CASE REPORT

Dengue Shock Syndrome Leading to Multi Organ Dysfunction Syndrome (MODS) and Fatal Outcome: A Case Report

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Abstract

Dengue shock syndrome (DSS) is a severe and life-threatening consequence of dengue fever, particularly in endemic places like Karachi, Pakistan. This report presents a case of a 30-year-old female patient with DSS who rapidly developed Multi-Organ Dysfunction Syndrome (MODS) and had a fatal outcome. The patient was presented with thrombocytopenia, petechiae, and a high-grade fever and was diagnosed with a positive NS1 antigen test. Within 48 hours, she developed septic shock, Acute Respiratory Distress Syndrome (ARDS), Disseminated Intravascular Coagulation (DIC), and Acute Kidney Damage (AKI) despite early measures such as fluid resuscitation and vasopressor therapy. The main findings were anemia, coagulopathy, and severe thrombocytopenia. After undergoing rigorous fluid therapy, vasopressors, and intensive care unit care, the patient expired on the sixth day due to refractory shock. This instance highlights how crucial it is to identify DSS early and treat it thoroughly to stop its progression to MODS and fatality.

Keywords: *Dengue Shock Syndrome (DSS), Multi-Organ Dysfunction Syndrome (MODS), dengue fever, septic shock, Acute Respiratory Distress Syndrome (ARDS).*

INTRODUCTION

Dengue Shock Syndrome (DSS) which is characterized by severe hypotension, hypoperfusion, and plasma leakage, is a potentially fatal consequence of dengue fever that may end in Multi-Organ Dysfunction Syndrome (MODS), which is the failure of two or more organ systems. Dengue fever is a viral infection that is transmitted by the Aedes mosquito. It is caused by one of the dengue virus's four serotypes (DENV1-4). In Pakistan, dengue is a serious public health issue, especially in endemic urban areas like Karachi. The dengue virus is a member of the Ribonucleic Acid (RNA) viral family Flaviviridae. The most serious manifestation of dengue infection is DSS, which can have a 20% fatality rate if left untreated. Pakistan sees yearly outbreaks, particularly during the monsoon season, according to local epidemiological data, with thousands of cases recorded each year [1]. A sudden drop in blood pressure that leads to shock and organ failure is a characteristic of DSS. Early ICU admissions and vigorous fluid administration in DSS are important for enhanced patient outcomes [1].

The common signs of a dengue infection include a high fever, rigors, chills, body pains, and a temporary macular rash. The World Health Organization (WHO) states that severe dengue is characterized by severe hemorrhage, organ damage, or both, whereas DSS is characterized by severe plasma leakage that results in shock. To identify complications early, tests like hematocrit, platelet count, coagulation profile, and liver enzymes need to be regularly checked. Serious side effects, including acute hepatic failure, renal failure, encephalitis, encephalopathy, neuromuscular and ocular abnormalities, seizures, and cardiomyopathy, can, however, very rarely result from a severe dengue infection [1]. Approximately 2.5-3 billion people worldwide live in 112 countries where dengue is prevalent. An estimated 50 to 528 million people become infected each year, leading to approximately 0.5 million hospitalizations [2]. To lower mortality and morbidity, DSS must be identified early and addressed. This case emphasizes how critical it is to identify early warning signs and use aggressive treatment methods for DSS. This case report aims to recognize the risk of rapid deterioration and implement prompt interventions to stop the development of multi-organ failure. Clinicians can increase survival rates in critical dengue cases by spotting clinical deterioration early.

CASE PRESENTATION

A 30-year-old female patient resident of Karachi presented to the emergency department with complaints of fever, nausea, and decreased oral intake for 4 days. There was no past medical history or recent history of endemic areas. The patient's initial examination showed a fever of 39.5°C, blood pressure (BP) of 90/60 mmHg, heart rate (HR) of 120 beats per minute, respiration rate (RR) of 28 breaths per minute, and petechia scattered throughout the body which indicated hemodynamic instability. At the time of admission, her Glasgow Coma Scale (GCS) score was 15. Laboratory tests revealed leukopenia (WBC count: 2,800/µL), increased liver enzymes (ALT: 450 IU/L), thrombocytopenia (platelet

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count: 45,000/μL), and hemoconcentration with a hematocrit of 52%. Polymerase chain reaction (PCR) results for the DENV-2 serotype and the dengue non-structural protein 1 (NS1) antigen confirmed dengue infection.

The patient's hypotension persisted despite rigorous fluid resuscitation in the emergency department. Within 24 hours she was transferred to the Intensive Care Unit (ICU) for further treatment. Her condition quickly worsened, requiring the start of vasopressors to maintain perfusion after her blood pressure dropped to 70/40 mmHg and her heart rate increased to 140 beats per minute. She was started on aggressive intravenous fluid therapy, broad-spectrum antibiotics, and electrolyte control. Over 48 hours, the patient developed acute renal injury (creatinine: 3.5 mg/dL), hepatic dysfunction (total bilirubin: 5.8 mg/dL), and severe respiratory distress for which the patient was taken on Non-Invasive Ventilation (NIV). Continuous Renal Replacement Therapy (CRRT) was started within 48 hours to treat renal damage. As her respiratory condition deteriorated with evidence of pulmonary edema and hypoxemia, the patient was electively intubated. Despite the maximum supportive treatment, the patient ended up in multi-organ failure. She developed Disseminated Intravascular Coagulation (DIC) and required Multiple blood transfusions, such as platelets and fresh frozen plasma. According to the timeline of events, the patient suffered from refractory shock and deteriorating MODS, which ultimately resulted in cardiac arrest, within 72 hours after being admitted to the intensive care unit. After unsuccessful efforts at resuscitation, the patient expired. Refractory shock as a result of severe DSS and MODS was identified as the ultimate cause of mortality.

Etiology

Dengue 1, 2, 3, and 4 are the four serologically distinct viral serotypes that constitute dengue. Since they share structural and pathogenic similarities, any of the serotypes can cause severe forms of illness, though serotypes 2 and 3 have been linked to a higher number of severe cases and fatalities. These viruses are made up of spherical particles that have a diameter of 40 to 50 nm. They are found in the RNA genome of the viruses as well as the structural proteins of the envelope (E), membrane (M), and capsid (C). Other non-structural (NS) proteins, such as NS1, NS2A, NS2B, NS3, NS4A, NS4B, and NS5-3, are also present in them. The dengue viruses are members of the Flaviviridae family, specifically the genus Flavivirus [3].

PATHOPHYSIOLOGY

The pathophysiology of DSS involves viral factors, immune response, and vascular integrity.

Viral Infection

When an Aedes mosquito bites a victim carrying the dengue virus, the virus mostly targets monocytes, macrophages, and dendritic cells. After the injection, the virus infects these cells, causing viral replication and the release of inflammatory mediators and cytokines such as interleukin-6 (IL-6), interferons, and tumour necrosis factor-alpha (TNF-α) [4].

Immune Response

The pathophysiology of dengue fever syndrome heavily relies on the host's immunological response to the virus. Sickness is characterized by two main phases: the critical period and the febrile phase. Viral replication is active during the febrile phase, which is characterized by a high temperature, myalgias, and rash. During the crucial phase, which usually coincides with defervescence, immunological dysregulation takes place [4].

Vascular Integrity

Increased vascular permeability that results in plasma leakage is one of the characteristics of DSS. The integrity of the endothelium barrier is thought to be compromised by immune mediators like cytokines and vasoactive chemicals (like bradykinin) that cause endothelial dysfunction. Hemoconcentration and hypovolemia are caused by the extravasation of fluid and plasma proteins into extravascular compartments due to this disturbance [4].

MODS can arise from impaired tissue perfusion and reduced microcirculation in DSS. Particularly vulnerable organs include the brain, kidneys, liver, and lungs.

CLINICAL PRESENTATION

The clinical presentation of DSS is defined by distinctive characteristics during the disease's different phases.

Febrile Phase

Dengue fever usually has a sudden start and takes four to ten days to incubate. Sudden, high-grade fever that frequently reaches temperatures exceeding 104°F (40°C) characterizes the febrile phase. Severe headache, retro-orbital discomfort, myalgias, arthralgias, and widespread malaise are some of the accompanying symptoms. Additionally, patients may develop a maculopapular rash, moderate gastrointestinal symptoms including nausea and vomiting, and mild hemorrhagic signs like petechiae or easy bruising [5].

Critical Phase

The disease enters a critical phase that lasts for three to seven days after the fever. The greatest risk of developing severe dengue, including DSS, is linked to this period, which frequently occurs around the time of defervescence. While some patients exhibit warning signals of vascular leakage and impending shock during this time [5].

Warning Signs

Some warning signs are intense stomach ache, frequent vomiting, clinical fluid accumulation (ascites, pleural effusion), mucosal bleeding (such as epistaxis and gum bleeding), agitation, hepatomegaly and a sharp rise in hematocrit and a sharp fall in platelet count [6].

Multi-Organ Dysfunction

Multi-organ dysfunction can include acute renal injury with decreased urine output, respiratory distress due to pulmonary edema, neurological symptoms such as altered consciousness seizures, and hepatic impairment indicated by increased liver enzymes (AST/ALT) [7].

DISCUSSION

Dengue has grown rapidly in the past few years and is now a major worldwide health concern. Recent years have seen an increase in dengue infections due to haphazard urbanization, which involves uncontrolled infrastructure development and subpar sanitary amenities. This has created an abundance of mosquito breeding grounds [8]. In line with the study's objectives of evaluating environmental risk factors, this case demonstrates how dengue has increased due to growing urbanization.

Rapid and accurate confirmation of dengue infections is essential to the effectiveness of vector prevention measures and enhances the disease surveillance program. In this case, the NS1 Antigen Test allowed early diagnosis by identifying the dengue virus's non-structural protein 1 (NS1) antigen in the acute stage of sickness. Furthermore, serological assays for dengue-specific IgM and IgG antibodies also played a role in confirming both current and previous dengue infections. However, despite early diagnosis, the patient's rapid progression to DSS contrasts with the classic, indicating additional investigation.

IgG antibodies last longer and are useful for retroactive diagnosis, but IgM antibodies are only detected during the acute phase. Also, when the sickness is still in its early stages, viral RNA can be found using molecular tests such as Polymerase Chain Reaction (PCR). PCR helps confirm dengue infection because of its excellent sensitivity and specificity [8]. Numerous factors, including the unique characteristics of each patient, the type of virus, or other unidentified environmental factors that require more research, could have contributed to the abnormally rapid progression to DSS in this case.

Besides the above diagnostic measures, some studies have proposed other investigations as well to diagnose dengue fever. A major laboratory finding in severe cases, including DSS is thrombocytopenia which is a hallmark finding in dengue infection. Also, severe dengue is associated with elevated hematocrit levels as a result of plasma leakage. The rapid deterioration observed in this case might have been caused by plasma leakage, which increases vascular permeability. Identification of plasma leakage is aided by serial monitoring of hematocrit levels [9]. Understanding why some individuals experience severe DSS symptoms more promptly than others requires understanding this pathophysiological mechanism.

Due to financial constraints and the need for rapid life-saving treatments, a cytokine work-up was not conducted in this case, even though it may have offered further information on inflammatory reactions that might have contributed to the patient's quick decline. Although cytokine profiling is not usually included in normal dengue management, it could provide important information in the future for instances of DSS that present atypically.

Aspartate Aminotransferase (AST) and Alanine Aminotransferase (ALT) values are frequently elevated as a result of liver dysfunction. In situations of severe dengue, Prothrombin Time (PT) and Activated Partial Thromboplastin Time (APTT) prolongation may be indicative of coagulation problems [9]. In this case, the patient's progression to severe DSS highlights the challenges in accurately predicting the disease course during the acute phase, even in the presence of early diagnostic tests such as the NS1 Antigen Test and serological assays.

A severe dengue infection's critical manifestation, DSS, is characterized by severe shock, vascular leakage, and multi-organ failure. Supportive treatment, careful observation, and focused interventions designed to restore vascular stability and avoid complications are the main components of DSS management.

Management of DSS requires early and effective fluid management. Plasma leakage causes hypovolemia, which is countered by aggressive fluid resuscitation. Carefully supplied crystalloid solutions, like Ringer's lactate or isotonic saline, sustain organ perfusion without aggravating fluid overload [10]. The patient's

requirement for vasopressor support (dopamine, norepinephrine, or vasopressin) in this case, despite the early effectiveness of fluid management, emphasizes the limitations of fluid therapy alone, particularly in refractory shock.

Vasopressor medications such as dopamine, norepinephrine, or vasopressin are used to maintain appropriate blood pressure and tissue perfusion in cases of refractory shock or insufficient response to fluid resuscitation [11]. There are restrictions on the completeness of the patient history and specific clinical facts because this case analysis is retrospective. These inadequacies could affect the findings and point to the need for more research on rapid DSS progression in similar situations.

Some studies have recommended platelet transfusions in cases of severe thrombocytopenia that result in bleeding problems or imminent hemorrhage. Transfusion decisions should, however, be made on an individual basis, taking the patient's clinical status and the possibility of consequences into account [10, 12]. Understanding the unusual progression in this case teaches us important lessons about how to stay alert for early indications of serious disease in dengue patients who may seem stable at first.

Taking care of DSS-related problems is essential. Interventions specific to respiratory distress, neurologic symptoms, hepatic failure, and renal impairment are needed. Proactive monitoring for patients exhibiting early warning signs may enable prompt management in similar cases in the future, thus averting the abrupt onset of DSS.

In order to effectively manage these problems, supportive treatments such as respiratory assistance (ventilator), Continuous Renal Replacement Therapy (CRRT), hepatic protective medicines, and neurological monitoring are essential [10]. However, it is crucial to note that this specific case might not be representative of all dengue presentations, which would restrict how broadly these results can be applied.

To further understand why some cases, proceed more rapidly than others, more research could examine particular patient or environmental characteristics linked to rapid DSS progression. The effectiveness of targeted treatments for high-risk patients could potentially be investigated in such studies.

While this case demonstrates atypical DSS presentation, these results should be evaluated with caution, acknowledging the importance of single-case investigations without drawing too many conclusions. This discussion is in line with research that highlights the need for quick identification and treatment of severe dengue as well as the vital role that multidisciplinary approaches play in managing complications in unusual instances [10].

To sum up, this case highlights the necessity of early diagnostic tools, close monitoring, and flexible management techniques in order to successfully handle atypical dengue presentations. In circumstances like this, future studies that concentrate on prediction markers for quick advancement may improve patient care and outcomes.

CONCLUSION

In conclusion, this case report aims to highlight how crucial early detection and prevention measures are to lower the prevalence of DSS by implementing strict preventative practices like eliminating mosquito breeding sites, using insect repellents, wearing protective clothing and taking preventive measures to avoid endemic areas of dengue transmission are the first steps towards preventing DSS. This case illustrates the necessity of close monitoring in high-risk situations by highlighting the development of DSS and the key events that indicated this patient's swift decline. The lessons learnt in treating severe dengue are illustrated in this case, particularly with regard to early detection and treatment of atypical cases, which may have a major effect on patient outcomes. Even in young people with no medical history, dengue shock can result in fatal consequences thus, early identification and effective treatment greatly lower the chance of developing dengue shock and can lower the mortality rates. Stabilizing the patient's condition required prompt and intensive supportive care, including fluid management and hemodynamic support. Furthermore, in order to better manage challenging manifestations and provide better care in similar cases in the future, healthcare providers must get ongoing education on uncommon dengue symptoms.

CONSENT FOR PUBLICATION

Verbal Informed consent was obtained from the patient's family for the publication of this report, including anonymized details of the patient's clinical course and outcome. All personal identifying information has been removed to ensure confidentiality.

CONFLICT OF INTEREST

The author declares no competing interests.

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AUTHOR CONTRIBUTION

I, Rakhshanda Zareen, am the sole author of this case report. I obtained the patient's history from the family, performed the clinical assessment, and prepared the report entirely by myself.

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