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Dengue Hemorrhagic Fever with Disseminated Intravascular Coagulation

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Abstract: ***Dengue fever is the worlds most important viral hemorrhagic fever disease, caused by arbovirus having four antigenically virus serotypes of the family flaviviridae. It causes wide clinical spectrum of disease. We report a case of dengue hemorrhagic fever complicated by DIC. The initial picture of classical dengue fever was followed by vomiting, hematemesis and hematochezia. Severe liver injury with SGOT more than 1000 IU/lt and SGPT 694 IU/lt with hematocrit of 52.4%, platelet count less than 15,000 and deranged coagulation profile with INR- 8.4 and aPTT-52.4 sec, with DIC score of 7.***

Keywords: Dengue, Dengue hemorrhagic fever, Disseminated intravascular coagulation

1. Introduction

Dengue infections caused by the four antigenically distinct dengue virus serotypes of the family flaviviridae are most important arboviral diseases in humans, most important viral hemorrhagic fever affect upto 100 million individuals per year with two-fifths of the world population being at risk[1]. Dengue infection may be asymptomatic or may lead to an undifferentiated fever, dengue fever or dengue hemorrhagic fever (DHF) [2]. All 4 serotypes of dengue virus can cause DHF, but this is more likely to occur with secondary infections with a different serotypes [3]. Immunity is antibody mediated. After acute infection by a particular serotype there is antibody response to all four serotypes. There is long lasting immunity to the homologus serotype of the infected strain. A cross reactive heterotypic immunity has been reported by Sabin [4] to last about 2 to 12 months [5]. It is suggested that second infection with a heterotypic virus which differs from the first one, pre existing enchance viral uptake and replication in the mononuclear phagocytes which trigger the production of mediators with activation of compliment and the clotting cascade and eventually produce DHF [6].

2. Case Report

A 23 year old gentleman, contractor from Mumbai presented to Medicine Outpatient department of Krishna Hospital, Karad, with a history of fever with chills and generalized weakness for 4 days, vomiting, burning micturition and cough with expectoration for 3 days and aphasia 15 minutes prior to attending Outpatient department. There was no rash or bleeding from any site. The fever was continuous and was associated with chills and headache. It was relieved by drugs which he received form a general practitioner. The vomiting was non bilious, non projectile, 2-3 episodes per day and was associated with nausea. Aphasia was sudden in onset and not associated with loss of consciousness or convulsions.

Patient was a chronic cigarette smoker since 4 years. There was no history of loose stools. There was neither remarkable drug history nor history of blood transfusion.

On general physical examination patient was aphasic, conscious and oriented with temperature of 102° F , conjuctival congestion and right eye mild ptosis. BP was 110/80 mm Hg with pulse rate of 126/min.

On systemic examination there was right hypochondriac tenderness with no organomegaly. CNS examination showed absent bilateral triceps and bilateral knee jerks with weak gag reflex. Examination of respiratory system and cardiovascular system revealed no abnormalities.

Based on history and clinical examination differential diagnosis of Malaria, Enteric fever, leptospirosis and Dengue fever was considered. Initial investigations revealed Hb- 18.2gm%; TLC- 5220/cumm; N- 68, L- 32; platelet count-15,000/cumm; PCV- 52.4%; MCV- 86.8fl and deranged coagulation profile PT- 73 sec, INR- 8.4(Day 1) and abnormal aPTT - 54.2 sec, urea- 50mg/dl, creatinine-1.7mg/dl, sodium- 134mg/dl, potassium-2.4mg/dl, calcium-6.3mg/dl. Deranged liver functions(total bilirubin – 1.0mg/dl,Direct-0.2mg/dl,Indirect-0.8mg/dl; AST>1000IU/l; ALP- 64IU/l); peripheral blood smear for malarial parasite and WIDAL test for enteric fever were negative. Rapid test for leptospira (IgG and IgM antibodies) were negative. HBsAg, Anti-HCV Ab, Anti-HAV, Anti-HEV were also negative. As the patient was residing in Mumbai which is endemic and high prevelance area for Dengue and platelet count were on decreasing trend, the patients serum was tested for Dengue serology (IgM,IgG,NS1 Ag) which showed NS1 Antigen positive. During the period of hospital stay there was marked deterioration in patient's general condition and was progressive derangement of coagulation profile, patient started having malena within 6 hours of admission followed by dark coloured blood with clots per rectum. He also had ecchymotic patches at the site of intravenous cannula insertion. Patient was transfused FFP for profuse bleeding per rectum. As patient became disoriented within 8 hours of admission to rule out intracranial bleed CT Head was done which was normal. INR on Day 2 after FFP transfusion became 2.2. On Day 2 his Hb reduced to 8.3gm%, TLC- 3780/cumm, platelet count- <15,000/ccmm and due to persistent bleeding tendencies, platelets and PCV were transfused. Fibrin

degradation products (FDP) was >20µg/ml. DIC score was 7. His respiration became irregular, with drowsiness and low oxygen saturation, patient was intubated and kept on ventilator support. During the course of treatment, patient's BP started falling gradually for which he required ionotropic support.

Patient was treated with ceftriaxone, Vitamin K, blood and blood products, adequate intravenous fluid and other symptomatic support. Inspite of close monitoring and active management, patient's condition deteriorated and unfortunately succumbed 32 hours after admission. With history, clinical examination and investigations he was diagnosed as a case of Dengue Hemorrhagic Shock with Disseminated Intravascular Coagulation.

3. Discussion

Patient presented with classical symptoms of dengue fever and was complicated with Dengue Hemorrhagic fever and Disseminated Intravascular Coagulation. Asia has been area of highest endemicity, with all four dengue serotypes circulating in the large urban centres in most countries.[7] The patient had all the criteria which was established by World Health Organization for clinical diagnosis which are follows: high continuous fever for 2-7 days; a hemorrhagic diathesis; hepatomegaly and shock; together with two laboratory changes thrombocytopenia with concurrent hemoconcentration (hematocrit elevation of 20% or more) [8]. The pathophysiological hallmarks of DHF are plasma leakage and abnormal hemostasis. A significant loss of plasma leads to hypovolemic shock and death. A disorder in hemostasis involves all major components. (1). Vascular changes including capillary fragility changes that leads to a positive tourniquet test and easy bruisability ; (2) thrombopathy, with impaired platelet function and moderate to severe thrombocytopenia; (3) coagulopathy, acute type disseminated intravascular clotting is documented in severe cases, often with prolonged shock and responsible for severe bleeding [9].Evidence for DIC includes hypofibrinogen and presence of fibrinogen degradation products (FDP) and raised D-dimer values [10] which was present in our case (FDP score-7).

4. Message

This case jolted wide awake because of rapid deterioration of the patient unfortunately leading to death due to severe life threatening complications of dengue hemorrhagic fever with DIC which kills hundreds of people across India every year. Noteworthy is the fact that errantly growing metropolitan cities are devoid of basic sanitation facilities due to inadequate housing and overcrowding.

5. Conflict of Interest

It is declared that all the authors of this report have no conflict of interest.

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