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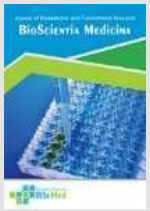

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Shock Management and Hemodynamic Monitoring of Severe Dengue with Fluid Overload: A Case Report

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A B S T R A C T

Background: Dengue shock syndrome (DSS) is a life-threatening complication of dengue infection characterized by plasma leakage, shock, and organ dysfunction. Fluid management is crucial in DSS, but fluid overload can lead to adverse outcomes. This case report highlights the challenges and strategies in managing DSS with fluid overload. **Case presentation:** An 8-year-old girl presented with severe dengue, DSS, encephalopathy, and fluid overload. She had a history of high fever, vomiting, and altered consciousness. Initial management focused on fluid resuscitation, but the patient developed signs of fluid overload. Hemodynamic monitoring using USCOM (Ultrasonic Cardiac Output Monitor) revealed low cardiac output and high systemic vascular resistance. Fluid restriction and inotropic support with epinephrine were initiated. The patient's condition gradually improved, and she was discharged after complete recovery. **Conclusion:** This case emphasizes the importance of early recognition and careful monitoring of fluid status in DSS. Hemodynamic monitoring tools like USCOM can aid in guiding fluid management and identifying complications like fluid overload. Prompt intervention with fluid restriction and inotropic support can improve outcomes in DSS patients with fluid overload.

1. Introduction

Dengue is a significant global health concern, affecting millions annually and posing a substantial risk of severe complications and mortality, particularly in tropical and subtropical regions. The disease spectrum ranges from asymptomatic infections to severe manifestations, including dengue hemorrhagic fever (DHF) and dengue shock syndrome (DSS). DSS, the most critical form of dengue, is characterized by plasma leakage, thrombocytopenia, and circulatory shock, often necessitating intensive care management. The pathophysiology of DSS involves a complex interplay of viral factors, host immune responses, and vascular dysfunction. Infection with the dengue virus triggers an immune response that can lead to increased vascular permeability, plasma leakage, and hemoconcentration. This cascade of events can culminate in shock, organ dysfunction, and, in severe cases, death.[1,2]

Fluid management is a cornerstone of DSS treatment, aiming to restore intravascular volume and maintain tissue perfusion. Intravenous fluid resuscitation is initiated to counteract plasma leakage and prevent shock. However, the judicious administration of fluids is crucial, as excessive fluid administration can lead to fluid overload, a potentially detrimental complication. Fluid overload in DSS can have several adverse consequences. It can exacerbate respiratory distress by increasing pulmonary capillary pressure and promoting pulmonary edema. Additionally, fluid overload can impair cardiac function by increasing preload and afterload, leading

to decreased cardiac output and worsening shock. Furthermore, fluid overload can delay recovery and increase the risk of complications such as acute kidney injury and prolonged hospitalization. Hemodynamic monitoring plays a pivotal role in guiding fluid management in DSS. It enables real-time assessment of the patient's circulatory status, including cardiac output, preload, afterload, and contractility. This information allows clinicians to tailor fluid therapy, optimize tissue perfusion, and detect early signs of fluid overload. Various hemodynamic monitoring tools are available, including invasive methods like pulmonary artery catheterization and less invasive techniques like echocardiography and bioimpedance analysis.[3,4]

Ultrasonic cardiac output monitor (USCOM) is a non-invasive hemodynamic monitoring device that has gained popularity in recent years. It utilizes Doppler ultrasound to measure cardiac output, stroke volume, and other hemodynamic parameters. USCOM has been shown to be accurate and reliable in various clinical settings, including critical care and perioperative medicine. In the context of DSS, USCOM can provide valuable insights into the patient's hemodynamic status and guide fluid management decisions. It can help clinicians assess the adequacy of fluid resuscitation, identify early signs of fluid overload, and monitor the response to interventions such as inotropic support and diuretics.[5,6] This case report presents an 8-year-old girl with severe dengue and DSS who developed fluid overload despite initial fluid resuscitation. The case highlights the challenges in managing fluid balance in DSS and the importance of hemodynamic monitoring in optimizing treatment and improving outcomes. The use of USCOM in this case facilitated the early detection of fluid overload and guided the adjustment of fluid therapy, ultimately contributing to the patient's recovery.

2. Case Presentation

An 8-year-old girl presented to the emergency department with a 5-day history of high-grade fever, headache, vomiting, and altered consciousness. The fever had been continuous and was accompanied by recurrent episodes of headache and joint pain. The patient also reported nausea and vomiting, with the vomitus containing black-colored material for the past three days. Additionally, she had experienced abdominal pain and had become increasingly sleepy and delirious in the days leading up to admission. There was no history of cough, cold, or bleeding from the gums, nose, or skin. The patient had received incomplete immunizations and was undernourished. Upon arrival at the emergency department, the patient appeared severely ill and was somnolent, with a Glasgow Coma Scale (GCS) score of E4M6V4. Her vital signs included a blood pressure of 116/60 mmHg, a heart rate of 148 beats per minute with a weak pulse, a respiratory rate of 24 breaths per minute, and a temperature of 37.2°C. Oxygen saturation was 99% on 2 liters per minute of supplemental oxygen. The patient's physical examination reveals a child who is severely ill and experiencing dengue shock syndrome (DSS). The presence of palpebral edema (swelling around the eyes) and crackles in the lungs suggests fluid overload, a common complication of DSS. The cold extremities and prolonged capillary refill time (CRT) are indicative of poor perfusion due to shock. An enlarged liver with a blunt edge is a typical finding in dengue infection. The absence of rashes, skin bleeding, or other hemorrhagic manifestations is noteworthy. The patient's vital signs are also consistent with DSS. The low blood pressure (116/60 mmHg) and tachycardia (148 beats per minute) reflect the body's attempt to compensate for shock. The normal respiratory rate and oxygen saturation suggest adequate respiratory function despite the fluid overload in the lungs. The nutritional assessment indicates that the patient is undernourished, which is common in children with severe dengue due to decreased appetite and fluid loss. The neurological examination reveals that the patient is somnolent (drowsy) with a GCS score of E4M6V4, indicating impaired consciousness. This could be due to dengue encephalopathy, a complication of severe dengue that affects the brain (Table 1).

Table 1. Patient's physical examination.

Physical examination	Result
General condition	Severely ill
Consciousness	Samnolen (GCS E4M6V4)
Blood pressure	116/60 mmHg (p5 81/41; p50 98/59; p90 112/73)
Heart rate	148 times/minute, weak pulse
Respiratory rate	24 times per minute
Temperature	37.2°C
Oxygen saturation	99% (with O_2 2l/minute)
Nutritional status	Undernourished
Body weight	23 kg
Body length	128 cm
Height for age	98%
Weight for height	88%
Weight for age	85%
Skin	Warm, cyanotic (-), rash (-), BCG scar (+), petechiae (-)
Head	Round, symmetric, head circumference 52 cm normocephalic
Hair	Black
Eyes	Non-pale conjunctiva, non-icteric sclera, pupil isochoric with a diameter of 3 mm/3 mm, positive and normal light reaction, doll's eyes movement normal, palpebral edema +/+
Ears	No abnormality
Nose	No nasal flare, no secretions or epistaxis
Mouth	No cyanosis, pharynx not hyperemic, T1-T1
Neck	Lymph nodes not palpable, JVP 5-2 cmH_2O, no nuchal rigidity
Chest	Symmetrical chest movement, no chest retraction
Lungs	Vesicular breath sounds, no wheezing, crackles (+) in both lungs
Heart	Normal heart borders, regular rhythm, no murmurs
Abdomen	Not distended, supple, liver 1/4-1/4, blunt edge, spleen not palpable, normal peristaltic sounds, tympanic
Genitalia	No abnormality
Pubertal status	A1M1P1
Extremities	Cold acral, CRT 3 seconds, no pitting edema. Physiological reflexes +/+, pathological reflexes -/-. Brudzinski I sign -/-, Brudzinski II sign -/-, and Kernig sign -/-

The laboratory data from 07/01/2024 reveals several abnormalities consistent with severe dengue and its complications: Hemoglobin (Hb): The hemoglobin levels are within the normal range for children (11.5-13.5 g/dL), although there is a slight decrease from 13.1 g/dL to 11.9 g/dL over the 6-hour period, which could indicate early signs of bleeding or hemodilution due to fluid resuscitation. White blood cell (WBC) count: The WBC count is significantly low (2,150/mm^3), indicating leukopenia, a common finding in dengue infection. The increase to 4,540/mm^3 at 18:00 might suggest an ongoing inflammatory response or recovery phase. Platelet count: The platelet count is critically low (19,000/mm^3), indicating thrombocytopenia, a hallmark of dengue. This predisposes the patient to bleeding complications. The slight increase to 20,000/mm^3 at 18:00 could be due to natural fluctuation or early bone marrow response. Hematocrit (Hct): The hematocrit, a measure of red blood cell concentration, is initially elevated (36%), suggesting hemoconcentration due to plasma leakage, a key feature of DSS. The decrease to 34% at 18:00 might indicate the effectiveness of fluid resuscitation or ongoing plasma leakage. Differential count: The differential count shows a predominance of neutrophils (48%) and lymphocytes (46%), which is typical in dengue infection. MCV/MCH/MCHC: These red blood cell indices are within the normal range, indicating that the anemia, if present, is not due to a

nutritional deficiency. Sodium (Na): The serum sodium level is low (129 mmol/L), indicating hyponatremia. This could be due to several factors, including fluid overload, inappropriate antidiuretic hormone secretion (SIADH), or salt loss due to vomiting. Potassium (K): The serum potassium level is within the normal range (4.0 mmol/L). Chloride (Cl): The serum chloride level is low (87 mmol/L), which often accompanies hyponatremia. Calcium (Ca): The corrected serum calcium level is within the normal range (8.6 mg/dL). Albumin: The serum albumin level is low (2.6 g/dL), indicating hypoalbuminemia. This could be due to decreased liver production, increased loss due to capillary leak, or malnutrition. Blood Random Glucose: The random blood glucose level is slightly elevated (130 mg/dL), which could be due to stress or a mild degree of insulin resistance associated with dengue infection. Overall, these laboratory findings are consistent with severe dengue and DSS, with evidence of plasma leakage, thrombocytopenia, hyponatremia, and hypoalbuminemia (Table 2).

Table 2. Hematologic and clinical chemistry laboratory data 07/01/2024.

Parameters	**07/01/2024 (12.00)**	**07/01/2024 (18.00)**
Hemoglobin	13.1 g/dL	11.9 g/dL
White blood cell	2,150/mm^3	4,540/mm^3
Platelet count	19,000/mm^3	20,000/mm^3
Hematocrit	36%	34%
Differential count	0/0/1/46/48/5	
MCV/MCH/MCHC	72/26/37	74/26/35
Sodium	129 mmol/L	
Potassium	4.0 mmol/L	
Chloride	87 mmol/L	
Calcium	7.9 mg/dL (Corrected 8,6)	
Albumin	2.6 mg/dL	
Blood random glucose	130 /dL	

The findings in Table 3 provide crucial insights into the patient's hemodynamic status and confirm the diagnosis of fluid overload: Ultrasonography: Wide inferior vena cava (IVC): The IVC is a large vein that carries deoxygenated blood back to the heart. Its diameter is an indicator of right heart preload (the amount of blood returning to the heart). A dilated IVC suggests increased preload, which is often seen in fluid overload. B-lines in the lungs: B-lines are vertical artifacts seen on lung ultrasound and are a sensitive indicator of pulmonary edema (fluid accumulation in the lungs). The presence of B-lines in this patient further supports the diagnosis of fluid overload. USCOM (Ultrasonic Cardiac Output Monitor): High FTC (Flow Time Corrected): FTC is a measure of preload. A high FTC indicates increased preload, consistent with fluid overload. Low INO (Smith-Madigan Inotropy Index): INO is a measure of cardiac contractility (the heart's ability to pump blood). A low INO suggests impaired cardiac function, which can be caused by fluid overload or other factors like myocardial dysfunction. High SVRI (Systemic Vascular Resistance Index): SVRI is a measure of afterload (the resistance the heart has to pump against). A high SVRI indicates increased afterload, which can be a compensatory mechanism in response to low cardiac output or a direct effect of dengue infection on the blood vessels. Overall, the combination of dilated IVC, B-lines in the lungs, high FTC, low INO, and high SVRI strongly suggests that the patient is experiencing fluid overload, which is compromising her cardiac function and contributing to her shock state.

Table 3. Patient's ultrasonography and USCOM findings.

Examination	Parameter	Results	Interpretation
Ultrasonography	Inferior vena cava (IVC)	Wide	Fluid overload
	Lung	B-line (+)	Fluid overload
USCOM (Ultrasonic Cardiac Output Monitor)	FTC	474 (353-415)	High (preload)
	INO	0.91 (1.56-2.32)	Low (contractility)
	SVRI	2402 (1078-1607)	High (afterload)

Based on the clinical and laboratory findings, the patient was diagnosed with Severe dengue with dengue shock syndrome (fever 6th day), fluid overload, suspected dengue encephalopathy, upper GIT (gastrointestinal tract) bleeding due to thrombocytopenia, bleeding in CVC due to thrombocytopenia. She was started on an O_2 nasal cannula 2 l/minute: Supplemental oxygen is administered to ensure adequate oxygenation, especially in the context of respiratory distress due to fluid overload. IVFD asering 5 1000cc/day = 43cc/hour: Intravenous fluid administration (IVFD) with Asering (a balanced electrolyte solution) is continued, but at a reduced rate (43cc/hour) to avoid exacerbating fluid overload. The total daily fluid intake is restricted to 1000cc. Ceftriaxone 2x1gr IV: Ceftriaxone, a broad-spectrum antibiotic, is given to address the possibility of bacterial co-infection, a common complication in dengue. Epinephrine 0.05 mcg/kgBW/minute: Epinephrine, a vasopressor and inotropic agent, is administered at a low dose to improve cardiac contractility and blood pressure, counteracting the effects of shock. Paracetamol 4x250mg IV: Paracetamol is given to manage fever and provide pain relief. Ranitidine 2x20mg IV: Ranitidine, a histamine H2-receptor antagonist, is used to reduce gastric acid production and prevent stress ulcers, which can occur in critically ill patients. Omeprazole 1x20 mg IV: Omeprazole, a proton pump inhibitor, also helps to reduce gastric acid production and protect the gastric mucosa. Compression bandage in CVC: A compression bandage is applied to the central venous catheter (CVC) insertion site to control bleeding, which is likely due to thrombocytopenia.

Table 4 summarizes the patient's clinical course over four days of hospitalization, highlighting the progression and resolution of symptoms, changes in laboratory parameters, and corresponding treatment modifications. January 8th: The patient remained severely ill, although without fever, nausea, or vomiting. She was still delirious and had bleeding from the central venous catheter (CVC). Fluid overload persisted, as evidenced by palpebral edema and crackles in the lungs. Laboratory tests showed a further decrease in hemoglobin and platelets, indicating ongoing bleeding and thrombocytopenia. The treatment plan focused on managing fluid overload, continuing antibiotic therapy, and addressing bleeding complications. January 9th: The patient showed improvement, with decreased abdominal pain and improved consciousness. However, anemia and bleeding from the CVC persisted. The fluid overload showed signs of improvement, with decreased palpebral edema and fewer crackles in the lungs. The treatment plan included initiating oral rehydration, reducing intravenous fluids, stopping epinephrine, and administering a packed red blood cell (PRC) transfusion to address the anemia. January 10th: The patient continued to improve, with resolution of fever, nausea, vomiting, and abdominal pain. She was alert and no longer bleeding from the CVC. Laboratory tests showed an increase in platelet count and improvement in hemoglobin levels. The fluid overload had significantly improved, with a resolution of palpebral edema and normal lung findings. The patient was transferred to the high care unit (HCU) and started on a regular diet. January 11th: The patient's condition further improved, with a resolution of all symptoms and normalization of laboratory parameters. She was breathing spontaneously without oxygen support and had no signs of fluid overload or bleeding. The final

assessment indicated resolution of severe dengue, improvement in dengue shock syndrome and fluid overload, and resolution of encephalopathy and upper gastrointestinal bleeding. The patient was subsequently discharged from the hospital.

Table 4. Resume follow-up.

Follow up	Subjective	Objective	Assessment	Plan
January 8th, 2024	No fever, no nausea nor vomiting, the patient remains delirious, no spontaneous active bleeding. The patient was fasting. NGT yellow-brownish residual. CVC had been inserted, bleeding from CVC.	The patient looked severely ill. GCS 14 E4M6V4, BP 116/60 (P90), HR 120x/m weak pulse, RR 22x/m, T 37,5 C, SpO_2 99% (O_2 2l/minute). Conjunctiva palpebra edema +/+. Chest movements were symmetrical, with no retraction, crackles at both lungs, the liver was palpable \u00bc - \u00bc blunt edge and spleen were not palpable, bowel sound was normal. Extremities were cold, CRT 3 seconds. No skin bleeding. Fluid balance: - 248 cc diuresis 1,3 cc/hour.	Severe Dengue with dengue Shock syndrome (fever 6th day), fluid overload, suspect encephalopathy dengue, upper GIT bleeding due to thrombocytopenia, bleeding in CVC e.c thrombocytopenia.	O_2 nasal canul 2 l/minute, IVFD asering 5 1000cc/day = 43cc/hour, ceftriaxone 2x1gr IV, epinephrine 0,05 mcg/kgBW/minute, paracetamol 4x250mg IV, ranitidine 2x20mg IV, omeprazole 1x20 mg IV, compression bandage in CVC.
January 9th, 2024	Patients with O_2 nasal canul 2lpm, no desaturation, no breathlessness. No fever, no nausea nor vomiting, abdominal pain has decreased, patient alert, there is active bleeding from CVC. NGT was inserted with a yellowish residual.	The patient looked moderately ill. GCS 15 E4M6V5, BP 112/61mmHg, HR 98x/m, strong pulse, RR 24x/m, T 36,9 C, SpO_2 99% (O_2 2l/minute). Eyes Conjunctiva anemic, palpebra edema +/+ (decrease than before). Chest movements were symmetrical, with no retraction, crackles (+) at both lungs lessened than before, the liver was palpable \u00bc - \u00bc sharp edge and spleen were not palpable, bowel sound was normal. Extremities warm acral, CRT 2 seconds. No skin bleeding. Fluid balance -84 cc, diuresis 1,1 cc/kg/hour.	Severe Dengue (Fever 7th day), dengue Shock syndrome with fluid overload (Improvement), encephalopathy dengue (Improvement), Upper GIT bleeding (Improvement), Under Nourished, anemia e.c bleeding in CVC e.c thrombocytopenia.	Starting oral intake Liquid meal 6x50 cc, IVFD Kaen 1B 600cc/24 jam, epinephrine \u2013 stop, PRC transfusion 150cc, IgM and IgG dengue test, recheck routine hematology, Ceftriaxone 2x1gr IV, paracetamol 4x250mg IV (as necessary), ranitidine 2x20mg IV \u2013 Stop. Omeprazole \u2013 stop.
January 10th, 2024	Patient\u2019s with O_2 nasal canul 2lpm, no desaturation, no breathlessness. No fever, no nausea nor vomiting, no abdominal pain, patient alert, there is no active bleeding from CVC.	The patient looked moderately ill. GCS 15 E4M6V5, BP 101/61mmHg (P50), HR 90x/m, RR 22x/m, T 36,5 C, SpO_2 99% (O_2 2l/minute). The conjunctiva was not anemic, the sclera was not icteric both pupil diameters 3mm/3mm, were reactive, and the palpebra was not edema. Chest movements were symmetrical, with no retraction, vesicular, no rales no wheezing, liver, and spleen were not palpable, and bowel sound was normal. Extremities warm acral, CRT 2 seconds. No skin bleeding. Fluid balance \u2013 792 cc diuresis 2,4 cc/kg/hours.	Severe Dengue (fever 8th day), dengue Shock syndrome with fluid overload (Improvement), encephalopathy dengue (Improvement), upper GIT bleeding (Improvement), undernourished, anemia e.c bleeding in CVC e.c thrombocytopenia (improvement).	O_2 2l/minute \u2013 stop, transfer to HCU, regular meal 1600 kkal, ceftriaxone 2x1gr IV.
January 11th, 2024	Spontaneous breathing, no desaturation, no breathlessness. No fever, no nausea nor vomiting, abdominal pain has decreased, patient alert, there is no bleeding from CVC.	The patient looked moderately ill. GCS 15 E4M6V5, BP 106/62 (P50) HR 92x/m, RR 24x/m, T 37,1 C, SpO_2 99% (Free air). Conjunctiva was not anemic, sclera was not icteric, and palpebra was not edema. Chest movements were symmetrical, with no retraction, no retraction, vesicular, no rales no wheezing, liver and spleen were not palpable, and bowel sound was normal. Extremities CRT 2 seconds. No skin bleeding.	Severe Dengue (Resolve), Dengue Shock syndrome with fluid overload (Improvement), encephalopathy dengue (Improvement), upper GIT bleeding (Improvement), undernourished, anemia (improvement).	Discharge

3. Discussion

This case report illustrates the complexities and challenges in managing severe dengue with dengue shock syndrome (DSS) complicated by fluid overload. The patient, an 8-year-old girl, presented with classic symptoms of severe dengue, including high fever, headache, vomiting, and altered consciousness. The presence of thrombocytopenia, leukopenia, and elevated liver enzymes further supported the diagnosis. The initial management of the patient-focused on fluid resuscitation to address the hypovolemic shock resulting from plasma leakage, a characteristic feature of dengue shock syndrome (DSS). This approach is consistent with standard guidelines for managing DSS, which emphasize the importance of restoring intravascular volume to maintain adequate tissue perfusion and organ function. However, the patient subsequently developed signs of fluid overload, a common complication in DSS, particularly in patients referred from other healthcare facilities. This complication underscores the critical need for meticulous fluid management in dengue patients, as excessive fluid administration can have detrimental consequences. Fluid overload in DSS can lead to a cascade of adverse effects. The excess fluid can accumulate in various organs, including the lungs, leading to respiratory distress and impaired gas exchange. It can also strain the heart, leading to decreased cardiac output and worsening shock. Additionally, fluid overload can exacerbate edema, delay recovery, and increase the risk of prolonged hospitalization and mortality. In this case, the development of fluid overload despite initial fluid resuscitation highlights the dynamic nature of DSS and the need for continuous reassessment of the patient's fluid status. The use of hemodynamic monitoring with the Ultrasonic Cardiac Output Monitor (USCOM) proved invaluable in this regard. USCOM provides real-time information on various hemodynamic parameters, including cardiac output, systemic vascular resistance, and contractility.[7,8]

The USCOM findings in this patient revealed a low cardiac output, high systemic vascular resistance, and low inotropy, indicating that the patient was in a state of cardiogenic shock with fluid overload. Cardiogenic shock is a condition where the heart cannot pump enough blood to meet the body's needs, and it can be precipitated or exacerbated by fluid overload. The high systemic vascular resistance suggests that the blood vessels were constricted, further impeding blood flow and contributing to the shock state. The low inotropy indicates impaired cardiac contractility, which can be a direct consequence of dengue infection or a result of fluid overload. The information obtained from the USCOM was instrumental in guiding the subsequent management decisions. The findings prompted a de-escalation of fluid resuscitation, as continuing aggressive fluid administration would have worsened the fluid overload and its associated complications. Instead, the focus shifted to improving cardiac function and reducing fluid overload. To achieve this, inotropic support with low-dose epinephrine was initiated. Epinephrine is a potent inotrope and vasopressor that can increase cardiac contractility, heart rate, and blood pressure. The low dose used in this case aimed to strike a balance between providing adequate hemodynamic support and minimizing potential adverse effects, such as tachycardia and arrhythmias. The patient's response to treatment was closely monitored through regular assessment of vital signs, fluid balance, and laboratory parameters. The gradual improvement in her hemodynamic status, as evidenced by the normalization of blood pressure, increased urine output, and resolution of pleural effusions and ascites, confirmed the effectiveness of the therapeutic interventions.[9,10]

The resolution of the patient's encephalopathy following the correction of fluid overload and shock strongly suggests a causal relationship. In the context of dengue shock syndrome (DSS), encephalopathy is a recognized complication, often attributed to cerebral hypoperfusion and edema. This is due to the pathophysiological mechanisms underlying DSS, where increased vascular permeability leads to plasma leakage and subsequent hypovolemia. This hypovolemia, if not promptly addressed, can result in

inadequate cerebral perfusion, leading to cerebral ischemia and subsequent encephalopathy. Furthermore, the leakage of plasma into the interstitial spaces can also contribute to cerebral edema, further exacerbating the neurological dysfunction. The patient's initial presentation with altered consciousness (GCS E4M6V4) and subsequent improvement following fluid management and hemodynamic stabilization support this hypothesis. The restoration of adequate intravascular volume and perfusion likely alleviated the cerebral hypoperfusion and reduced edema, leading to the resolution of encephalopathy. This observation underscores the critical importance of early recognition and prompt management of fluid overload in dengue patients. Fluid overload, a common complication of DSS, can have detrimental effects on multiple organ systems, including the brain. By meticulously monitoring fluid status and adjusting fluid therapy accordingly, clinicians can prevent or mitigate the development of encephalopathy and other complications associated with fluid overload. In addition to fluid management, other supportive measures played a crucial role in the patient's recovery. The administration of ceftriaxone, a broad-spectrum antibiotic, addressed the potential for bacterial co-infection, a common complication in dengue patients. Paracetamol was given for fever and pain management, while ranitidine and omeprazole, both proton pump inhibitors, were used to prevent stress ulcers, a common complication in critically ill patients.[11,12]

The bleeding from the central venous catheter (CVC), likely due to thrombocytopenia associated with dengue, was managed with a compression bandage. The patient's anemia, also a consequence of dengue-related bleeding, was corrected with a packed red blood cell transfusion. These interventions highlight the importance of a multidisciplinary approach in managing severe dengue, where various specialists collaborate to address the diverse complications that can arise. The findings of this case report align with the existing literature on the management of DSS. Several studies have emphasized the importance of early recognition and prompt treatment of fluid overload in dengue patients. A landmark study demonstrated that aggressive fluid resuscitation in the early stages of DSS can significantly reduce mortality. However, the study also cautioned against excessive fluid administration, as it can lead to complications such as respiratory distress, pulmonary edema, and prolonged hospitalization. Hemodynamic monitoring has emerged as a valuable tool in guiding fluid management in DSS. A study showed that the use of dynamic parameters, such as stroke volume variation and pulse pressure variation, can accurately predict fluid responsiveness and help clinicians avoid fluid overload. Another study emphasized the importance of using multiple hemodynamic parameters to comprehensively assess cardiac function and guide therapy in critically ill patients. The use of inotropic agents, such as epinephrine, has been shown to improve outcomes in DSS patients with shock. A study compared dopamine and norepinephrine in the treatment of shock and found that both drugs were effective in improving hemodynamics. However, norepinephrine was associated with a lower incidence of arrhythmias.[13,14]

Hemodynamic monitoring has become an indispensable tool in the management of dengue shock syndrome (DSS), particularly in guiding fluid resuscitation and identifying complications like fluid overload. The dynamic nature of DSS, with its fluctuations in vascular permeability and cardiac function, necessitates real-time assessment of hemodynamic parameters to tailor therapy and optimize outcomes. Studies have shown that the use of dynamic parameters, such as stroke volume variation (SVV) and pulse pressure variation (PPV), can accurately predict fluid responsiveness in patients with DSS. SVV refers to the change in stroke volume during mechanical ventilation, while PPV is the variation in systolic blood pressure during the respiratory cycle. These parameters reflect the interaction between cardiac function, intravascular volume, and the effects of positive pressure ventilation. In patients with DSS, hypovolemia due to

plasma leakage is a major contributor to shock. However, not all patients respond to fluid administration with an increase in cardiac output. This is where dynamic parameters come into play. By assessing SVV and PPV, clinicians can identify patients who are likely to benefit from additional fluids (fluid responders) and those who are not (fluid non-responders). This personalized approach to fluid management helps to avoid unnecessary fluid administration, which can lead to fluid overload and its associated complications. A study systematically reviewed and meta-analyzed the use of dynamic indices of fluid responsiveness in critically ill patients, including those with DSS. The study found that SVV and PPV were reliable predictors of fluid responsiveness, with high sensitivity and specificity. This suggests that these parameters can be used to guide fluid resuscitation in DSS, ensuring that patients receive the optimal amount of fluid without risking fluid overload.[15,16]

While SVV and PPV are valuable tools, a comprehensive hemodynamic assessment requires the evaluation of multiple parameters. This includes not only preload (assessed by SVV and PPV) but also cardiac output, afterload, and contractility. By monitoring these parameters, clinicians can gain a deeper understanding of the patient's cardiovascular status and tailor therapy accordingly. For example, a patient with DSS may present with low cardiac output due to hypovolemia. However, as fluid resuscitation progresses, the patient may develop signs of fluid overload, such as increased central venous pressure and pulmonary edema. In this scenario, continuing aggressive fluid administration would be detrimental. By monitoring cardiac output and other hemodynamic parameters, clinicians can identify the optimal point at which fluid resuscitation should be de-escalated and other interventions, such as inotropic support, should be considered. A consensus emphasized the importance of using multiple hemodynamic parameters to assess cardiac function and guide therapy in critically ill patients. The statement recommended a combination of static parameters (e.g., heart rate, blood pressure) and dynamic parameters (e.g., SVV, PPV) to provide a comprehensive picture of the patient's hemodynamic status. This approach is particularly relevant in DSS, where the hemodynamic profile can change rapidly and unpredictably.[17,18]

In patients with DSS who do not respond adequately to fluid resuscitation, inotropic agents may be necessary to improve cardiac contractility and support circulation. Epinephrine, a catecholamine with both alpha- and beta-adrenergic effects, is commonly used in this setting. Epinephrine increases cardiac contractility and heart rate, leading to improved cardiac output. It also causes vasoconstriction, which can help to maintain blood pressure in the setting of shock. However, the use of epinephrine should be carefully monitored, as it can also increase myocardial oxygen demand and potentially worsen myocardial dysfunction. A study compared dopamine and norepinephrine in the treatment of shock and found that both drugs were effective in improving hemodynamics. However, norepinephrine was associated with a lower incidence of arrhythmias, making it a potentially safer option in some patients. The choice of inotropic agent and its dosage should be individualized based on the patient's specific hemodynamic profile and clinical condition. Close monitoring of hemodynamic parameters is essential to assess the response to therapy and adjust the dosage as needed.[19,20]

4. Conclusion

This case report demonstrates the successful management of severe dengue with DSS and fluid overload through a combination of fluid restriction, inotropic support, and supportive care. The use of hemodynamic monitoring played a crucial role in guiding therapy and preventing complications. This case highlights the importance of early recognition and prompt intervention in managing fluid overload in dengue patients to improve outcomes.

5. References

1. World Health Organization. Dengue and severe dengue. 2022.
2. Katzelnick LC, Gresh L, Halloran ME, Mercado JC, Kuan G, Gordon A, et al. Antibody-dependent enhancement of severe dengue disease in humans. Science. 2017; 358(6365): 929-32.
3. Pang J, Lee V, Yan H, Wilder-Smith A. Clinical progression of dengue and its impact on the healthcare system: a systematic literature review. BMC Infect Dis. 2020; 20(1): 1-13.
4. Srikiatkhachorn A, Nimmannitya S, Permpikul P, Kittigul L, Yoksan S, Bhamarapravati N. Dengue hemorrhagic fever: a study of the clinical course and pathophysiologic features. Southeast Asian J Trop Med Public Health. 1980; 11(3): 420-33.
5. Halstead SB. Dengue. Lancet. 2007; 370(9599): 1644-52.
6. Yacoub S, Werer C, Becker JU, Fischer J, Bogner V, Martin E, et al. Hemodynamic coherence during fluid challenge in patients with septic shock. Crit Care Med. 2018; 46(1): e11-e9.
7. Cecconi M, De Backer D, Antonelli M, Beale R, Bakker J, Hofer C, et al. Consensus on circulatory shock and hemodynamic monitoring. Task force of the European Society of Intensive Care Medicine. Intensive Care Med. 2014; 40(12): 1795-815.
8. Ranjit S, Kissoon N. Dengue hemorrhagic fever and shock syndromes. Pediatr Crit Care Med. 2011; 12(1): 90-100.
9. Wills BA, Dung NM, Loan HT, Tam DT, Thuong TC, Thuy TT, et al. Comparison of three fluid solutions for resuscitation in dengue shock syndrome. N Engl J Med. 2005; 353(9): 877-89.
10. Lampah DA, Simmons CP. The dynamic immune response to dengue virus infection. Nat Rev Immunol. 2019; 19(1): 11-22.
11. Yacoub S, Reinhart K, Wolff G, Uddin M, Bein B. Assessment of dynamic indices of fluid responsiveness in critically ill patients: a systematic review and meta-analysis. Crit Care. 2015; 19(1): 1-14.
12. De Backer D, Biston P, Devriendt J, Madl C, Chochrad D, Aldecoa C, et al. Comparison of dopamine and norepinephrine in the treatment of shock. N Engl J Med. 2010; 362(9): 779-89.
13. Ventura AM, Shieh HH, Bousso A, Annane D. Catecholamine use in cardiogenic shock: insights from a large multinational cohort study. Crit Care. 2018; 22(1): 1-11.
14. Phu NH, Tuan TV, Hien TT, Dung NT, Chau NV, Thuy TT, et al. Clinical features and prognostic factors of dengue shock syndrome in Vietnamese children: a multi-center study. BMC Infect Dis. 2012; 12(1): 1-9.
15. Kalayanarooj S, Vaughn DW, Nimmannitya S, Green S, Suntayakorn S, Kunentrasai N, et al. Early clinical and laboratory indicators of acute dengue illness. J Infect Dis. 1997; 176(2): 313-21.
16. Guzman MG, Halstead SB, Artsob H, Buchy P, Farrar J, Gubler DJ, et al. Dengue: a continuing global threat. Nat Rev Microbiol. 2010; 8(12s): S7-S16.
17. Simmons CP, Farrar JJ, Nguyen vV, Wills B. Dengue. N Engl J Med. 2012; 366(15): 1423-32.
18. Thomas SJ, Rothman AL. Immunopathogenesis of dengue disease and dengue vaccines. Curr Top Microbiol Immunol. 2011; 338: 21-36.
19. Rothman AL. Immunity to dengue virus: a tale of original antigenic sin and tropical cytokine storms. Nat Rev Immunol. 2011; 11(8): 532-43.

20. Screaton G, Mongkolsapaya J, Yacoub S, Roberts C. New insights into the immunopathology and clinical spectrum of dengue. Nat Rev Immunol. 2015; 15(12): 745-755.