary care facility, where bacterial tonsillitis was diagnosed and penicillin prescribed. Due to persistent fever, abdominal pain, and repeated vomiting, she reconsulted. Laboratory tests revealed leukopenia and thrombocytopenia (platelet count: 31,000/mm^3), raising suspicion of dengue fever with warning signs.

During the first day of hospitalization, the patient experienced generalized tonic-clonic seizures. A brain CT scan revealed a right temporoparietal intraparenchymal hemorrhage measuring 40.7 × 46.5 mm, with surrounding edema (Figure 1), located within a residual malacic area corresponding to her prior traumatic brain injury. Given the clinical context and laboratory findings, the case was classified as suspected severe dengue, and she was referred to as a higher-level care facility for further management.

Clinically worsening condition, a follow-up brain CT scan (Figure 2) was performed, revealing an increase in the size of the hemorrhage with associated midline shift, but without additional neurological deterioration. Platelet transfusion was initiated, with two units administered and three additional units reserved. Upon evaluation by the neurosurgery team, marked thrombocytopenia was noted. Surgical evacuation was planned, contingent upon optimization of coagulation parameters, with a target platelet count of ≥100,000/mm^3. Although the patient initially presented with a markedly reduced platelet count, the presence of intraparenchymal hematoma and rapid clinical deterioration, characterized by signs of severe intracranial hypertension, including decerebrate posturing and declining neurological status, necessitated urgent surgical intervention. A few hours after admission, the patient exhibited worsening signs of intracranial hypertension,

prompting the initiation of deep sedation, invasive mechanical ventilation, and osmotic therapy with boluses of 3% hypertonic saline.

Figure 1. Simple brain computed tomography revealed right frontoparietal hemorrhagic lesion with perilesional edema with expansive effect and deviation of structures from the midline and subfalcine herniation. A. Right frontoparietal craniectomy area with intraparenchymal parietal bleeding, B. increased bleeding area in the right hemisphere, and mass effect C. massive right frontotemporoparietal hematoma, with midline deviation.

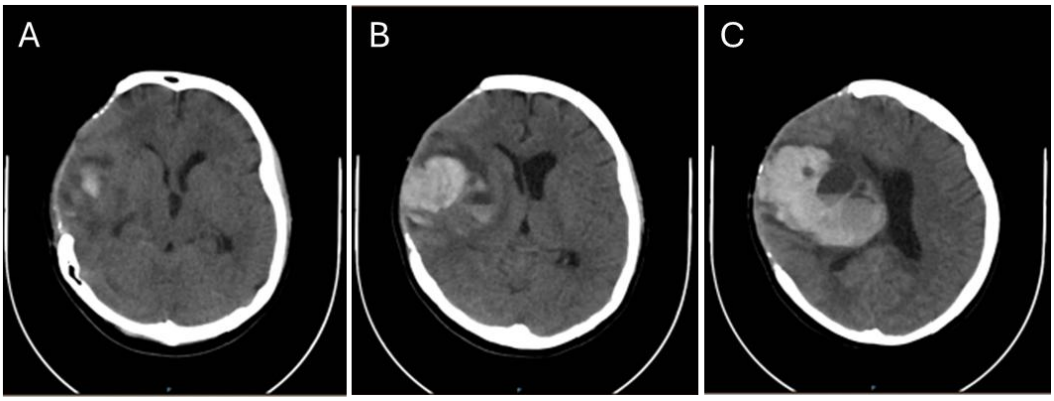
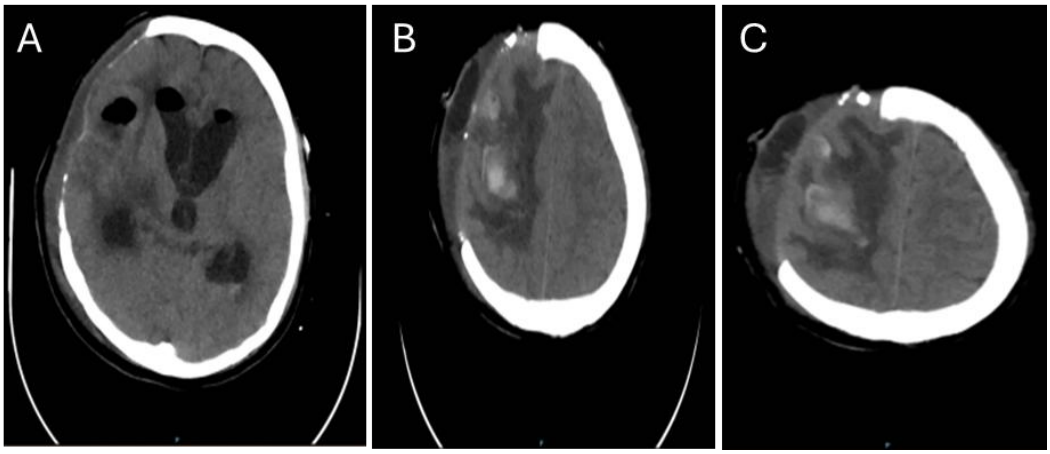


Figure 2. Simple CT brain. A. Pneumoencephalus in the drainage area of the hematic collection. normally inserted ventricular catheter., and Satisfactory drainage of the hematoma. B, C. Vasogenic severe edema with bleeding at the right frontoparietal level. Postsurgical changes of the encephalic tissue at the level of the craniectomy.



The critical neurological status, an emergency decompressive craniectomy combined with evacuation of the hematoma was performed as a life-saving measure. Although platelet transfusions had already been administered, and a post-transfusion platelet count of 137,000/mm^3 was subsequently confirmed, clarification of the precise count at the time of surgical decision-making would offer important insights into the clinical balance between surgical urgency and hemostatic optimization in neurocritical care. The surgical procedure was completed successfully, with minimal intraoperative blood loss and no immediate complications. This case highlights the complexity of managing life-threatening intracerebral hemorrhage in the setting of dengue-associated thrombocytopenia, particularly in patients with pre-existing neurological vulnerability.

Postoperatively, a follow-up CT scan (Figure 3) showed new intraventricular hemorrhage, prompting surgical placement of an external ventricular drain (EVD). Despite intervention, she continued to exhibit signs of intracranial hypertension with sustained elevated intracranial pressure reaching values of even 44 mmHg. Repeat imaging revealed

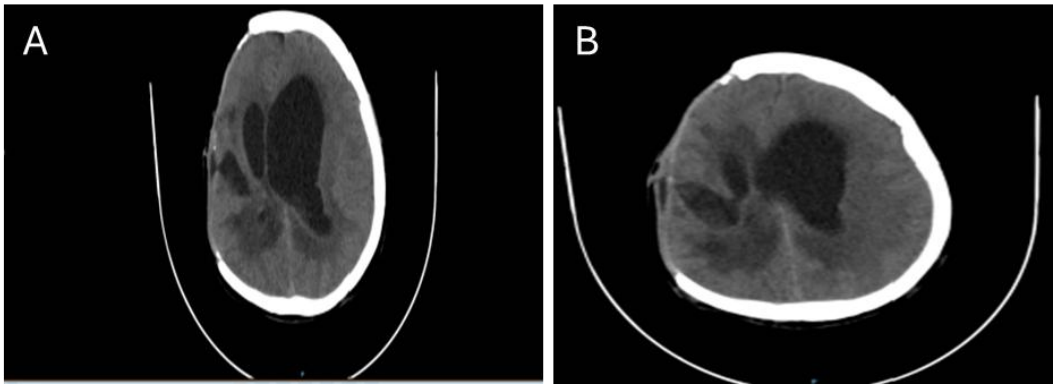
persistent hematoma, midline shift, and hemostatic material within the hematoma cavity. She remained in a critical neurological state, sedated for neuroprotection, under vasopressor support and anti-edema therapy (continued boluses of 3% hypertonic saline solution, underwent deep sedation, and was placed on mechanical ventilation with controlled hyperventilation).

Dengue IgM was positive, confirming the diagnosis of severe dengue with central nervous system involvement. Over the following days, the patient's condition remained critical, on vasopressor support, sedoanalgesia, and measures to control cerebral edema. However, after several weeks in the intensive care unit, she developed ventriculitis due to Klebsiella pneumoniae and a mixed fungal infection involving both yeast and filamentous fungi. Targeted intravenous antimicrobial and antifungal therapy was initiated (vancomycin, meropenem, and caspofungin), but despite maximal supportive and therapeutic efforts, the patient's clinical course remained poor. She succumbed to her illness and died.

3. Discussion

Intraparenchymal hemorrhage in patients with dengue has infrequently been reported. Dengue is an arbovirus of the Flaviviridae family, it is transmitted by arthropods, mainly by Aedes aegypti. Dengue is the most common infectious disease by vectors, most common in the world. The infection may be asymptomatic, and sometimes evolves into a life-threatening condition, called severe dengue, which can progress to multi-organ disease, and death [1]. Hemorrhagic complications in dengue fever are multifactorial, resulting from a combination of vascular endothelial injury, platelet dysfunction, and thrombocytopenia [12,13]. During the acute phase of infection, elevated levels of inflammatory mediators such as interleukin-8 (IL-8) and tumor necrosis factor-alpha (TNF-α) contribute to increased vascular permeability. This leads to plasma leakage into interstitial spaces, resulting in serosal effusions and tissue edema, which are hallmark features of severe dengue [13].

Figure 3. A and B. Right parietal encapsulated organized hypodense area with ventricular dilatation on the left side and severe cerebral edema with post-surgical changes of the encephalic tissue at the level of the craniectomy.



Although neurological complications in dengue are considered rare, reported in only 0.8% to 5.4% of cases, they are often severe and associated with high mortality [12, 13]. These complications may include encephalitis, intracranial hemorrhage, cerebral edema, and seizures, and can result from direct viral neurotropism, metabolic disturbances, or coagulopathy-related bleeding. Despite their low incidence, the clinical impact is significant and demands a high index of suspicion to ensure timely diagnosis and intervention [12]. Neuroimaging, particularly cranial computed tomography (CT) and magnetic resonance imaging (MRI), plays a critical role in the evaluation of these patients. Reported findings include intraparenchymal hemorrhage, often affecting the thalamus, basal ganglia, cortex, or subcortical white matter, as well as cerebral edema and diffuse brain swelling [13,14].

There is no specific antiviral treatment for dengue. Management of intracerebral hemorrhage in this setting is largely supportive and focuses on controlling bleeding, ensuring hemodynamic and intracranial stability, and avoiding further neurological compromise. Conservative fluid management is essential, as hypovolemia and shock can exacerbate cerebral injury. Platelet transfusion is typically indicated when platelet counts fall below 20,000/mm^3, or in the presence of active bleeding [15]. However, in the setting of neurosurgical intervention, guidelines recommend maintaining platelet counts ≥100,000/mm^3 and correcting any coagulopathy prior to invasive procedures [15, 16].

Importantly, patients with a history of prior neurosurgical intervention may be at greater risk for secondary intracerebral hemorrhage in the context of severe dengue [14]. Neurosurgery can leave residual structural and vascular vulnerabilities, including scar tissue, altered autoregulation, and weakened vessel walls, which, when coupled with dengue-induced coagulopathy, significantly increases the risk of hemorrhagic events [10]. In such cases, even minor physiological insults may precipitate devastating intracranial bleeding due to the limited compensatory reserve of previously injured brain tissue.

Post-traumatic epilepsy and vascular vulnerability in encephalomalacic brain tissue are well-documented sequelae of traumatic brain injury and neurosurgical interventions such as craniectomy. Regions of encephalomalacia are characterized by gliosis, microvascular remodeling, and impaired autoregulation, rendering them structurally and functionally compromised. These areas are particularly susceptible to hemorrhagic events under conditions of systemic stress. Literature suggests that such regions may serve as foci for epileptiform activity and exhibit reduced vascular integrity due to chronic inflammatory changes and altered perfusion dynamics. In the context of severe dengue infection, systemic endothelial dysfunction, profound thrombocytopenia, and increased vascular permeability converge to create a high-risk environment for spontaneous bleeding. When these systemic factors intersect with preexisting cerebral vulnerabilities, as in this case, the risk of catastrophic intracranial events, such as massive intraparenchymal hemorrhage, seems amplified. This highlights the need for heightened clinical suspicion and early neuroimaging in dengue patients with prior neurosurgical history who present with new-onset neurological symptoms.

The case presented in this report exemplifies this risk. An 18-year-old female with a prior history of decompressive craniectomy for traumatic brain injury developed severe thrombocytopenia due to dengue, followed by intraparenchymal hemorrhage. Management required admission to the intensive care unit, initiation of cerebral anti-edema measures (including deep sedation, hyperosmolar therapy, and controlled hyperventilation), and emergency surgical decompression. Despite aggressive management, the prognosis in such scenarios remains guarded.

When compared with previously published case series, the risk of intracranial complications in dengue, while low in the general population, increases substantially in the presence of additional risk factors. In a small series of five patients with dengue-related intracranial hemorrhage, they all exhibited thrombocytopenia and coagulation abnormalities; two patients who underwent surgical intervention had favorable outcomes [17]. Another series reported a significant burden of neurosurgical emergencies during a dengue outbreak, with coagulopathy complicating the clinical and surgical management [18]. These findings support the notion that prognosis depends not only on the severity of the hemorrhage but also on early recognition, the ability to correct hemostatic parameters, and access to timely neurosurgical intervention. The overall prognosis for dengue-associated intracerebral hemorrhage remains poor, with high rates of morbidity and mortality, especially in cases requiring intensive care admission. Patients often experience prolonged hospitalization and are at risk for secondary complications, such as nosocomial infections, nutritional deficits, multiorgan dysfunction, and long-term neurological sequelae [13].

In summary, while rare, intracranial hemorrhage secondary to dengue fever represents a life-threatening complication, particularly in patients with preexisting neurological injury. Timely recognition, neuroimaging, and a multidisciplinary approach, including critical care, infectious disease, and neurosurgical teams, are essential to optimize outcomes in this high-risk population.

4. Conclusion

This case poignantly illustrates the complex pathophysiological mechanisms by which dengue-induced thrombocytopenia and endothelial dysfunction can precipitate life-threatening intracranial hemorrhage, particularly in regions of the brain previously compromised by trauma or neurosurgical intervention. It brings to light the heightened vulnerability of structurally altered neural tissue, where disrupted vascular integrity may predispose to secondary hemorrhagic complications in the context of severe dengue infection. The interplay between pre-existing neurological damage, coagulopathy, and dengue-induced vascular fragility likely played a central role in the hemorrhagic event and its progression. This case highlights how reduced neurological reserve after prior neurosurgery can significantly worsen the consequences of dengue-associated bleeding.

Clinicians should maintain heightened vigilance for signs of intracranial bleeding in dengue patients exhibiting profound thrombocytopenia, especially those with a history of neurosurgical procedures or traumatic brain injury. Early diagnosis through neuroimaging and aggressive multidisciplinary management, including hematological support, neurocritical care, and timely surgical intervention, are paramount to improving clinical outcomes in this vulnerable subgroup. Moreover, this case underscores the imperative for further clinical studies to elucidate the precise mechanisms linking dengue virus pathogenesis with cerebrovascular complications, and to develop evidence-based protocols for risk stratification, monitoring, and management of dengue patients with preexisting neurological lesions. Such research will be vital to inform clinical guidelines and optimize care for this high-risk population.

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