Thrombosis of the inferior vena cava and acute kidney injury in dengue shock syndrome: A rare case with unique nursing challenges

Nurhasan Agung Prabowo[1,3*], Yulia Sari[2], Desy Puspa Putri[1, 3], Sopia Nur Habibah[1], Azkia Rachmah[1], Hartono Hartono[3]

[1] Internal Medicine Department, Faculty of Medicine, Universitas Sebelas Maret, Surakarta, Indonesia
[2] Parasitology Department, Faculty of Medicine, Universitas Sebelas Maret, Surakarta, Indonesia
[3] Universitas Sebelas Maret Hospital, Universitas Sebelas Maret, Surakarta, Indonesia

Abstract

Background: Dengue Shock Syndrome (DSS) is a severe complication of dengue fever that can lead to life-threatening conditions such as thrombosis and acute kidney injury (AKI). These rare complications highlight the importance of early recognition and multidisciplinary management to improve clinical outcomes.
Purpose: This case study aims to report and discuss the clinical management and outcomes of a patient with DSS complicated by inferior vena cava thrombosis and AKI, focusing on nursing interventions and therapeutic strategies.
Methods: This manuscript reports a case by utilizing the patient's baseline data, clinical progression, and treatment outcomes. A middle-aged male patient presented to the emergency department with high fever, shock, and signs of plasma leakage. Laboratory and imaging findings confirmed DSS with AKI and inferior vena cava thrombosis. The patient received fluid resuscitation, inotropic support, anticoagulation therapy, and multidisciplinary care, including close nursing monitoring.
Results: After seven days of hospitalization, the patient showed significant improvement, with resolution of shock, normalization of platelet count, and recovery of renal function. Comprehensive nursing care, including vital sign monitoring, medication management, and patient education, played a pivotal role in the recovery process. At a follow-up visit on day 12, the patient demonstrated full recovery with no complications.
Conclusion: This case highlights the importance of early diagnosis and a multidisciplinary approach, including cautious anticoagulation therapy, to effectively manage DSS with thrombosis and AKI. Integrating nursing interventions and strict monitoring were crucial to achieving positive clinical outcomes. This report provides valuable insights into managing similar cases in endemic areas.

Keywords: dengue shock syndrome; thrombosis; acute kidney injury; inferior vena cava; anticoagulants

Introduction

Dengue shock syndrome is a severe form of dengue infection caused by the dengue virus transmission through an infected Aedes mosquito. Each year, there are an estimated 50 to 100 million cases of dengue fever, as reported by the World Health Organization (WHO). Out of these cases, 500,000 go on to develop DHF, leading to 22,000 deaths, mostly children (Sanyaolu, 2017). Indonesia ranked first in deaths due to DHF with 22.55 per 100,000 person-years (Harapan et al.., 2019). Due to the mortality and endemic that occurs, it is important to understand the complications of infection with dengue fever,

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***Corresponding author**

Nurhasan Agung Prabowo*
Internal Medicine Department, Faculty of Medicine, Universitas Sebelas Maret, 57126, Surakarta, Indonesia; Address: Jalan Kolonel Sutarto, Jebres, Surakarta City, Central Java, Indonesia, 57126; Phone: +6282328010430 ;E-mail: dr.nurhasan21@staff.uns.ac.id

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including shock, acute renal failure and thrombotic events. Dengue dengue has been associated with glomerular damage and the presence of antigens in tubular epithelial cells. Epidemiological data show that AKI develops in about 13.3% of cases of dengue fever (Khalil et al.., 2012; Oliveira & Burdmann, 2015).

Thrombosis of the inferior vena cava (IVC) is a poorly understood condition with many clinical manifestations. IVC thrombosis is a severe condition with a high mortality rate. Vena cava thrombosis (presumptive IVCT) accounted for just 1.3 per cent of all hospitalized patients diagnosed with venous thrombosis (Lin et al.., 2021). Virchow's Triad can explain the mechanism that leads to IVC thrombosis. This Triad comprises blood flow stasis, endothelial damage, and hypercoagulability (Hollingsworth & Mead, 2022).

In DHF, plasma leakage occurs. Release of chemical cascades during infection can cause activation and contraction of actin filaments of endothelial cell capillaries. The condition make the linkage proteins between endothelial cells enter the cell widen the gap between cells, and then cause plasma leakage. The combination of these mechanisms causes dengue shock syndrome (Ministry of Health Indonesia, 2021). Dengue shock syndrome, accompanied by acute kidney injury and thrombus in the inferior vena cava, is fatal. The pathophysiology of AKI in dengue involves a complex interplay of direct viral effects, hemodynamic changes, and secondary complications such as rhabdomyolysis. The resultant renal failure can be fatal due to its association with severe disease manifestations and the complications that arise from impaired renal function. Patients with acute kidney injury (AKI) may experience exacerbated risks due to altered pharmacokinetics and the potential for increased bleeding complications. Management is tricky because anticoagulants such as heparin can induce bleeding and thrombocytopenia. Anticoagulants, such as low-molecular-weight heparin (LMWH) or unfractionated heparin, are often indicated in patients with confirmed IVC thrombosis. However, the decision to initiate anticoagulation must be weighed against the risk of bleeding, particularly in patients with severe thrombocytopenia. Here, we describe our cases and the successful management of this patient with the administration of heparin and strict monitoring of signs of bleeding. The role played by health professionals, especially nurses, in the early diagnosis, intensive surveillance and management of these challenging complications in cases such as Dengue Shock Syndrome (DSS) is crucial. They are responsible for vital sign monitoring and clinical decision-making through communication with other disciplines. They also have an essential role in maintaining fluid, watching for signs of bleeding, and medication administration in therapy success.

In addition, the biopsychosocial implications of nursing practice in DSS cases need to be considered. In addition to establishing effective health systems, continuous education and training for healthcare personnel are also required to respond to the challenges posed by tropical diseases, especially DSS, in areas where it is endemic, like Indonesia. However, increasing the abilities of healthcare providers to identify and mitigate DSS complications like inferior vena cava thrombosis and acute kidney injury will substantially improve care quality and clinical outcomes. The purpose of this case report is to describe and discuss an uncommon but essential presentation of dengue shock syndrome complicated by inferior vena cava thrombosis and acute renal failure. We hope this report can provide documentation of these severe complications, increase diagnostic awareness, improve treatment strategies and help guide clinical outcomes so that future management can be established with data from similar cases.

Ethical consideration

This case report was conducted with the patient's informed consent, ensuring that the patient understood the purpose and scope of sharing medical information for educational and research purposes. The report maintains the anonymity and confidentiality of the patient's data. Institutional guidelines were followed to ensure ethical compliance in reporting this case, and no harm was posed to the patient during data collection and publication. The study adhered to the ethical principles outlined in the Declaration of Helsinki regarding medical research involving human subjects.

Case Presentation

A middle-aged male patient presented to the emergency department with complaints of high fever, weakness, nausea, vomiting, and epigastric pain that had persisted for five days. He also reported reduced urination, with no urination in the last 10 hours. Physical examination revealed hypotension (blood pressure: 80/50 mmHg), tachycardia (pulse: 110 beats/min), respiratory rate of 24 breaths/min, a temperature of 38°C, petechiae on the hands and feet, mild hepatomegaly, cold extremities, and epigastric tenderness.

Initial laboratory tests showed thrombocytopenia (80 × 10^3/μL), hematocrit of 30%, leukocytosis (13.51 × 10^3/μL), elevated creatinine (3.2 mg/dL), and urea (113 mg/dL). Dengue serology was positive for IgM and IgG, confirming dengue infection. Based on clinical and laboratory findings, the patient was diagnosed with dengue shock syndrome (DSS) complicated by acute kidney injury (AKI) and inferior vena cava thrombosis.

The patient was admitted to the intensive care unit and received immediate fluid resuscitation with Ringer's lactate, followed by inotropic support (epinephrine) due to persistent shock. Additional therapies included antibiotics (ceftriaxone), proton

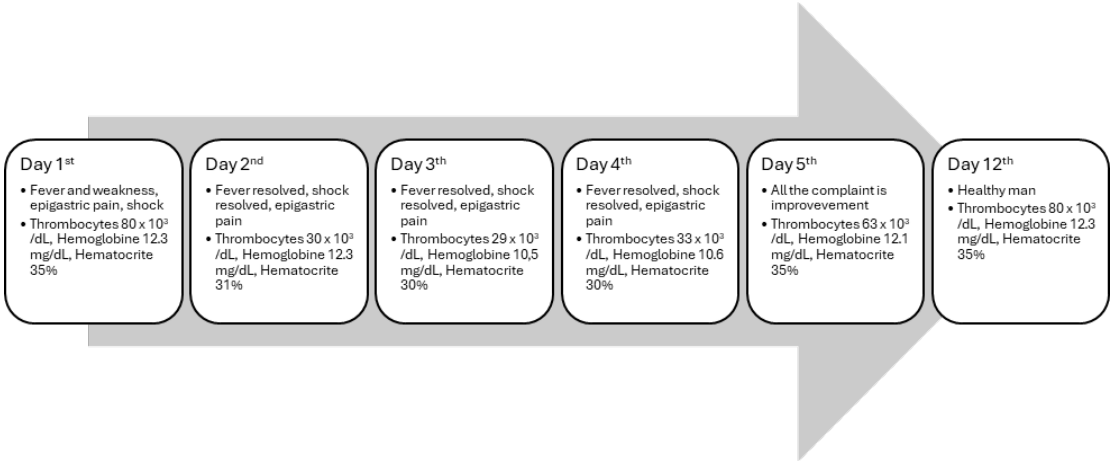

Figure 1. Scheme of the patient's disease course during the stay in the hospital. On the fifth day of treatment, the patient is discharged, and on the 12th day, the patient makes a polyclinic visit with data on improvements in clinical and laboratory conditions

Figure 2. Abdominal ultrasonography of the patient shows that the right kidney (RD) and left kidney (RS) size and normal eco structure, and the boundaries of the cortex and medulla are clear. An isolusen image in the veena cava inferior with a smokey appearance that indicates a picture of the vena cava inferior thrombus

pump inhibitors (omeprazole), methylprednisolone, anticoagulation with unfractionated heparin, and supportive measures such as sucralfate and ondansetron.

On the second day of hospitalization, ultrasound imaging revealed normal kidney structure but showed a smokey appearance in the inferior vena cava, indicative of thrombosis. Over the next few days, the patient's condition gradually stabilized. Fever resolved, platelet counts improved, and renal function began to normalize. By the fifth day, his platelet count increased to 33 × 10^3/μL, and his hematocrit stabilized at 30%.

Further evaluation on the seventh day showed that the initial shock was resolved, and the patient's general condition, vital signs, and platelet levels improved. On the last day of treatment, platelet levels were 63 x 103 per dL and 35% hematocrit, as illustrated in Figure 1. On the seventh day, the patient was discharged in a stable condition. He was prescribed warfarin (2 mg/day) for continued anticoagulation therapy and was advised to follow up for monitoring. At a follow-up visit on the twelfth day, the patient reported no complaints, and laboratory tests showed normal platelet (434 × 10^3/μL) and creatinine levels (0.75 mg/dL), confirming

full recovery.

Abdominal pain in patients with dengue infection gives rise to many possible causes. In the anamnesis, there is no previous history of gastritis. Although most causes of abdominal pain in dengue are due to the effect of dr hepatomegaly, there are still many other causes, such as cholecystitis or pancreatitis. Ultrasonography in the abdomen was performed to exclude it. Therefore, it is necessary to have supporting examinations for a decrease in the frequency of urination, and an increase in creatinine can signal the presence of acute renal failure. For further reasoning, an abdominal ultrasound examination is performed to see abdominal to see if there is kidney damage or not, and a re-creatinine test is after the patient recovers. Ultrasonography examination of the abdomen shows a picture of the presence of the right kidney, and the left kidney is still normal. There is no visible picture of obstruction in the kidney. The kidney's normal size and structure and the renal cord's boundaries are still clear. Another image found is an isolated image of the inferior vena cava and a smokey appearance image that supports the presence of inferior vena cava thrombus, as in Figure 2.

Fever with thrombocytopenia, shock conditions, plasma signs of leakage such as increased hematocrit, and positive dengue antibody examination indicate that dengue shock syndrome occurs in the patient. The abdominal ultrasound results show the presence of inferior thrombus vena cava. Angiography examination cannot be done because of contraindications such as acute renal failure. The subsequent investigation was the D Dimer examination obtained a result of D Dimer 1567 ng/mL (normal levels <500 ng / mL). These developments in the diagnosis of inferior thrombus vena cava. Creatinine re-examination becomes normal, and kidney ultrasound results are obtained by normal kidneys, thus supporting the diagnosis of acute renal failure.

Patients get dengue treatment with warning signs guidelines (Ministry of Health Indonesia, 2021). The therapy is administering ringer lactate fluid seven ccs per kg body weight (kgBW) in 1 hour. The evaluation shows that the patient is still in a state of shock. After that, ringer lactate 30 ccs / kgBW is carried out in 30 minutes. The condition was still so, so the epinephrine inotropic was given at 0.1 mcg per kgBW per minute. Another drug administration carried out is ceftriaxone antibiotic 2 g per day i.v. (intravenously), omeprazole 40 mg per 12 hours i.v., sucralfate ten cc per 8 hours orally, ondansetron 8 mg per 8 hours i.v., Heparin 80 unit per B.W and 18 unit per kgBW per hour methylprednisolone 62.5 mg/dL. After discharge, the patient takes warfarin 2 mg per day.

Nurses played a vital role during the treatment in managing the patient's care and facilitating recovery. They continuously monitored the patient's vital signs, including blood pressure, heart rate, respiratory rate, and temperature, to detect any signs of hemodynamic instability or worsening shock. Nurses ensured the accurate administration of intravenous fluids, such as Ringer's lactate, to manage shock and address dehydration due to plasma leakage. They were responsible for administering all prescribed medications, including epinephrine, ceftriaxone, omeprazole, heparin, methylprednisolone, and other supportive therapies, while closely monitoring for side effects and therapeutic responses. Given the risk of bleeding associated with thrombocytopenia and anticoagulation treatment, nurses carefully observed for signs of bleeding, such as bruising or petechiae. They ensured the safe administration of heparin and, later, warfarin. They also provided emotional support to the patient and family, addressing their concerns and explaining the care plan to enhance understanding and compliance. Additionally, nurses ensured the patient maintained proper nutrition and hydration, adjusting dietary recommendations as the patient's condition improved. They served as a crucial link between the patient and the medical team, promptly reporting changes in the patient's condition to facilitate timely adjustments to the treatment plan. Toward the end of the hospital stay, nurses educated the patient about the safe use of warfarin, including dietary considerations and the importance of follow-up visits, while ensuring the patient understood their discharge plan. Their comprehensive care and attention to detail significantly contributed to the patient's recovery.

Results

After one week of hospitalization, the patient was discharged. One week later, the patient visits the outpatient polyclinic. At the examination of the visit, the patient did not feel any complaints. In the laboratory, tests obtained normal hematocrit (31%), normal platelets (434 x 103 per dL), and normal creatinine levels (0.75 mg / dL).

During the five days of hospitalization, nursing care was pivotal in the patient's recovery. Nurses continuously monitored vital signs, including blood pressure, pulse, respiratory rate, and temperature, to detect any signs of hemodynamic instability or worsening shock. They administered intravenous fluids such as Ringer's lactate to manage dehydration and plasma leakage and ensured precise delivery of medications, including epinephrine, ceftriaxone, omeprazole, methylprednisolone, and anticoagulants like heparin.

Due to the patient's thrombocytopenic state, close observation for bleeding complications, such as bruising or petechiae, was maintained. Nurses provided timely feedback to the medical team, enabling adjustments to the treatment plan when necessary. They also supported the patient emotionally, offering reassurance and educating him about the disease and its treatment. By the fifth day, the patient showed significant improvement, with resolution of shock, stabilization of hematocrit

levels, and an increase in platelet count to 33 × 10^3/µL.

On the seventh day, the patient was discharged in stable condition with clear instructions for home care, including warfarin management and follow-up visits. Nursing interventions were instrumental in ensuring the patient's smooth recovery and preparedness for discharge.

Discussion

Various mechanisms have been suggested to elucidate the pathogenesis of renal impairment in dengue fever. In patients with this condition, the capillaries leak, and so some fluid seeps from the bloodstream, leading to shock with diminished renal perfusion and acute tubular necrosis. Severe hypotension, hemolysis, rhabdomyolysis, and shock have all been identified as immediate contributory causes of AKI in dengue from the case series cited above. In addition, cases of AKI have been seen that need to be clearly explained (Khalil et al.., 2012; Oliveira & Burdmann, 2015).

There are several mechanisms through which dengue could result in thrombotic events. The dengue virus disrupts this anti-coagulation pathway by downregulating the formation of the thrombomodulin-thrombin-protein C complex. It thereby leads to impaired production of activated protein C, which, together with reduced concentrations in proteins C and S as well as antithrombin III, has been associated with DSS featuring capillary leakage. Although thrombotic complications may occur at all levels of dengue severity from DF to DSS, they are more commonly associated with DSS cases. Long-lasting shock in Dengue Shock Syndrome (DSS) may induce and accelerate the onset of disseminated intravascular coagulation (DIC) or microthrombi. These are relatively common conditions, but they have not usually been associated with thrombosis of larger vessels. But the more severe the dengue infection, the greater its association with thrombotic events. Furthermore, the dengue virus upregulates thrombomodulin expression in endothelial cells and activates them into a prothrombotic state (Azeredo et al.., 2015; Nugraha et al.., 2022).

Reports of dengue cases with venous thrombosis have been previously reported in the femoral vein. This case has a therapeutic dilemma due to severe thrombocytopenia 12.000/dL and thrombus in the iliofemoral veins. After administering unfractionated heparin at a dose of 500 IU per hour and re-examining the next day, platelets increased to 58.000/dL (Ranasinghe et al.., 2020). Other case reports mention that dengue coincides with portal vein thrombosis, in which 7000/dL close thrombocytopenia occurs. Heparin's low molecular content in these patients is pronounced after platelet levels above 70000/dL (Gonzalez et al.., 2022). Da Costa et al. reported that 5.4% of thrombotic events in critically ill dengue cases include four DVTs and one mesenteric vein thrombosis (da Costa Campos et al.., 2012). Other reports showed the dengue complexity as in patients with dengue shock syndrome (DSS), acute liver failure, kidney injury, infective endocarditis and deep vein thrombosis were present. The management in these cases entailed 60 mg of enoxaparin subcutaneously every 12 hours along with 5 mg of warfarin daily. The warfarin dose was titrated to 6 mg daily to achieve an international normalized ratio (INR) between 2 and 3. When the INR was in range, enoxaparin was stopped, and warfarin continued for 3 months (Samarasekara & Munasinghe, 2018).

This Case emphasizes how healthcare professionals, particularly nurses, should be alert to such serious sequelae of DSS as inferior vena cava thrombosis and acute kidney injury. Nurses are the first line of defence for monitoring patient status, initiating timely interventions, and modifying care plans to meet the constantly changing needs of critically ill patients. During the treatment, the nurses played a crucial role in ensuring the success of the therapy by closely monitoring for signs of bleeding, a critical risk in managing dengue shock syndrome with thrombocytopenia and anticoagulation therapy. They carefully observed for bruising, petechiae, or other indications of haemorrhage while ensuring precise administration of anticoagulants like heparin and warfarin. Their vigilance and timely reporting of any changes to the medical team allowed for prompt adjustments in treatment, ultimately preventing complications and supporting the patient's recovery. This meticulous attention to bleeding risk was key to the patient's successful outcome.

Conclusions

The successful management of this case highlights the importance of early recognition of complications, timely administration of fluid resuscitation and inotropic support, and the cautious use of anticoagulants under close monitoring for bleeding, emphasizing a multidisciplinary approach to ensure optimal outcomes in severe dengue shock syndrome. Case in point is with the right treatment, the patient can recover from emergency as we see in this patient. The use of heparin in thrombocytopenic conditions represents a challenging yet essential intervention, requiring meticulous monitoring to balance the benefits of preventing thrombosis against the risks of bleeding.

Declaration of Interest

The authors declare no conflicts of interest related to this case report.

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Data Availability

The data supporting this case report's findings are included in the document. All other data will be available upon reasonable request to the correspondent author within patient confidentiality standards.

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