Pulmonary Haemorrhage Syndrome Associated with Dengue Haemorrhagic Fever

SK Sharma*, BS Gupta**, G Devpura***, A Agarwal****, S Anand*

Abstract
Dengue fever is a major public health problem in India. Dengue haemorrhagic fever (DHF), a more serious form of disease, occurs when a person previously infected with dengue is reinfected with a different serotype. Besides common manifestations pleural effusion, pneumonitis, haemoptysis and pulmonary haemorrhage have rarely been seen. We report a case of 30 years old male, who developed pulmonary haemorrhage, haemoptysis requiring blood transfusion. Serology was consistent with the diagnosis of dengue haemorrhagic fever. ©

INTRODUCTION
Dengue fever is caused by four different serotype of dengue virus viz. DEN-1, DEN-2, DEN-3, and DEN-4. Infection with any one of these serotype leads to classic dengue fever and life long immunity to that particular serotype. However, dengue haemorrhagic fever (DHF), a more serious type of dengue, occurs when a person is infected with a second different serotype. In this report, we describe a patient with DHF who subsequently developed pulmonary haemorrhage that started resolving during treatment.

CASE REPORT
A 30 years Hindu male patient without significant past medical or surgical history presented with a one week history of low grade fever and continuous nonproductive cough followed by generalized rashes and haemoptysis.

He gave no history of diarrhoea, vomiting, pain abdomen, joint pains, leg swelling, weakness in limbs, chest pain, drug ingestion, or bleeding disorder. He did not smoke or consume alcohol. There was no history of hypertension, diabetes, or tuberculosis.

On examination patient was conscious, alert, oriented to time, place, and person. Pallor was present. The heart rate was 88/min. The blood pressure 130/80 mmHg and there was no postural hypotension. Oral temperature was 37.4°C. Examination of oral cavity revealed no active source of haemorrhage. A diffuse, nonblanching, confluent rash was present on his back, buttocks, palms, soles, and abdomen.

Respiratory rate was 28/ min. Chest examination revealed end inspiratory crepts. Examination of other systems was unremarkable.

Initial laboratory investigations showed total leukocyte count 5440/mm³, haemoglobin 11.1 g/dl, haematocrit 32%, and platelets 0.41 lakh/mm³. Peripheral blood film was normal except for reduced platelets.

Serum electrolytes and renal profile were normal. AST and serum bilirubin were slightly deranged, rest of liver profile was normal. Prothrombin time, bleeding time, clotting time, aPTT, CPK, and LDH were normal. Urine analysis showed pH of 6.0 with no proteins, blood, bilirubin; microscopic examination revealed 8 RBC / HPF; no pus cells, or urinary cast. Dengue serology IgM and IgG were positive. Serology screening for HIV, hepatitis B virus, hepatitis C virus was negative. Anti- nuclear antibody, rheumatoid factor, C-reactive protein, ASLO, c-ANCA, p-ANCA were negative. Peripheral blood film for malarial parasite was negative. Malarial antigen was negative. FDP < 5µg/ml and d-dimer < 1µg/ml were in normal range.

Chest X-ray showed fluffy shadows in both lung fields (Fig. 1). Abdominal ultrasound showed minimal ascites. High resolution CT thorax showed patchy ground glass opacities in both lungs predominantly in central and basal lung fields consistent with pulmonary haemorrhage (Fig. 2A). Gastroscopy and stool examination were normal. Bronchoscopy biopsy was not done because of haemorrhagic manifestation.

The patient was treated conservatively. He received random donor platelet transfusions, and intravenous fluids according to his volume status and urine output. He improved over next seven days; a repeat platelet count was 190,000/mm³ HRCT thorax showed remarkable improvement with only sparse patchy opacities in either lung (Fig. 2B).

*Resident; **Professor; ***Associate Professor; ****Assistant Professor, Upgraded Department of Medicine, SMS Medical College, Jaipur. Received: 4.4.2007; Revised: 23.8.2007; Accepted: 28.8.2007
DISCUSSION

The patient had clinical spectrum consistent with DHF. Serological analysis was consistent with the diagnosis. Typical manifestation of DHF include an initial fever usually lasting 2 to 7 days. After defervescence, patient usually has thrombocytopenia, signs of haemoconcentration, petechiae, purpuric lesions and ecchymosis. Less frequent symptoms include epistaxis, bleeding gums, gastrointestinal haemorrhage, haematuria and intracranial haemorrhage. Pulmonary manifestations such as pneumonitis, pleural effusion, haemoptysis, and pulmonary haemorrhage are rarely seen in DHF. Haemoptysis has been reported in 1.4% of dengue infection. The pathogenesis of bleeding in DHF patient is not well understood. It is thought to be a multifactorial process with abnormalities in the coagulation cascade, thrombocytopenia, platelet dysfunction, disseminated intravascular coagulation, vascular defects and increased vascular permeability thought to be mediated by histamine.

REFERENCES


Announcement

National Haematology Update - VII and A Symposium on Lymphoplasmacytic Disorders, February 23rd-24th, 2008. Organized by the Department of Hematology, All India Institute of Medical Sciences, New Delhi.

For further details, please contact: Dr. Seema Tyagi, Organizing Secretary, NHU-VII, 2008, Department of Hematology, AllMS, New Delhi 110029.
E-mail: drseematyagi@hotmail.com