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A CASE OF SEVERE DENGUE FEVER PROGRESSING TO MULTIORGAN FAILURE: REPORT FROM RURAL CRITICAL CARE IN INDIA

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ABSTRACT

Dengue fever is a major public health problem in India. Shock syndrome is a dangerous complication of dengue infection and is associated with high mortality. Increased vascular permeability, together with myocardial dysfunction and dehydration, contribute to the development of shock, with resultant multiorgan failure. Diagnosis is largely clinical and is supported by serology and No specific methods are available to predict outcome and progression. We report a case of severe dengue fever that later developed multiorgan failure and was successfully treated in our ICU. In conclusion, we suggest that clinicians in areas where dengue fever is endemic should be made aware of these unusual complications of Dengue fever and appropriate therapy without delay results in better outcome.

KEYWORDS: DHF (Dengue hemorrhagic fever), DSS Dengue shock syndrome, ARDS (Acute respiratory distress syndrome), AKI (Acute kidney injury).

INTRODUCTION

Dengue is one of the most important tropical viral diseases of humans in the world today and also is a major public health problem in India. Global incidence of dengue has grown dramatically in recent decades. The World Health Organization estimates that there are 50 to 100 million infections yearly, including 50,000 Dengue Hemorrhagic Fever (DHF).[1] Dengue hemorrhagic fever (DHF) is a more serious form of the disease. As dengue infections have

become more common, an increasing number of DHF cases are associated with unusual presentations.

The pathophysiological basis for the development of severe dengue, characterized by plasma leakage and the "shock syndrome" are poorly understood. No specific treatment or vaccine is available, and careful monitoring and judicious administration of fluids forms the mainstay of management at present.[2]

Patients with severe dengue die of progressively worsening shock and multiorgan failure. The exact mechanism of this phenomenon is not fully understood although it is thought that increased vascular permeability occurs largely due to malfunction of vascular endothelial cells induced by cytokines or chemical mediators [2] as also occurs in severe sepsis.

We report a case of severe dengue fever that later developed multiorgan failure and was successfully treated in our ICU.

CASE REPORT

A 17 year old male patient, presented with high grade fever for 6 days, pain abdomen & vomitting for 3 days (Fig 1). He has no other co morbid medical conditions.

On examination he was afebrile, conscious and coherent, spontaneous breathing was labored with severe tachypnoea+ (40/min), dyspnea and maintaining saturation of 90% on 10L O2/ min, severe dehydration with hypotension (60/40 mmHg) and tachycardia (140/min). Evidence of petichae/ bleeding from the gums and icterus noticed. Chest auscultation revealed bilateral basal crepitations. Per abdomen exam showed minimal distension, tender hepatomegaly with diffuse tenderness (Fig 1).

His investigations revealed hemoglobin: 19gm%, PCV: 54 & platelets of 10,000. His liver function tests were deranged with AST: 9440, ALT: 3180, Total bilirubin 2.8 & direct bilirubin of 1.8. His coagulation profile was abnormal with PT: 25, APTT: 45 & INR: 2.4. His chest x-ray revealed bilateral massive pleural effusions, 2D echo (FATE) showed IVC: 1cm, mildly dilated RA& RV & fair LV function with EF: 45-50% and bilateral pleural effusion, minimal pericardial effusion and USG abdomen showed hepatomegaly, edematous gall bladder with Ascites. His ABG with 10L O2 showed compensated metabolic acidosis with hyperlactatemia (base excess of -13.2, bicarbonate of 10 & Serum lactate of 76mg %). His dengue serology revealed NS1 & IgM positive.

He was initially resuscitated with crystalloids and started on Non- invasive ventilation and was transfused 4 units of platelets (RDP) (Fig 2) & 4 units of FFP and serial hematocrit monitored. In view of severe plasma leakage with third spacing, he was started on Human Albumin 5% infusion (25ml/hr). Gradually, his shock and acidosis resolved and coagulopathy was corrected.

Second day, respiratory failure worsened, necessitating intubation & ventilation and chest x-ray revealed bilateral non homogenous opacities and ABG showed severe hypoxemia with PF ratio of <150, consistent with moderate ARDS. He developed fever (101F), hypotension requiring pressor supports, liver injury worsened (Fig 2) and serum creatinine increased (1.5), hence started on empirical antibiotics after sending appropriate cultures (fig: 1).

Next few days (3^{nd} to 5^{th} day), he was continued to be ventilated as per ARDSnet protocol, hemodynamically stable and weaned off pressor supports on 5^{th} day, was transfused 4units of platelets on 5^{th} day (Fig 2) in view of hematuria and platelet count of 44000. His blood & ET cultures yielded no growth and Antibiotics were stopped on 5^{th} day.

Subsequent days (6^{th} to 9^{th} day), his vitals were stable, hematuria stopped and platelet count showed upward trend. Gradually, his liver injury and acute kidney injury were resolving by 6^{th} day. He was extubated on the 7^{th} day and transferred to ward on 9^{th} day (Fig 1).

DISCUSSION

Dengue disease ranges from asymptomatic or self-limiting dengue fever (DF) to severe dengue characterized by plasma leakage (dengue hemorrhagic fever [DHF], grades 1 and 2) that can lead to a life-threatening syndrome (dengue shock syndrome [DSS], grades 3 and 4)**{1}**. The combination of the NS1 antigen and antibody tests could increase the diagnostic efficiency for early diagnosis of dengue infection.[3]

Liver enzyme elevations are common in dengue. Usually the SGOT levels are more than SGPT levels probably due to skeletal muscle injury.[4] Haemostatic derangements in DHF are a multifactorial mechanism. Vasculopathy, thrombocytopenia, platelet dysfunction were found in most cases.[5]

Pulmonary manifestations are uncommon in this disease, except for pleural effusions. ARDS is the most serious manifestation of acute lung injury which arises as a complication of a widespread systemic response to acute inflammation or injury. The etiology for ARDS is

varied and Dengue is reported as a rare cause for this condition.[6] Of late there are increasing number cases of ARDS being reported in dengue patients. Wang et al reported an incidence of 1.8% ARDS in their study involving 606 dengue patients in China.[7]

AKI seems to be a frequent complication of severe dengue. Its etiopathogenesis is probably multifactorial, caused by intense systemic inflammation, hemodynamic instability, hemolysis, rhabdomyolysis and acute glomerulitis. Currently, there are no specific recommendations for either conservative treatment or dialysis of patients with dengue, and the effects of AKI on the quality of life, survival and kidney function of survivors are unknown.[8]

Our case reported here were clinically diagnosed to be DSS based on WHO criteria. Dengue fever was confirmed with bedside serology (Ns1 & IgM positive). Resuscitation with crystalloids followed by Human Albumin 5% infusion in the initial phase of shock [9] was beneficial. A diagnosis of ARDS was made on the basis of chest X-ray findings and the PaO2/FiO2 ratio, in accordance with ARDS berlin definition [10] and accordingly ventilated as per ARDSnet protocol.[11] The good prognosis of the patient could be attributed to patient's younger age, minimal delay in administering necessary treatment and good supportive care.

In conclusion, we suggest that clinicians in areas where dengue fever is endemic should be made aware of these unusual complications of Dengue fever and appropriate therapy without delay results in better outcome, including in a rural critical care setting.

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