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Ali Madian

Department of Internal Medicine, Faculty of Medicine, Al-Azhar University-Assiut, Assiut 71524, Egypt,
a.madian@azhar.edu.eg

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CASE REPORT

Sickle Cell Patients With Dengue Infection Waiting for Dawn: Case Report

Ali Madian*

Department of Internal Medicine, Faculty of Medicine, Al-Azhar University Assiut, Assiut, Egypt

Abstract

We report a case of dengue infection in a 20-year-old male with sickle cell anemia who presents to the emergency department with fever, retro-orbital headache, bone pain, back pain, and myalgia. Laboratory tests revealed elevated hematocrit, thrombocytopenia, IgM, and IgG antibodies positivity to dengue virus. The patient was treated with intravenous fluid hydration and paracetamol with rapid deterioration and development of acute respiratory distress and hypovolemic shock due to alveolar hemorrhage.

Keywords: Dengue fever, Dengue hemorrhagic fever, Dengue shock syndrome, Sickle cell anemia

1. Introduction

Dengue is a mosquito-borne infectious disease, that is caused by dengue viruses. Dengue virus is an RNA virus belonging to the Flavivirus group,^{1,2} there are four serotypes of dengue viruses DENV-1, DENV-2, DENV-3, and DENV-4. Dengue is common in humid tropical and subtropical areas during rainy seasons.³

Approximately 80 % of dengue infections are asymptomatic, less than 20 % infected persons present with clinical manifestations, and 2.5 % die annually.⁴ About 100 million cases of dengue infection are reported each year, and around 500 000 suffer from potentially fatal dengue and need hospitalization.⁴

Clinical presentation of dengue varies from a symptomatic infection to undifferentiated fever (viral syndrome), classic dengue fever, or dengue hemorrhagic/shock syndrome. The frequency of severe cases depends on serotypes, age, and immune status of the patient.^{4,5}

Dengue is characterized by three phases: febrile, critical, and recovery phase. The febrile phase is characterized by fever, retro orbital headache,

myalgia, low back pain, arthralgia, flushing of face and skin, anorexia, nausea, vomiting, abdominal pain, petechiae over upper and lower extremity, itching, and progressive decline of platelet count. After disappearance of fever and resolution of febrile phase symptoms, a small percentage of patient progress to the critical phase that characterized by increased capillary permeability that leads to leakage of plasma and increase of hematocrit.¹

2. Case presentation

A 20-year-old male known to have sickle cell anemia presented to emergency department with fever, bone pain, and myalgia for two days. On examination he was fully conscious, with a temperature of 38 °C, blood pressure of 110/70 mmHg, puls 98 bpm, and respiratory rate 17 cycle/min. He had pallor, jaundice, and tenderness over both lower limbs and back. No other abnormalities could be detected on physical examination. This clinical presentation was compatible with the sickle cell crisis precipitated by infection. Due to presentation of fever during rainy season in an endemic area of dengue virus, we suspect dengue virus infection and a viral panel was requested.

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* Corresponding author at: Department of Internal Medicine, Faculty of Medicine, Al-Azhar University Assiut, Assiut, 71524, Egypt. Fax: +2-088-2180445. E-mail address: a.madian@azhar.edu.eg.

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2.1. Investigations

Preliminary lab results revealed, anemia (hemoglobin: 8.6 gm/dl), hematocrit (23.7), corrected reticulocyte (2.8 %), thrombocytopenia (116 000), leukocytosis (24 000), CRP 25. NS1-antigen tested positive and dengue serology was positive for IgM and positive for IgG. Elevated liver enzymes (aspartate transferase:195 U/l, alanine transferase:132 U/l) and total bilirubin (3.1 mg/dl). Normal electrolytes and renal panel. Based on these findings our diagnosis was dengue infection affecting patient with sickle cell anemia.

2.2. Treatment and outcomes

The patient was treated with intravenous paracetamol and normal saline. On the evening of the second day of admission, despite of antipyretic and fluid infusion patient was feverish, with cold extremity and dyspneic. Complete blood picture showed (hemoglobin: 8.6 gm/dl), increased hematocrit (27.2 %), progressive thrombocytopenia (66 000), leukocytosis (22 000) and CRP (25). Electrolyte disturbances, hypernatremia (150 mmol/l), hyperkalemia (5.7 mmol/l). Chest radiography revealed multiple variable sized patchy areas of consolidation are seen scattered among both lungs giving a fluffy-cotton appearance. At this time, we faced diagnostic challenge were these radiological findings compatible with plasma leakage into alveolar spaces or acute chest syndrome? Patient transferred to intensive care unit and within 2 h patient developed severe pallor, peripheral cyanosis, hemoptysis, hypotension (his blood pressure declined to 60/40 mmHg), anuria and reduction of hemoglobin to (4.8 gm/dl), and hematocrit decreased to (13.4 %). At this stage alveolar hemorrhage and hemorrhagic shock was highly suspected. Patient intubated and central line inserted and received packed red blood cells. Repeated chest radiography (Portable) revealed diffuse haziness in both lungs with ill-defined patchy opacities. A multidisciplinary team was initiated and blood exchange was decided. During blood exchange, abdominal ultrasound repeated and revealed marked colonic distension without peritoneal collection. However, shock was irreversible both pupils became nonreactive to light and patient died during blood exchange due to cardiac arrest. No autopsy was performed.

3. Discussion

Endothelium plays a central role in inflammation and coagulation. Several studies reported that sickle

cell disease is characterized by chronic systemic inflammation that results in tissue injury and endothelial dysfunction. In addition, monocytes are in a pre-activation state which amplify production of inflammatory cytokines.⁶

Martina and colleagues proposed that dengue virus might replicate in endothelium. This will result in release of inflammatory cytokines, increased capillary permeability, plasma leakage and finally hypovolemic shock.⁷

In our case, patient developed acute respiratory distress, hypovolemic shock and hemoptysis. This could be explained with plasma leakage then intra alveolar hemorrhage triggered by dengue infection. Selective replication of dengue virus within pulmonary endothelium could explain localization of leakage and hemorrhage within alveoli.

At cellular level, dengue infection triggers increased expression of adhesion molecules to the pulmonary capillaries, amplified production of inflammatory cytokines, exaggerated oxidative stress. This facilitates interactions between inflammatory cells and red blood cells, and vascular endothelium, resulting in disruption of alveolar blood barrier, plasma leakage and alveolar hemorrhage. These events in turn result in a 'vicious' cycle of lung injury and hypoxemia.^{6,7}

In treating patients with sickle cell anemia and dengue infection we are facing a great dilemma. An increase of hematocrit is a sign of plasma leakage with subsequently a decrease that may be due to hemorrhage. On the other side, in sickle cell anemia, it is preferred to keep hematocrit low to decrease the risk of Vaso-occlusion. So, depending on hematocrit alone should not be used as an index for plasma leakage or hemorrhage in sickle cell anemia with dengue infection. In addition, it is better to follow those patients with chest radiography and abdominal ultrasound to detect plasma leakage/hemorrhage as early as possible. As a result, interpretation of blood indices in combination with chest and abdominal imaging is mandatory during the management of sickle cell patients with dengue infection.

The fatal outcome of dengue infection reported among sickle cell patients could be explained by endothelial dysfunction and preactivation state observed in sickle cell patients.

Unfortunately, there is no available guideline to highlight the way to treat such patients, and clinicians in areas with a high prevalence of sickle cell anemia and endemic dengue should be aware of the challenges they will face in this battle.

3.1. Learning points

- (1) Interpretation of blood indices in combination with chest and abdominal imaging is mandatory during the management of sickle cell patients with dengue infection.
- (2) Clinical presentation of dengue infection mimics Vaso occlusive crisis of sickle cell anemia regarding headache, bone pain, back pain, and myalgia. Therefore, clinicians should be aware of this differential in areas with endemic dengue and a high prevalence of sickle cell anemia.

Conflicts of interest

None declared.

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