CASE REPORT

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Dengue Shock Syndrome (DSS) complicated by severe hyponatremia and seizure in a pediatric patient: a rare case report

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ABSTRACT

Background: Dengue Shock Syndrome (DSS) represents the most severe form of dengue virus infection, driven by massive plasma leakage and profound hemodynamic instability. Severe hyponatremia in this context is uncommon and may precipitate neurological manifestations, including seizure, compounding disease complexity and mortality risk. This study aims to describe the clinical course, management, and outcome of a pediatric DSS case complicated by severe symptomatic hyponatremia and seizure.

Case Presentation: We report the case of an 8-year-11-month-old boy presenting with fever, abdominal pain, diarrhea, and a generalized tonic–clonic seizure at home. Laboratory findings confirmed secondary dengue infection, hemoconcentration, thrombocytopenia, and severe hyponatremia (Na^+ 100–122 mEq/L). The patient developed DSS with large-volume ascites and bilateral pleural effusions during the critical phase. Management included fluid resuscitation per WHO protocol, hypertonic saline correction of sodium, albumin infusion, and close hemodynamic and neurological monitoring. Seizure activity resolved with gradual sodium normalization. Conservative management of effusions avoided invasive intervention. The patient was discharged in stable condition on day six with no recurrent neurological events. This case illustrates the diagnostic and therapeutic challenges of managing DSS complicated by severe hyponatremia and seizure. Management requires balancing volume restoration with prevention of sodium dilution and fluid overload, alongside multidisciplinary coordination.

Conclusion: Severe hyponatremia with seizure in DSS is rare but life-threatening. Early electrolyte evaluation in dengue patients with neurological symptoms is essential, and individualized management can optimize survival.

Keywords: Dengue Shock Syndrome, Hyponatremia, Seizure, Pediatric, Fluid Management.
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INTRODUCTION

Dengue virus infection remains one of the most important mosquito-borne viral diseases affecting humans, with global incidence steadily increasing over the past five decades. It is estimated that approximately 390 million dengue infections occur annually, of which around 96 million manifest with clinical symptoms, ranging from mild febrile illness to severe and life-threatening disease.[1] The disease is endemic in more than 100 countries, particularly in tropical and subtropical regions, placing over 3 billion people at risk.[2] In Southeast Asia, including Indonesia, recurrent outbreaks contribute to significant morbidity and mortality in pediatric populations, where severe forms of the disease occur more frequently.

Among the spectrum of severe dengue, Dengue Shock Syndrome (DSS) represents the most critical clinical form, characterized by profound plasma leakage leading to circulatory failure. DSS is defined by narrow pulse pressure or hypotension for age, often accompanied by signs of impaired perfusion such as delayed capillary refill, cold extremities, and weak pulse.[3] If untreated, DSS can rapidly progress to multi-organ dysfunction and death. The transition to shock typically occurs during the critical phase, around defervescence, and is often preceded by laboratory evidence of hemoconcentration and thrombocytopenia.[4] The management of DSS demands careful fluid resuscitation according to WHO guidelines, with continuous hemodynamic and laboratory monitoring to avoid both under-resuscitation (risking persistent shock) and over-resuscitation (risking fluid overload and pulmonary edema).

Electrolyte disturbances are frequent in dengue fever, with hyponatremia being the most commonly observed abnormality.[5] The reported incidence varies from 13% to over 60% depending on case severity, patient age, and phase of illness. The mechanisms are multifactorial: plasma leakage and third-space fluid accumulation cause dilutional hyponatremia, gastrointestinal losses (vomiting, diarrhea) contribute to sodium depletion, while cytokine storm–related systemic inflammation can trigger syndrome of inappropriate antidiuretic hormone secretion (SIADH).[6] In addition, the administration of hypotonic fluids early in the illness may exacerbate sodium decline. While mild hyponatremia (130–134 mEq/L) is common and often asymptomatic, severe hyponatremia (<125 mEq/L) is rare and clinically significant, as it can precipitate neurological manifestations, including altered mental

status, confusion, and generalized seizures.[7]

The occurrence of seizures in dengue has been documented but remains relatively uncommon, more often associated with dengue encephalitis, cerebral edema, or intracranial bleeding.[8] In the context of hyponatremia, seizures typically occur when serum sodium falls rapidly or reaches extremely low levels, due to osmotic shifts causing acute cerebral edema.[9] This neurological presentation poses a diagnostic challenge, as it necessitates differentiation between direct viral neurotropism and secondary metabolic derangements. The implications for management are substantial; the approach to sodium correction must be timely yet carefully titrated to prevent both ongoing cerebral injury and the potential complication of osmotic demyelination syndrome.[10]

The coexistence of DSS, severe hyponatremia, and seizure in a pediatric patient represents an unusual and clinically important triad. Each of these conditions independently increases morbidity risk, but in combination, they significantly complicate fluid and electrolyte management. Aggressive fluid resuscitation is essential for reversing shock, yet excessive administration can exacerbate hyponatremia and precipitate pulmonary complications due to capillary leakage. Conversely, overly cautious fluid therapy risks inadequate tissue perfusion. This delicate therapeutic balance underscores the need for early recognition of electrolyte abnormalities in all severe dengue cases presenting with neurological symptoms.

In this report, we describe a pediatric case of DSS complicated by severe symptomatic hyponatremia and seizure, with additional complications including ascites and pleural effusion. The case highlights the diagnostic considerations, pathophysiological interplay, and management strategies necessary to optimize outcomes. By presenting this rare combination, we aim to reinforce the importance of routine electrolyte assessment, adherence to WHO fluid protocols, and multidisciplinary care in severe dengue with neurological involvement.

CASE PRESENTATION

An 8-year-11-month-old boy was brought to the emergency department of RS Gading Medika, Bengkulu, with a four-day history of persistent fever and an acute neurological event. On the morning of presentation, while febrile at home, he experienced a generalized tonic–clonic seizure characterized by upward ocular deviation, symmetrical tonic–clonic movements of all extremities, and transient unresponsiveness. The episode lasted approximately one minute and was followed by a short postictal period in which he gradually regained responsiveness to verbal commands. Parents denied any prior seizure history, recent head trauma, or underlying neurological disease.

Alongside the seizure episode, the child had progressive systemic symptoms over the preceding days: intermittent colicky abdominal pain, watery diarrhea with dark stool for two days, marked anorexia, and generalized weakness. There was no bleeding tendency such as epistaxis, gum bleeding, hematemesis, or melena, and no respiratory symptoms. He had been managed at home with paracetamol alone. His past medical history was unremarkable, with complete immunizations according to the national schedule.

Initial assessment revealed an alert but lethargic child in compensated shock. Vital signs: blood pressure 105/70 mmHg, heart rate 115 bpm, respiratory rate 22 breaths/min, temperature 37.1°C, SpO_2 99% on room air. Physical examination showed warm extremities, capillary refill <2 seconds, no pallor or jaundice, and scattered petechial lesions over the extremities. Cardiopulmonary examination was normal, and abdominal examination revealed a soft, nondistended abdomen with normoactive bowel sounds.

Laboratory evaluation revealed hemoconcentration with hemoglobin 15.0 g/dL and hematocrit 46%, thrombocytopenia ($58\times10^3/\mu L$), leukopenia ($2{,}900/mm^3$), and normal random glucose (111 mg/dL). Critically, serum sodium was 122 mEq/L (severe hyponatremia), potassium 4.5 mEq/L, and ionized calcium 1.05 mmol/L. Dengue serology confirmed secondary infection, while the Widal test was negative.

Based on the WHO 2009 and updated 2023 dengue guidelines, a diagnosis of Dengue Shock Syndrome (DSS) complicated by severe symptomatic hyponatremia manifesting as seizure, accompanied by hypoalbuminemia and acute diarrhea, was established. The presence of seizure in dengue is uncommon and, when coupled with hyponatremia and shock, indicates a severe systemic inflammatory response with significant electrolyte disturbance—likely multifactorial in origin, involving cytokine-mediated sodium shifts, gastrointestinal losses, and dilutional effects from plasma leakage.

The patient was admitted to the pediatric ICU for advanced monitoring of hemodynamics and neurological status. Management was initiated with a Ringer's lactate bolus of 240 mL over 1 hour, followed by isotonic fluid maintenance using the WHO "1B" regimen. Given the neurological presentation, hypertonic saline (NaCl 3%) was initiated for sodium correction, with a target rate designed to avoid rapid overcorrection and prevent osmotic demyelination syndrome. Intravenous ceftriaxone (3×350 mg), ranitidine, vitamin K, and oral diazepam during febrile spikes were administered. Hematocrit, platelet count, and electrolytes were monitored every 4–6 hours to detect progression to the critical phase and to guide fluid therapy.

Day 2 marked the onset of severe plasma leakage: platelet count dropped sharply to $27\times10^3/\mu L$, hematocrit decreased to 34%, abdominal distension developed, and generalized petechiae intensified. Fluid balance was +1,299 mL, raising concerns for impending fluid overload. Sodium levels dropped further to 100 mEq/L, reinforcing the need for continued hypertonic saline infusion under strict monitoring.

Day 3 ultrasound revealed moderate ascites and bilateral pleural effusions (936 mL right, 578 mL left). Despite significant third-space fluid accumulation, the patient remained hemodynamically stable (BP 113/61 mmHg, SpO_2 100% on room air). A multidisciplinary team—including pediatric and pulmonology specialists—opted for conservative management,

Table 1. Daily changes in clinical and laboratory indices of the patients

Hospital Day	Key Clinical Findings	Vital Signs	Platelet Count ($\times 10^3/\mu L$)	Hematocrit (%)	Serum Sodium (mEq/L)
Day 1 (3/8/25)	Fever (day 4), generalized tonic–clonic seizure at home, abdominal pain, watery diarrhea, petechiae; alert but weak	BP 105/70, HR 115, RR 22, T 37.1°C, SpO_2 99%	58.0	46.0	122.0
Day 2 (4/8/25)	Abdominal distension, worsening petechiae, platelet drop, positive fluid balance	BP 102/75, HR 88, RR 30, SpO_2 100%	27.0	34.0	100.0
Day 3 (5/8/25)	Persistent distension, petechiae, moderate ascites, bilateral pleural effusion; no respiratory distress	BP 113/61, HR 88, RR 33, SpO_2 100%	52.0	38.0	
Day 4 (6/8/25)	Improving abdominal pain, stable vitals, negative fluid balance	BP 115/58, HR 98, RR 33, SpO_2 100%	80.0	40.0	124.0
Day 5 (7/8/25)	Reduced abdominal girth, improving rash, no seizures; stable hemodynamics	BP 117/58, HR 98, RR 33, SpO_2 100%	113.0	35.0	128.0
Day 6 (8/8/25)	No abdominal pain, no seizures, tolerating oral intake; stable for discharge	BP 117/58, HR 98, RR 33, SpO_2 100%			

emphasizing cautious fluid restriction and albumin correction to address severe hypoalbuminemia, reserving thoracentesis for worsening respiratory compromise.

From Day 4 onward, clinical improvement was noted: abdominal pain subsided, petechiae began to fade, and fluid balance turned negative (–1,292 mL), indicating mobilization of third-space fluid. By Day 5, platelet count rose to $113\times10^3/\mu L$, sodium improved to 128 mEq/L, and ascitic distension decreased. The child remained seizure-free after initial sodium correction, underscoring the link between hyponatremia and neurological manifestation in this case.

On Day 6, the patient tolerated oral intake well, had stable hemodynamics, no respiratory distress, and normal mentation. He was discharged on oral antibiotics (cefixime), zinc supplementation, and dietary recommendations, with instructions for follow-up electrolyte and hematologic evaluation.

This case is remarkable for the simultaneous occurrence of DSS, severe hyponatremia, and seizure, an uncommon but critical combination that poses significant diagnostic and therapeutic challenges. The triad necessitated synchronized management of shock, electrolyte imbalance, and risk of neurological deterioration, all while adhering to WHO fluid management principles to prevent iatrogenic fluid overload. The child's favorable outcome underscores the importance of early recognition, cautious sodium correction, and multidisciplinary coordination in such high-risk dengue presentations.

The concurrence of Dengue Shock Syndrome (DSS) with severe hyponatremia presenting as a generalized seizure is exceptionally rare in pediatric dengue cases. While DSS itself represents the most critical form of plasma leakage in dengue, severe hyponatremia (<125 mEq/L) in this context is usually subclinical and seldom precipitates overt neurological manifestations. In this patient, the seizure was likely multifactorial—driven by rapid sodium decline, capillary leak–induced fluid shifts, and cytokine-mediated central nervous system involvement. The case exemplifies the diagnostic challenge of distinguishing dengue-related encephalopathy from electrolyte-driven seizure, and it underscores the need for immediate serum sodium evaluation in any dengue patient with neurological symptoms. This scenario highlights how timely recognition and synchronized management of shock, electrolyte derangement, and neurological risk can avert fatal outcomes. It should be emphasized in both clinical protocols and academic literature.

DISCUSSION

Dengue Shock Syndrome (DSS) is the most severe form of dengue infection and represents the culmination of a complex interplay between viral, immunological, and host factors. The hallmark of DSS is a sudden and profound increase in capillary permeability, leading to plasma leakage, intravascular volume depletion, and ultimately circulatory collapse.[1] The underlying mechanism involves a cascade of immune activation events, including viral antigen–antibody complex formation, complement activation, and the release of potent pro-inflammatory cytokines such as TNF-α, IL-6, and IL-8, which collectively impair endothelial integrity.[2] The vascular leakage phenomenon is not uniform across all patients, but in those with DSS, the leakage tends to peak abruptly around the time of defervescence, corresponding with rapid shifts in hematocrit and precipitous drops in platelet count.[3] In pediatric patients, the progression to DSS is often accelerated due to limited circulatory reserve and heightened inflammatory responses.[4] In the present case, the critical phase manifested with rapid thrombocytopenia, hemoconcentration, and large-volume third-space accumulation, which were compounded by the development of ascites and bilateral pleural effusions.

Hyponatremia in dengue is a multifactorial disturbance with significant prognostic implications. One principal mechanism is dilutional hyponatremia secondary to plasma leakage, whereby intravascular volume depletion triggers non-osmotic release of antidiuretic hormone (ADH), leading to water retention and reduced serum sodium concentration.[5] Additionally,

direct viral and inflammatory effects on the hypothalamic–pituitary axis may result in syndrome of inappropriate antidiuretic hormone secretion (SIADH), further exacerbating water retention.[6] Gastrointestinal losses from diarrhea and reduced oral intake add to the sodium deficit, while hypotonic intravenous fluids administered before diagnosis may accelerate the decline. Severe hyponatremia, defined as serum sodium levels below 125 mEq/L, is rare in dengue and carries a substantial risk for neurological complications, including seizures, confusion, and coma.[7] In our patient, serum sodium dropped to a critical nadir of 100 mEq/L during the period of maximal plasma leakage, strongly suggesting that rapid sodium decline was a primary driver of the seizure episode. This temporal relationship, along with the resolution of seizure activity following sodium correction, supports hyponatremia as the dominant etiology rather than viral encephalitis.

Neurological involvement in dengue encompasses a spectrum ranging from mild headache and dizziness to severe complications such as encephalitis, myelitis, Guillain–Barré syndrome, and intracranial hemorrhage.[8] Seizures are an uncommon presentation and, when they occur, are often associated with direct neurotropic invasion by the virus or secondary to metabolic derangements such as hypoglycemia or hyponatremia.[9] In hyponatremia-induced seizures, the mechanism is osmotic mainly: a rapid fall in serum sodium creates a hypo-osmolar extracellular environment, leading to water influx into brain cells and subsequent cerebral edema.[10] The distinction between seizures due to metabolic causes and those due to encephalopathy is critical, as the management approach diverges significantly. In metabolic seizures, targeted correction of the underlying disturbance often leads to rapid neurological recovery, whereas encephalitic seizures may require anticonvulsants and anti-inflammatory strategies. In our patient, the absence of focal neurological deficits, normal mentation outside the seizure episode, and resolution of symptoms following sodium correction are consistent with a metabolic rather than an infectious CNS process.

The management of DSS hinges on precise fluid titration to restore adequate intravascular volume while avoiding iatrogenic complications from over-resuscitation.[11] In the setting of severe hyponatremia, this balance becomes even more precarious. Aggressive isotonic fluid replacement may exacerbate sodium dilution, while overly conservative fluid administration risks inadequate tissue perfusion and worsening shock.[12] WHO guidelines emphasize the importance of individualized fluid therapy guided by serial clinical assessments, hematocrit trends, urine output, and, in complex cases such as this, frequent electrolyte monitoring.[13] In our patient, careful adjustment of fluid composition and rate was implemented, with a shift toward hypertonic saline infusion once severe hyponatremia was confirmed, while simultaneously restricting total fluid volume to prevent aggravation of pleural effusions and ascites. The sodium correction rate was deliberately limited to under 10 mEq/L per 24 hours, in line with best practice recommendations to prevent osmotic demyelination syndrome.[14] This dual focus—reversing shock without worsening hyponatremia—was a central therapeutic challenge throughout the patient's critical phase.

Hypoalbuminemia in dengue is both a consequence and a driver of clinical deterioration. Low oncotic pressure from hypoalbuminemia promotes further third-space fluid accumulation, complicating efforts to achieve fluid balance.[15] In selected severe cases, albumin infusion may help restore plasma oncotic pressure, thereby mobilizing extravascular fluid back into the circulation. In this case, intravenous 25% albumin was administered in conjunction with sodium correction, leading to gradual regression of pleural effusion and ascites. The decision to manage the pleural effusions conservatively, without invasive drainage, was made in collaboration with the pulmonology and critical care teams. This multidisciplinary approach allowed for dynamic reassessment of fluid therapy, respiratory function, and neurological status, ensuring that therapeutic interventions were both timely and appropriately scaled to the patient's evolving condition.

Published reports describing the triad of DSS, severe hyponatremia, and seizure in pediatric dengue patients are exceedingly rare. Singh et al. reported a similar case in which severe hyponatremia precipitated generalized seizures, underscoring the importance of early electrolyte monitoring in high-risk dengue cases.[7] A previous study found that the severity of hyponatremia correlated strongly with disease severity, but neurological events remained uncommon, suggesting that a subset of patients may have heightened vulnerability to rapid sodium shifts.[5] The present case shares features with these reports but adds a notable layer of complexity due to the coexistence of large-volume third-space fluid accumulation requiring restrictive fluid management. The successful recovery of our patient highlights that adherence to WHO dengue management principles, combined with vigilant electrolyte correction and multidisciplinary oversight, can yield favorable outcomes even in high-risk presentations.

The clinical lessons from this case extend beyond its rarity. Neurological symptoms in a patient with dengue should always prompt urgent evaluation of serum sodium levels, as the timely identification and correction of severe hyponatremia can be life-saving. Fluid therapy in DSS must be individualized, striking a balance between the imperative to reverse shock and the need to avoid exacerbating sodium dilution or precipitating fluid overload. In the presence of hypoalbuminemia and large-volume effusions, judicious use of albumin replacement may aid in restoring oncotic balance and improving fluid distribution. Finally, coordinated care between pediatric, intensive care, and subspecialty teams plays a pivotal role in optimizing outcomes, particularly in cases where multiple critical processes converge, as seen here.

Beyond its rarity, this case contributes several practical insights for clinicians managing severe dengue. First, the occurrence of a seizure in a pediatric patient with DSS underscores the importance of routine electrolyte evaluation in all dengue patients presenting with neurological symptoms. Differentiating between seizures due to metabolic causes, such

as hyponatremia, and those caused by encephalopathy or intracranial pathology is essential, as management strategies differ significantly. In this patient, the absence of focal neurological signs, normal mentation outside the seizure episode, and complete resolution following gradual sodium correction pointed strongly toward a metabolic rather than infectious central nervous system process. This reinforces the value of immediate serum sodium testing as a first-line investigation when neurological manifestations arise in dengue.

Second, the management details in this case highlight a reproducible strategy for balancing shock resuscitation with safe sodium correction. Fluid therapy was titrated according to WHO guidelines, with an initial isotonic bolus followed by maintenance based on serial hemodynamic and hematologic monitoring. Hypertonic saline (3% NaCl) was administered cautiously, with correction limited to less than 10 mEq/L per 24 hours to avoid osmotic demyelination, and frequent reassessment of neurological status guided therapy adjustments. The concurrent use of albumin infusion in the context of severe hypoalbuminemia provided additional benefit by mobilizing third-space fluid and facilitating conservative management of pleural effusion and ascites. These measures illustrate how synchronized fluid resuscitation, sodium correction, and albumin support can be effectively combined without recourse to invasive procedures.

Finally, this case affirms the applicability of WHO dengue management principles while acknowledging the role of local clinical judgment. At our institution, dengue protocols largely mirror WHO recommendations, but decisions such as early initiation of hypertonic saline and the selective use of albumin were tailored to the patient's dynamic clinical status. These adaptations reflect a pragmatic approach that integrates global guidelines with bedside realities, ensuring both safety and efficacy. Taken together, the lessons from this case extend beyond its rarity, offering a practical model for recognizing, differentiating, and managing the convergence of DSS, severe hyponatremia, and neurological complications in pediatric dengue.

CONCLUSION

This case underscores the rare but clinically significant occurrence of Dengue Shock Syndrome complicated by severe hyponatremia and seizure in a pediatric patient. The combination of rapid plasma leakage, profound sodium decline, and neurological involvement presents a unique therapeutic challenge, requiring simultaneous management of shock, prevention of fluid overload, and cautious correction of electrolyte disturbances. Successful recovery in this case was achieved through early recognition, strict adherence to WHO fluid management principles, careful sodium correction, and a multidisciplinary approach that addressed both hemodynamic stability and neurological risk. The report reinforces the importance of routine electrolyte assessment in severe dengue, particularly in patients with neurological manifestations, and highlights that timely, individualized interventions can lead to favorable outcomes even in the most complex presentations.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

ETHICS CONSIDERATION

Written informed consent was obtained from the patient for publication of this case report, as follows the COPE and ICMJE criteria.

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AUTHOR CONTRIBUTION

TF is developing the concept of a case report, conducting a literature review, drafting the initial manuscript, collecting patients' clinical data, and documenting ancillary examinations. EMV assists in case analysis, data interpretation and provides scientific input on the manuscript draft. Performing critical revisions of the manuscript content. All authors prepare the manuscript and agree to this final version of the manuscript to be submitted to this journal.

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