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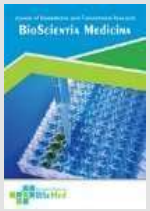

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Dengue Haemorrhagic Fever with Unusual Presentations and Complications in Sanjiwani General Hospital: A Case Series

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A B S T R A C T

Background: Dengue hemorrhagic fever (DHF) is a viral infection transmitted by *Aedes* mosquito with a wide clinical spectrum, ranging from mild fever to life-threatening complications. Atypical clinical manifestations of DHF can cause delays in diagnosis and treatment, so it is important to increase clinician awareness of these unusual manifestations. **Case presentation:** We report four cases of DHF with atypical manifestations: (1) A 50-year-old woman with spontaneous psoas hematoma, (2) An elderly man with pulmonary edema, (3) A young man with acute myocarditis, and (4) A middle-aged man. middle-aged with encephalopathy. **Conclusion:** Atypical manifestations of DHF can occur at various ages and can involve various organs. It is important for clinicians to consider dengue fever as a differential diagnosis in patients with fever and unusual clinical manifestations, especially in dengue-endemic areas. Early diagnosis and appropriate treatment can improve patient clinical outcomes.

1. Introduction

Dengue hemorrhagic fever (DHF) is a viral infection transmitted by the *Aedes aegypti* and *Aedes albopictus* mosquitoes. This disease is endemic in more than 100 countries in tropical and subtropical areas, including Indonesia. DHF has a wide clinical spectrum, ranging from dengue fever (DF) without complications to dengue fever with manifestations of bleeding and dengue shock which can be fatal. The classic clinical manifestation of dengue fever usually begins with a sudden high fever that lasts for 2-7 days. This fever is often accompanied by severe headaches, retro-orbital pain (pain behind the eyes), myalgia (muscle pain), arthralgia (joint pain), skin rashes, and bleeding manifestations such as petechiae, purpura, or gum bleeding. Thrombocytopenia (decreased platelet count) is also a characteristic feature of DHF.[1,2]

Apart from these classic manifestations, DHF can also manifest with atypical symptoms involving various organs and body systems. These atypical manifestations can occur at any phase of the disease, both in the fever phase, critical phase, and recovery phase. Some of the atypical manifestations of DHF that are often reported include Hepatic Manifestations: Elevated liver enzymes (AST and ALT), hepatomegaly (enlarged liver), jaundice (jaundice), and even fulminant liver failure; Renal Manifestations: Proteinuria (leakage of protein in the urine), hematuria

(blood in the urine), increased serum creatinine, and even acute renal failure; Neurological Manifestations: Encephalopathy (impaired brain function), aseptic meningitis, myelitis, peripheral neuropathy, and Guillain-Barré syndrome; Cardiovascular Manifestations: Myocarditis (inflammation of the heart muscle), pericarditis (inflammation of the lining of the heart), cardiac arrhythmias, and cardiogenic shock; Pulmonary Manifestations: Pulmonary edema, pleural effusion, acute respiratory distress syndrome (ARDS), and pneumonia; Hematological Manifestations: Coagulopathy (blood clotting disorders), hemolysis (destruction of red blood cells), and pancytopenia (decreased all types of blood cells); Gastrointestinal Manifestations: Nausea, vomiting, abdominal pain, hepatomegaly, splenomegaly (enlarged spleen), ascites (accumulation of fluid in the abdominal cavity), and gastrointestinal bleeding; Musculoskeletal Manifestations: Myalgia, arthralgia, and rarely, myositis (muscle inflammation) or rhabdomyolysis (muscle damage).[3,4]

Several risk factors are thought to be associated with an increased risk of developing atypical manifestations of dengue fever, including: Children and adults are more susceptible to experiencing atypical manifestations compared to adolescents; Secondary dengue infection with different virus serotypes increases the risk of atypical manifestations and severe dengue; Patients with comorbid diseases such as diabetes mellitus, hypertension, heart disease, kidney disease, or autoimmune diseases are more susceptible to complications from dengue fever; Malnutrition and micronutrient deficiencies can increase the risk of atypical manifestations and severe dengue fever; Genetic Factors: Several studies have shown a genetic predisposition to atypical manifestations of dengue fever. Atypical manifestations of dengue fever are often nonspecific and can resemble other diseases, making diagnosis difficult. In addition, delays in diagnosis and treatment can increase the risk of life-threatening complications. Therefore, it is important for clinicians to be highly alert to the possibility of dengue fever in patients with fever and unusual clinical manifestations, especially in dengue-endemic areas.[5,6]

The diagnosis of dengue fever is made based on clinical symptoms, laboratory examination results (dengue serology test, complete blood test), and sometimes supporting examinations such as abdominal ultrasound or chest X-ray. Treatment for dengue fever is primarily supportive, including administering intravenous fluids to prevent dehydration and shock, as well as close monitoring for signs of bleeding and other complications. Increasing clinician awareness of atypical manifestations of dengue fever is very important to ensure early diagnosis and appropriate treatment, thereby improving patient clinical outcomes. Therefore, it is important for clinicians to always update their knowledge about DHF, including atypical manifestations, risk factors, and the latest developments in diagnosis and treatment.[7] This case report presents four cases of dengue fever with rare atypical manifestations, namely spontaneous psoas hematoma, pulmonary edema, acute myocarditis, and encephalopathy. These cases highlight the importance of clinician awareness of the broad and complex clinical spectrum of DHF.

2. Case Presentation

Case 1: Severe dengue with atypical manifestation (Spontaneous Left Psoas Haematoma)

A 50-year-old woman arrived with a three-day history of high fever (with a maximum recorded temperature of 39.5°C) and widespread body aches. Heart rate (102/min) and blood pressure (110/70 mmHg) were measured physically. Both active bleeding and a rash were absent. Additional systemic and general exams turned up nothing unusual. Dengue fever was the working diagnosis at the time of admission. Day 3: Based on a right-sided pleural effusion, gall bladder wall oedema, and free fluid in the hepatorenal pouch, Dengue hemorrhagic fever (DHF) was identified, and the patient was started on critical phase care. The patient's right-sided pleural effusion fluid therapy was started in the critical phase once

there were signs of leakage. After the crucial stages were over, she reported having excruciating discomfort in her left inguinal region and groin. Examining the skin revealed no soreness and normal appearance, but flexing her left thigh caused excruciating discomfort. There was no swelling that could be seen. Every lower limb pulse was present, and the neurological examination revealed no abnormalities. Haemoglobin (Hb) 13.0 g/L (13–15 g/L), haematocrit (Hct) 45%, total leukocyte count (TLC) 2·94 k/μL, platelet count (PC) 15 k/μL, alanine aminotransferase (ALT) 70 u/L (Normal <40), aspertate aminotransferase (AST) 60 u/L (Normal <40), serum sodium 132 mmol/L (135–145), and C-reactive protein (CRP) 35 nmol/L (<6) were the results of significant initial laboratory investigations. APTT 37 s (30–40), PT 15 (11 to 13.5 s). Positive results for the dengue-NS1-antigen and anti-dengue-antibodies (IgM and IgG) confirmed the clinical suspicion of DHF. An immediate USG revealed a hypoechoic region including the superficial fibers of the middle half of the left psoas following the abrupt onset of left thigh pain. There is no increase in vascularity. The remaining PSOAs seem to be normal. The hip joint is in the normal range. The cause of appearance is bleeding into the left psoas, maybe accompanied by a subsequent infection. The lowest platelet count observed during her stay was 12 K/μL on the day the haematoma developed. The patient was kept under close observation. The patient experienced tachycardia and a decrease in PCV as the pain persisted. Additionally, she experienced a surge in temperature, with a CRP of 95. A second USG revealed a poorly defined hypoechoeic area in the left psoas region that was consistent with the previously discovered left psoas hemorrhatoma without growing or getting worse. There was also no increased vascularity. The patient's PCV and tachycardia were reduced, necessitating a red cell transfusion of 5 ml/kg. Hematocrit increased as the tachycardia subsided. No more support or care was needed. On the eleventh day of hospitalization, the patient was released to go home after making a steady improvement over the next few days.

Case 2: An elderly male with pulmonary oedema manifestation of DHF

Mr. R, a 53-year-old man, visited the ER with the primary complaint of a fever that had started three days earlier. The patient additionally reported having stomach aches that made her feel nauseous and aching all over her body. The patient has a history of hypertension and type 2 diabetes. The patient takes amlodipine 1 mg for hypertension, glimepiride 1 mg for diabetes, and metformin 3 mg for hypertension on a regular basis. Other than an epigastric ache, the patient's physical examinations were usually normal. Important preliminary laboratory tests revealed the following: total leukocyte count (TLC) 1·95 K/μL, platelet count (PC) 75 K/μL, hemoglobin (Hb) 11.5 g/L (13–15 g/L), and hematocrit (Hct) 45%. A cardiomegaly was seen on a chest radiograph. A typical sinus rhythm was seen on the ECG. On the sixth day of treatment, the patient suddenly developed dyspnea. Rales were found on the bases of both lungs during the physical examination; the critical sign was: 120/90 mmHg in blood pressure. Heart rate: 98 beats per minute; breathing rate: 25 times per minute. 38.2 degrees Celsius, 92% oxygen saturation, and 2 liters of oxygen per minute through a nasal cannula. The high care unit (HCU) was the new location for the patient. After a second examination, a full blood count revealed that the patient's platelet count had decreased to 25.000, with a hematocrit of 47%. IgG and IgM dengue tests for the patient came back positive. Compensated respiratory alkalosis was revealed by a blood gas study. Additionally, there was an increased blood sugar level of 278 mg/dl. A bilateral pleural effusion and perihilar haziness had been seen on a repeat chest X-ray. Acute pulmonary oedema, dengue hemorrhagic fever, hyperglycemia in type 2 diabetes, and cardiomegaly were then added to the original diagnosis. The patient underwent additional O_2 treatment using a non-rebreathing mask at a rate of 10 liters per minute, as well as an enhanced insulin regimen, two 40 mg injections of furosemide, and three 10-unit Aspart units with a 12-hour blood sugar monitoring schedule. On the

fourteenth day of the treatment, the patient's vital signs were stable, showing 140/80 mmHg for blood pressure, 98 BPM for heart rate, 20 times per minute for breathing, 36.5°C for temperature, 98% oxygen saturation with room air, 101.000 platelets per microliter of blood, and 170 mg/dl for blood sugar. After that, the patient was released from the hospital.

Case 3: Acute myocarditis in dengue hemorrhagic fever

A 19-year-old male patient who had previously been in good health arrived at the emergency room complaining of a two-day fever, nausea, and body aches. He did not exhibit any postural signs, bleeding symptoms, or abdominal pain. Upon examination, he did not appear pale or icteric, but he was flushed and febrile. His dehydration was moderate. The patient's pulse rate was 100 beats per minute, blood pressure was 110/70 mmHg, and capillary refilling time (CRFT) was less than two seconds. An examination of the abdomen revealed no loose fluid. Examining the respiratory system revealed clear lung areas. The assessment of other systems was normal. His serotype was determined to be DEN1 and his NS1 antigen was positive. He was under constant observation while being treated for dengue illness. He complained of excessive fatigue and retrosternal chest pain on the third day of the fever. Her electrocardiogram (ECG) at that time revealed acute T wave inversion in leads V2-V5. Her cardiovascular system evaluation was normal at that time. The 2D echo revealed widespread left ventricular hypokinesia and moderate LV function deterioration, while troponin I was negative. It was a 43% ejection fraction. He was diagnosed with myocarditis-complicated dengue fever. For two days, intravenous hydrocortisone 200 mg eight hours a day was given to lower myocardial inflammation. He complained of abdominal pain on the fifth day after being admitted, and an ultrasound scan indicated that the hepato-renal pouch contained free fluid. There was a 70 bpm heart rate, a blood pressure of 100/70 mmHg, and a CRFT of less than 2 s. After being admitted to the high dependency unit (HDU), he was treated for DHF worsened by myocarditis under cautious supervision to prevent fluid overload and constant monitoring. He recovered from the critical phase of dengue fever and was released on day seven of the illness. He was counseled to restrict his physical activity. On the fourteenth day of the sickness, the ECG revealed a reversal of T inversions. Left ventricular function improved, as evidenced by an echocardiogram with a 57% ejection fraction.

Case 4: Dengue hemorrhagic fever with encephalopathy in an adult

A 59-year-old man who had been in good condition was sent to the hospital to be evaluated for acute encephalopathy. His main complaints upon admission to the hospital were a fever, myalgia, vomiting, and diarrhea that persisted for three days. Upon physical examination, the individual was found to be alert and his body temperature was 38.5ºC. No further abnormalities were seen. Important preliminary laboratory tests revealed the following: total leukocyte count (TLC) 2·64 K/μL, platelet count (PC) 18 K/μL, hemoglobin (Hb) 13.0 g/L (13–15 g/L), and hematocrit (Hct) 45%. He received supportive care in addition to intravenous fluid therapy once DHF was suspected. On day 6 of the fever receded, but the patient started to feel sleepy and disoriented. The levels of magnesium and calcium in the serum were normal. Anti-HCV, anti-HIV, HBsAg, and HBsAb levels in serum were negative. TPHA and serum VDRL did not react. The brain's MRI and CT scan results were within normal bounds. Supportive care was given to the patient. The following day, widespread petechiae were found. Day 10 saw a significant improvement in his bewilderment. He recovered fully and was released. A lumbar puncture carried out with consent on follow-up day 18, revealed a clear cerebrospinal fluid (CSF) with an opening pressure of 130 mm H_2O. There were 5 cells/mm^3 of white blood cells. There was a concomitant serum glucose level of 80 mg/dl and a protein level of 48 mg/dl and 51 mg/dl, respectively. CSF culture was negative. Based on the clinical presentation, thrombocytopenia, and positive serum

dengue IgM antibodies, our patient was definitively diagnosed with DHF. The high titer of dengue IgG antibodies indicates that this is a case of dengue infection.

3. Discussion

Dengue hemorrhagic fever (DHF), a disease caused by the dengue virus which is transmitted through the of the bites *Aedes* mosquito, is known for its diverse clinical manifestations. Apart from common symptoms such as high fever, muscle and joint pain, and rash, dengue fever can also cause unusual complications and impact various organs. The four cases that have been presented provide an overview of the spectrum of atypical manifestations of DHF that are rare but important to recognize.[8]

Spontaneous psoas hematoma is a rare but significant complication of dengue fever. The psoas muscle, which is located on the inside of the hip, can bleed due to several factors associated with dengue infection. Severe thrombocytopenia, namely a decrease in the number of platelets that play a role in blood clotting, is one of the main risk factors. In DHF, the dengue virus can infect and damage megakaryocyte cells in the bone marrow, where platelets are produced. In addition, the immune response to the dengue virus can also cause platelet destruction. As a result, the body's ability to form blood clots is impaired, increasing the risk of spontaneous bleeding, including into the psoas muscle.[9,10]

Apart from thrombocytopenia, increased vascular permeability also plays a role in the occurrence of psoas hematoma in DHF. The dengue virus can induce the release of inflammatory mediators which cause damage to the walls of blood vessels, resulting in plasma and blood cells leaving the blood vessels. This condition, known as plasma leak syndrome, can cause bleeding in various locations, including the psoas muscle.[11,12]

Uncontrolled activation of the coagulation system may also contribute to the formation of psoas hematoma. In DHF, there is an imbalance between procoagulant and anticoagulant factors, which can cause excessive blood clot formation. These blood clots can block small blood vessels, causing ischemia and bleeding in the surrounding tissue, including the psoas muscle. Symptoms of a psoas hematoma include sudden and severe low back or pelvic pain, difficulty walking, and sometimes fever. The diagnosis is made through physical examination and imaging such as ultrasound or CT scan. Treatment involves supportive therapy, blood transfusions if necessary, and close monitoring for complications such as nerve or blood vessel compression.[13,14]

Pulmonary edema is a rare but potentially fatal atypical manifestation of dengue fever. Pulmonary edema occurs when fluid builds up in the lungs, disrupting the exchange of oxygen and carbon dioxide. In DHF, pulmonary edema can be caused by several mechanisms, including increased pulmonary capillary permeability, myocardial dysfunction, and fluid balance disorders. Increased pulmonary capillary permeability is one of the main mechanisms for pulmonary edema in DHF. The dengue virus can induce the release of inflammatory mediators that cause damage to the pulmonary capillary walls so that fluid and protein escape from the blood vessels into the lung tissue. This causes a buildup of fluid in the alveoli, the small air sacs in the lungs where gas exchange occurs.[15]

Myocardial dysfunction, namely impaired heart muscle function, can also contribute to the occurrence of pulmonary edema in DHF. The dengue virus can infect myocardial cells directly or cause myocardial damage through an excessive immune response. Myocardial dysfunction causes a decrease in heart contractility, resulting in the accumulation of blood in the lungs and an increase in pulmonary capillary hydrostatic pressure. This forces fluid out of the capillaries into the lung tissue, causing pulmonary edema. Fluid balance disorders also play a role in the pathogenesis of pulmonary edema in DHF. In the critical phase of DHF, plasma leakage often occurs which causes a decrease in blood volume. To compensate for the decreased blood volume, the body

will retain fluid and sodium, which can lead to fluid overload and pulmonary edema. Symptoms of pulmonary edema include shortness of breath, coughing, production of foamy sputum, and tachypnea (rapid breathing). Diagnosis is made through physical examination, chest X-ray, and sometimes arterial blood gas examination. Treatment involves oxygen therapy, diuretics to reduce excess fluid, and sometimes a mechanical ventilator if respiratory failure occurs.[15,16]

Acute myocarditis is a rare but potentially life-threatening complication of dengue fever. Myocarditis is inflammation of the heart muscle which can be caused by viral, bacterial or parasitic infections, as well as autoimmune reactions. In DHF, the dengue virus can infect myocardial cells directly or trigger an excessive immune response, causing inflammation and damage to the heart muscle. Dengue virus invasion of the myocardium can occur through several mechanisms. Dengue virus can enter myocardial cells through specific receptors or through endocytosis. After entering cells, the dengue virus will replicate and cause cellular damage. In addition, the dengue virus can also trigger an excessive immune response, including activation of cytotoxic T cells and release of proinflammatory cytokines, which can cause further damage to the myocardium.[17,18]

Endothelial dysfunction, namely disruption of the function of the inner lining of blood vessels, also plays a role in the pathogenesis of myocarditis in DHF. Dengue virus can infect endothelial cells and cause endothelial dysfunction, which is characterized by increased vascular permeability, platelet activation, and impaired regulation of vascular tone. Endothelial dysfunction can cause myocardial ischemia and exacerbate inflammation. Symptoms of myocarditis vary, ranging from asymptomatic to acute heart failure. Frequent symptoms include chest pain, shortness of breath, palpitations, fatigue, and edema. The diagnosis is made through physical examination, ECG, echocardiography, and examination of markers of myocardial damage such as troponin. Treatment involves supportive therapy, medications to reduce the heart's workload, and sometimes interventions such as angioplasty or pacemaker implantation.[16,18]

Encephalopathy is a brain function disorder that can be caused by various factors, including infection, metabolic disorders, or poisoning. In DHF, encephalopathy is a rare complication but can cause significant morbidity and mortality. The mechanism underlying the occurrence of encephalopathy in dengue fever is not fully understood but is thought to involve several factors, including impaired blood-brain barrier function, dengue virus invasion of the central nervous system, and excessive immune reactions. The blood-brain barrier is a structure that protects the brain from harmful substances in the blood. In DHF, the dengue virus can disrupt the function of the blood-brain barrier, allowing viruses and inflammatory mediators to enter the central nervous system. This can cause inflammation and damage to brain tissue, manifesting as encephalopathy.[18,19]

Dengue virus invasion of the central nervous system can also occur directly. The dengue virus can infect brain cells, such as neurons and glial cells, causing cellular damage and impaired brain function. In addition, the dengue virus can also trigger an excessive immune response in the central nervous system, including activation of microglia and the release of pro-inflammatory cytokines, which can worsen brain damage. Symptoms of encephalopathy in DHF vary, ranging from mild impaired consciousness to coma. Symptoms that often appear include headaches, fever, vomiting, seizures, behavioral disturbances, and impaired motor function. The diagnosis is made through physical examination, neurological examination, and sometimes supporting examinations such as lumbar puncture or electroencephalography (EEG). Treatment involves supportive therapy, including administration of intravenous fluids, anticonvulsant medications if seizures occur, and close monitoring for signs of increased intracranial pressure.[17,19]

Atypical manifestations of dengue fever represent a challenge in the diagnosis and management of this disease. The four cases presented demonstrate that

dengue fever can involve multiple organs and body systems, causing unusual and potentially fatal complications. Therefore, it is important for clinicians to be highly alert to the possibility of dengue fever in patients with fever and unusual clinical manifestations, especially in dengue-endemic areas. Early diagnosis and appropriate treatment can improve patient clinical outcomes.[20]

4. Conclusion

Atypical manifestations of DHF can occur at various ages and can involve various organs. It is important for clinicians to consider dengue fever as a differential diagnosis in patients with fever and unusual clinical manifestations, especially in dengue-endemic areas. Early diagnosis and appropriate treatment can improve patient clinical outcomes.

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