Fatal Dengue Hemorrhagic Fever with Multi-Organ Failure: A Case Report from Teaching Hospital Peradeniya, Sri Lanka

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Abstract

Background: Dengue Fever is the most geographically widespread arthropod-borne disease. It can present a variety of clinical spectra varying from asymptomatic illness to dengue shock syndrome.

Case Presentation: 42-year-old female patient who has presented with a three-day history of fever and subsequently developed dengue hemorrhagic fever with multi-organ failure. Despite intensive care management, the patient died three days after admission to a tertiary care hospital.

Conclusion: Early recognition of dengue hemorrhagic fever and appropriate management during the critical phase are crucial.

Introduction

Dengue Fever is the most geographically widespread vector-borne disease. It can present a various clinical spectrum varying from asymptomatic illness to dengue shock syndrome (1). Dengue Hemorrhagic Fever (DHF) remains a significant cause of morbidity as well as mortality in tropical countries. Early detection and appropriate fluid management are crucial for preventing fatal outcomes.

Dengue is a vector-borne disease caused by the dengue virus. A single–stranded RNA flavivirus with four distinct serotypes (DENV-1 to DENV-4). The disease is transmitted through the bites of infected Aedes mosquitoes, primarily Aedes aegypti. Clinical manifestations ranged from asymptomatic viral infection to severe stages, including dengue hemorrhagic fever (DHF) and dengue shock syndrome (DSS) (2).

The clinical spectrum of dengue encompasses four distinct categories, reflecting the varying severity and manifestations of the disease. (3). The first is undifferentiated febrile illness, which presents as a non-specific fever and is often difficult to distinguish from other viral infections, particularly in endemic areas. The second category is Dengue Fever (DF), a more defined illness characterised by high-grade fever, severe headache, retro-orbital pain, myalgia, arthralgia, and sometimes a rash. The third and more severe form is Dengue Hemorrhagic Fever (DHF), which includes all the features of DF along with increased vascular permeability, plasma leakage, thrombocytopenia, and bleeding tendencies, potentially progressing to shock. The final category is Expanded Dengue Syndrome, which

encompasses atypical or severe manifestations involving multiple organs, such as the liver, brain, heart, or kidneys, and may occur in patients with co-morbidities or those receiving prolonged or inappropriate treatment. (4).

In the initial stages, patients with DHF often present with signs and symptoms similar to those of classical dengue fever. However, as the illness progresses—typically after the third day—distinct features of plasma leakage begin to emerge, marking the transition to DHF. The diagnosis of DHF is based on specific criteria established by the World Health Organization. These include a history of high fever or a recent acute febrile illness, evidence of hemorrhagic manifestations such as a positive tourniquet test, and thrombocytopenia with a platelet count of less than 100,000 cells/mm³ (5). The identification of these features is important for early recognition and appropriate management of DHF to prevent progression to more severe complications such as DSS. (6).

Case presentation

A 43-year-old female patient presented to District Hospital Kadugannawa with a 3-day history of fever, vomiting and body pain. She was conscious and rational on admission. Her vital signs were stable with normal blood pressure. Before hospitalization, she had consulted a general practitioner who prescribed paracetamol and antihistamines.

Initial blood investigation on the third day showed:

- White blood cell count: normal range with viral pattern.
- Platelet count: >100,000/mm³

Initially, the patient was managed as a simple viral fever with antipyretics and normal saline infusions. Due to the limitations of laboratory facilities at the district hospital, further investigation was not immediately done.

On the sixth day of illness, repeat investigations revealed a white blood cell count of 2.1 × 10³/μL, a platelet count of 11 × 10³/μL, a hematocrit of 36%, and a haemoglobin level of 12.7 g/dL. Around this time, the patient suddenly developed severe headache, arthralgia, myalgia, and oliguria, which prompted her transfer to the Teaching Hospital, Peradeniya, for specialized care.

On admission to the Tertiary care hospital, there was no evidence of bleeding manifestations. An abdominal ultrasound scan revealed peri-cholecystic fluid, indicating mild plasma leakage. The patient was conscious and rational, and an erythematous rash was noted over her face. Despite having a low platelet count, there was no evidence of internal bleeding or significant fluid accumulation.

Table I: Laboratory results of a dengue hemorrhagic fever patient, according to the day of illness. The patient first sought medical care on day 6

Investigations	**Day 6**	**Day 7 (4 pm)**	**Day 7 (9 pm)**	**Day 8 (1:45 am)**
WBC (×10³/μL)	2.1	2.38	3.82	5.14
Platelets (×10³/μL)	11	6	11	28
Hematocrit (%)	36.9	24.4	36.5	40.4
Hemoglobin (g/dL)	12.7	8.5	12.8	14.2

The patient's condition deteriorated rapidly, marked by a significant drop in blood pressure to 80/60 mmHg and a fall in oxygen saturation to 80%. She progressed into shock, necessitating immediate intubation and initiation of mechanical ventilation. In response to her worsening condition, the patient was transferred to the Intensive Care Unit (ICU) for advanced supportive care.

In the ICU, she received positive pressure ventilation and inotropic support to stabilize hemodynamics. Serial arterial blood gas analyses revealed metabolic acidosis, which was managed with sodium bicarbonate. Multiple platelet transfusions were administered in view of her thrombocytopenia, and continuous monitoring of organ function was carried out throughout her stay in the ICU.

Laboratory investigations confirmed the presence of multi-organ failure. Liver function tests showed a progressive and severe rise in transaminase levels, with aspartate aminotransferase (AST) increasing from 1,017 to 10,271 U/L and alanine aminotransferase (ALT) from 881 to 3,806 U/L. Total bilirubin levels reached 73 mg/dL, and the international normalized ratio (INR) was elevated at 2.55, indicating significant hepatic dysfunction and coagulopathy.

Renal function also declined progressively, with serum creatinine rising from 33.8 to 106 µmol/L, and oliguria ultimately progressing to anuria. Arterial blood gas analysis demonstrated persistent metabolic acidosis, with a pH range of 7.33 to 7.49, elevated lactate levels ranging from 3.6 to 9.4 mmol/L, and a base excess between -19.6 and -10.3.

Despite comprehensive and intensive care measures, the patient's condition continued to deteriorate. On the night of day 12 of illness, she suffered a cardiac arrest. Cardiopulmonary resuscitation was initiated and continued for one and a half hours, including multiple defibrillation attempts. Unfortunately, despite all efforts, the patient could not be revived and was pronounced dead.

Postmortem Findings

Macroscopic Findings of Liver: Evidence of massive hepatic necrosis and internal bleeding was seen in the Liver (Figures 1&2)
Figure 1

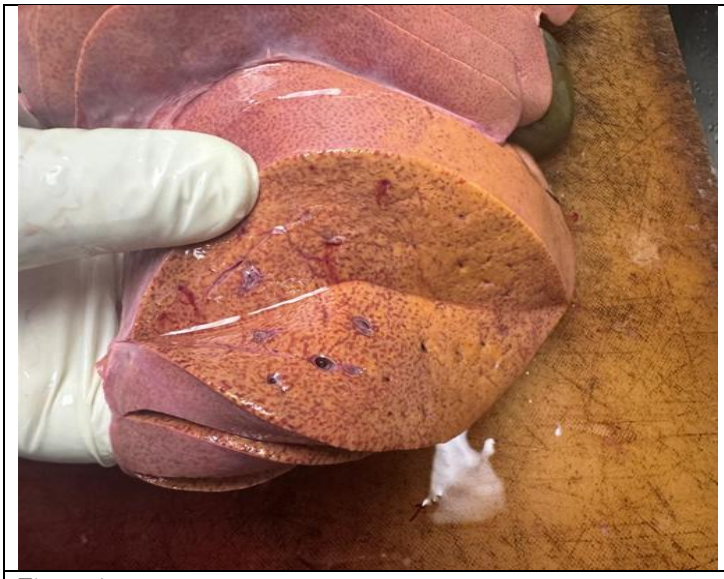

Figure 1

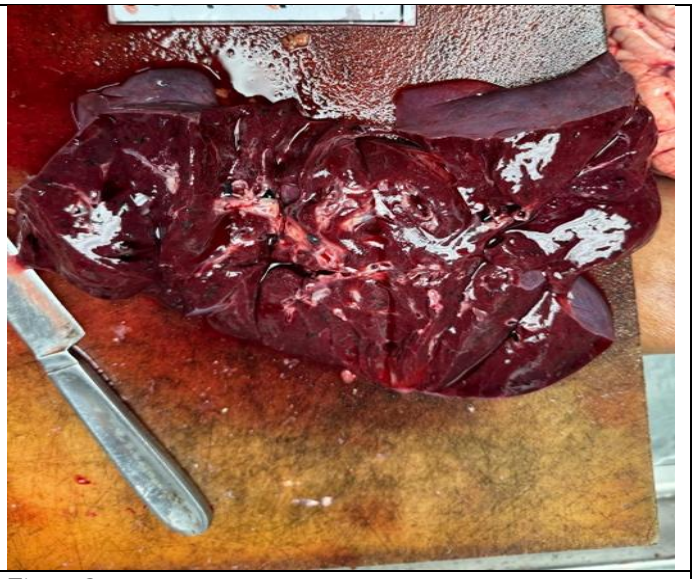

Figure 2

Histopathological examination of the liver reveals massive centrilobular necrosis with only thin rims of periportal viable hepatocytes remaining. These findings are consistent with viral hepatitis. In the context of dengue infection, the virus is known to replicate within hepatocytes and Kupffer cells, leading to direct cellular damage and subsequent liver cell necrosis. In addition to this direct cytopathic effect, liver damage can also result from cytokine-mediated injury due to the host's immune response, as well as from hemodynamic disturbances associated with shock. (Figure 3 & 4)

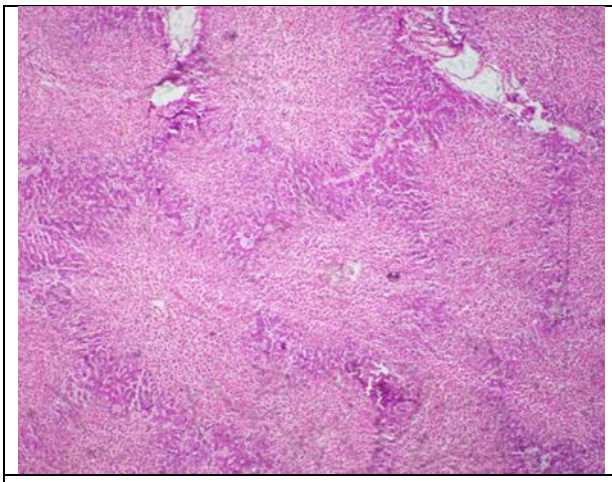

Figure 3

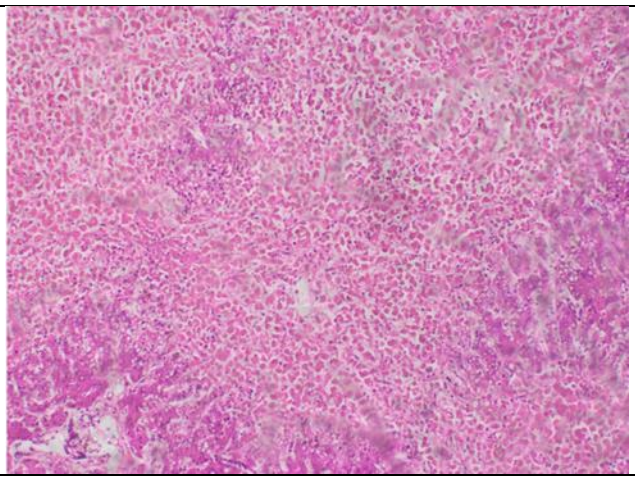

Figure 4

The gallbladder was congested with hemorrhagic changes (Figure 5), and the histology of the liver shows only thin rims of periportal viable hepatocytes. Additionally, features of acute cholecystitis with hemorrhagic changes were observed (Figure 6).

Figure 5

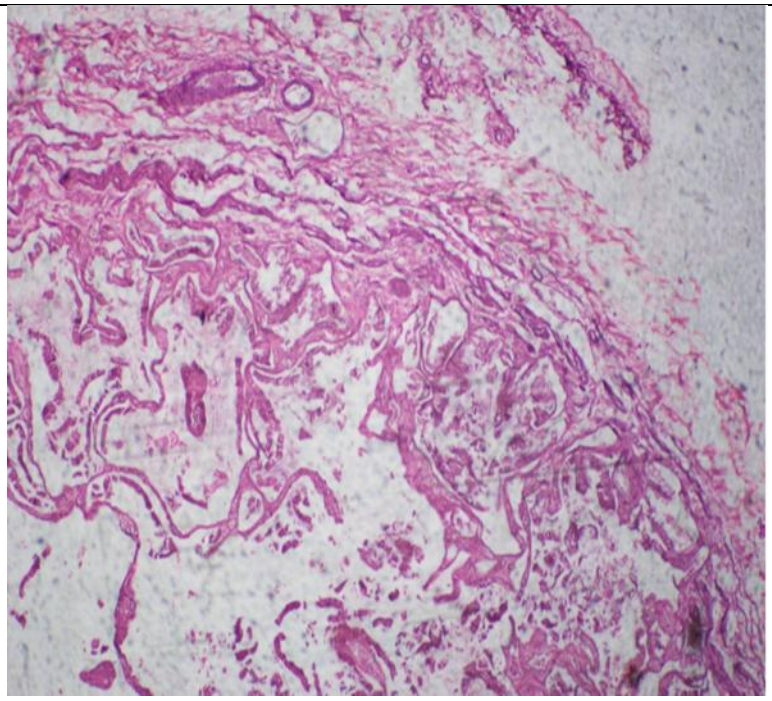

Figure 6

Cardiomegaly with myocardial edema was observed in the heart (Figure 7), and histological examination revealed marked interstitial edema, attenuation of myocardial fibres, and infiltration by mononuclear inflammatory cells. These findings are consistent with myocarditis (Figure 8).

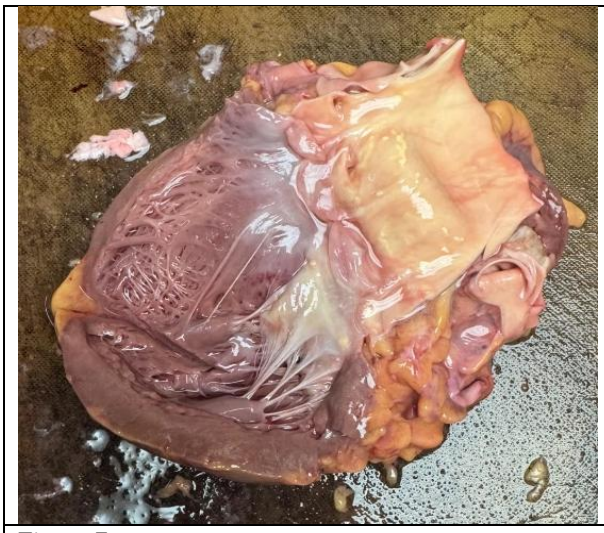

Figure 7

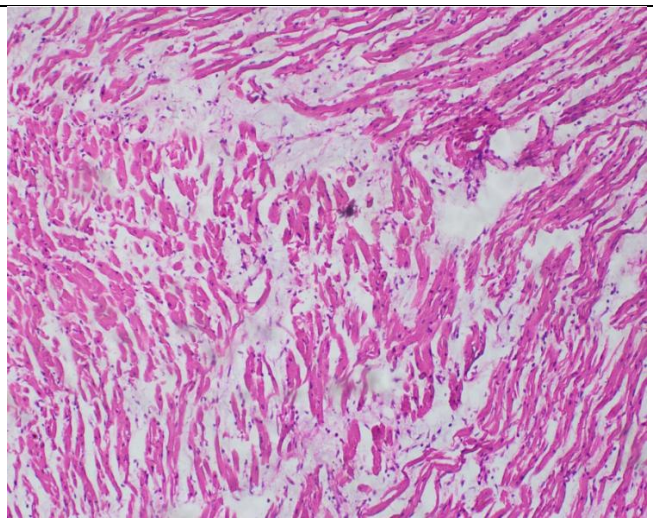

Figure 8

Examination of the kidneys revealed features of acute tubular necrosis with cortical and glomerular congestion, along with interstitial edema (Figures 9 and 10). These findings are indicative of significant renal injury, commonly seen in severe systemic infections or shock.

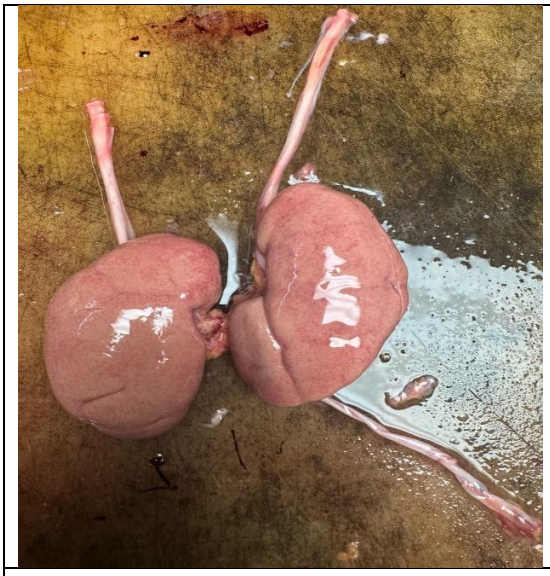

Figure 9.

Figure 10

Examination of the brain revealed cerebral edema (Figures 11 and 12), with no definitive evidence of encephalitis. Histological sections showed areas of edema and moderate mononuclear inflammatory cell infiltration (Figure 13). Additionally, findings were suggestive of a non-bacterial type of meningitis associated with edema (Figure 14).

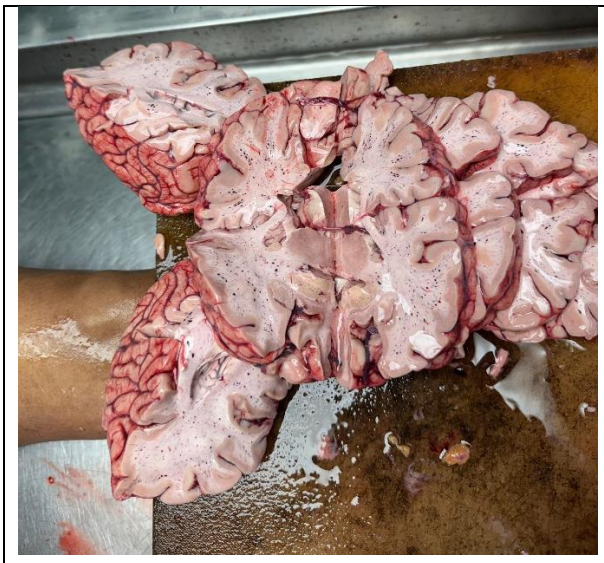

Figure 11

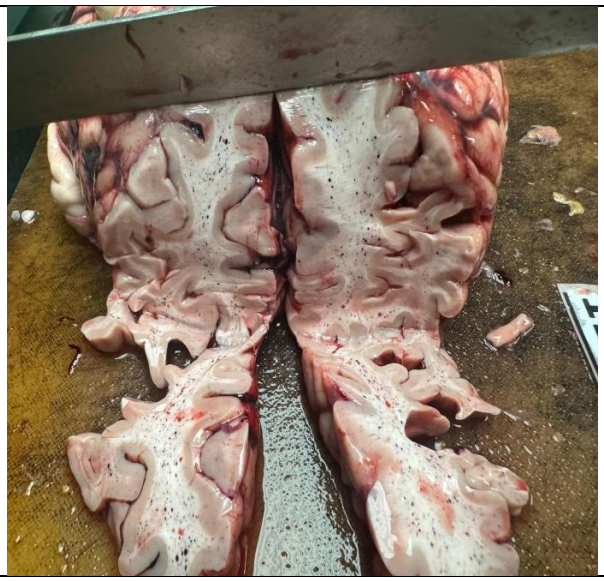

Figure 12

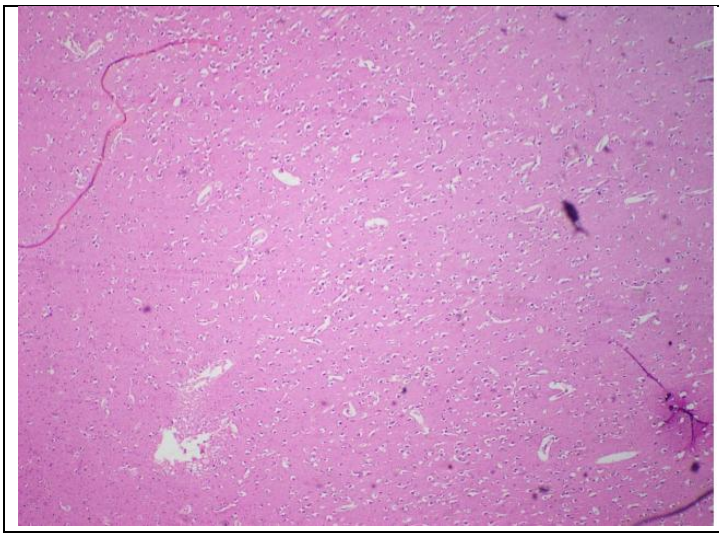

Figure 13

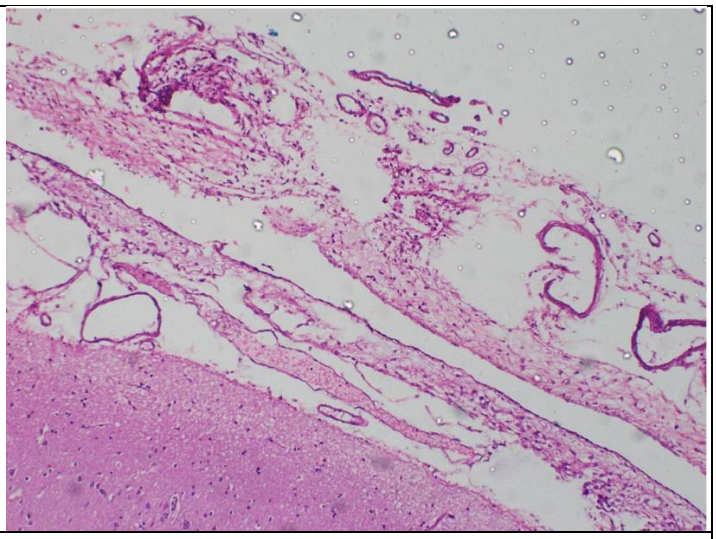

Figure 14

Histology Examination of the lungs showed alveoli containing lymphocytes and neutrophils within a fibrinous background (Figure 15), which is indicative of the resolving phase of pneumonia (Figure 16).

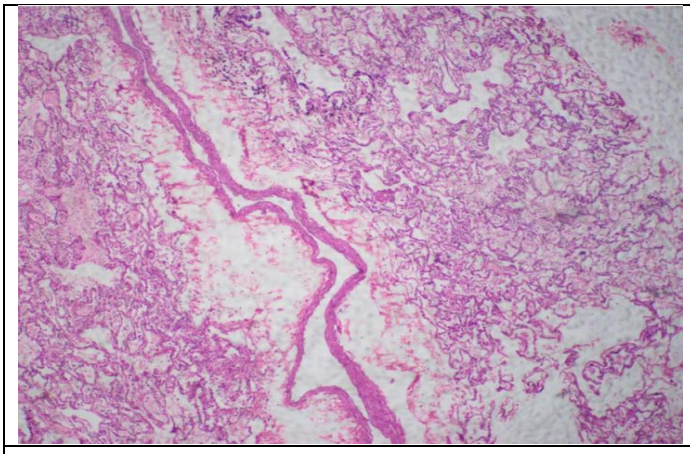

Figure 15

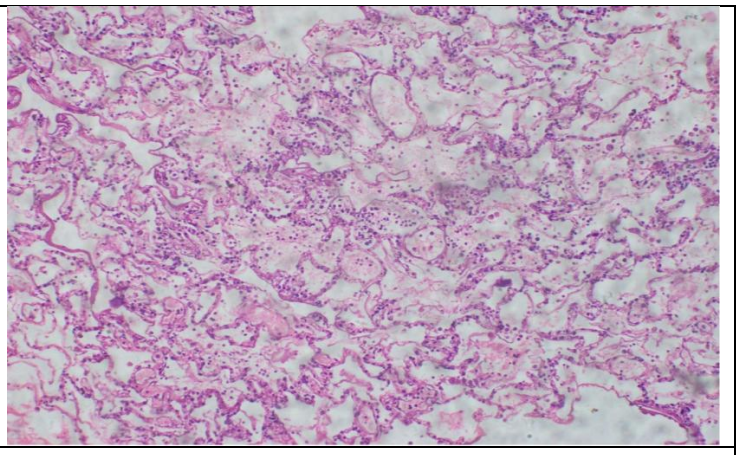

Figure 16

Histology examination of the Spleen showed Red pulp expansion and congestion (Figure 17).

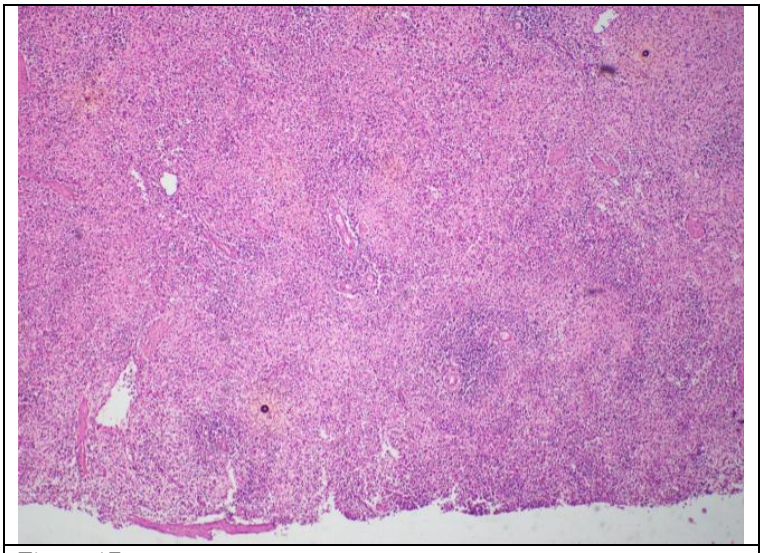

Figure 17

Discussion

This case illustrates the rapid progression of DHF to multi-organ failure despite intensive medical management. It underscores several critical aspects of dengue care and highlights key challenges encountered during its management.

The initial presentation at the district hospital resembled that of a simple viral fever, leading to a delayed diagnosis of the severe form of dengue. This delay was compounded by limited laboratory facilities, which hindered early identification of warning signs. The case emphasizes the urgent need to strengthen diagnostic capabilities, particularly in peripheral and resource-limited healthcare settings, to enable timely detection and intervention.

The patient's rapid deterioration occurred during the critical phase of illness, typically between days three to seven, which is characteristic of DHF. The emergence of plasma leakage, as evidenced by pericholecystic fluid accumulation, marked the transition from uncomplicated dengue fever to its more severe hemorrhagic form. This transition underscores the importance of close monitoring during this critical period.

Dengue's capacity to cause widespread organ involvement is clearly demonstrated in this case. The liver was severely affected, with massive hepatic necrosis and extremely elevated transaminase levels. Cardiac involvement was evident in the form of myocarditis, which contributed to hemodynamic instability. The kidneys also suffered significant injury, progressing to acute renal failure, while the

hematological system was compromised by severe thrombocytopenia and coagulopathy. These findings highlight the systemic nature of severe dengue infections and the complexity of managing such cases.

Fluid management remains a cornerstone of dengue treatment (7). However, this case illustrates the challenge of balancing adequate perfusion with the risk of fluid overload, particularly in the setting of capillary leakage. Despite intensive care and appropriate interventions, the patient's condition progressed to irreversible multi-organ failure, reflecting the potentially fatal course of severe dengue even with optimal supportive therapy.

Conclusion

This case highlights the critical importance of early recognition of dengue hemorrhagic fever, appropriate monitoring during the critical phase of illness, and timely transfer to facilities equipped with intensive care capabilities. It also underscores the urgent need to improve diagnostic facilities at district-level hospitals to facilitate earlier detection and intervention. Despite advances in the understanding of dengue pathophysiology and the development of standardized management protocols, severe cases that progress to multi-organ failure continue to carry a high risk of mortality. This case adds to the growing body of literature on fatal outcomes in dengue and may help inform future strategies for early identification, risk stratification, and clinical management of severe dengue infections.

Acknowledgments

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