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Acute Liver Failure in Dengue Shock Syndrome

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Abstract:- Dengue is a viral disease common in tropical countries. It frequently affects the liver, and its symptoms can range from transaminitis without symptoms to abrupt liver failure[i]. This case report features the significance of acute liver failure in dengue shock syndrome.

Keywords:- *Acute Liver Failure, Dengue Shock Syndrome.*

I. INTRODUCTION

Dengue fever is a viral infection that can progress to viral haemorrhagic fever. Dengue fever is transmitted by the Aedes aegypti mosquito. Dengue virus is an RNA virus of the genus flavivirus. It has an incubation period of 7 days. Dengue shock syndrome can deteriorate a patient's general condition and may even lead to death[ii]. Fever and at least two of the following symptoms ocular pain, headache, muscle/joint pain, rash, or leukopenia characterize dengue fever. Three phases of symptoms the febrile phase (lasting 2–7 days), the critical phase (lasting 3–7 days and characterized by vascular leakage, shock, and/or organ damage) followed by a convalescent phase[iii]. In this case report we discuss in detail about the acute liver failure in dengue shock syndrome. Although it also affects other organs, dengue primarily damages the liver[iv].

II. CASE REPORT

30-year-old male patient known case of anxiety disorder was presented to medical ICU with c/o fever, headache and joint pain for 5 days, insidious in onset, progressive in nature, associated with chills. The patient, who was employed in Mumbai, visited a local hospital there and got dengue NS1 tested positive. On examination, BP- 80/50 mmHg with NORAD infusion and IV Fluids HR:150 bpm, Regular rhythm RR:46cpm Spo2: 80% in RA >96% with face mask at 6 l/min GRBS:92mg/dl systemic examination: RS: air entry decreased in bilateral basal area, normal vesicular breath sound heard, P/A: Abdomen tense, shifting dullness present, bowel sound present CVS: Normal S1, S2, No murmur. CNS: Conscious, oriented, anxious. Blood investigations(Day 1)showed Hb:23.8, PCV 64.2% TC:24100, N:70, Platelet:0.14 (>1.50 lakhs), LFT showed Total bilirubin:3.25 (0.0 - 1.2 mg/dL), DIRECT BILIRUBIN: 2.43 mg/dL SGOT:6817 (0.0 - 50.0 IU/L), SGPT:3392 (0.0 - 40.0 IU/L), PT INR:2.05(<1), ALP: 427.0 IU/L (35.0 - 129.0 IU/L) Total protein 5.1 (6.4 - 8.3 g/dl), S. albumin - 2.8 (3.5 - 5.2 g/dl) S. globulin - 2.3 (2.0 - 3.5 g/dl), S. calcium - 6.3 (8.5 - 10.1 mg/dL) CRP: NEGATIVE, DENGUE NS1: POSITIVE, LEPTOSPIRA IgM: Negative, LEPTOSPIRAL IGG ANTIBODIES: Negative, HBs Ag CARD: Negative HCV (CARD): Negative HIV 1+2 CARD: Negative, RAPID MALARIAL TEST(RMT): Negative, URE showed 2-5 pus cells, Bacteria+ and 25-30 RBCs, urea – 43 (8.0 - 49.0 mg/dl), serum creatinine- 2.6 (0.7 - 1.2 mg/dl) serum sodium- 144 mmol/l (135.0 - 148.0 (mmol/l), serum potassium- 5.1 mmol/l (3.5 - 5.1 mmol/l). Arterial line inserted into the right Radial artery. On 28/10/22, at 10:30 pm, bystanders were counselled regarding the need for intubation in view of refractory septic shock, GCS drop and respiratory distress, and intubation was done with 8.5 size ET tube, fixed at level 21 and position was confirmed. ARDS Net protocol was followed and the patient was ventilated with PEEP-16, but the patient was still not able to maintain adequate saturation levels. Surgery consultation was placed in view of the tense abdomen and RT aspirate containing blood and was advised USG Abdomen. Gastroenterology consultation was placed in view of the same and suggested UGI Endoscopy when platelet and coagulopathy have been corrected. 6-pint Platelet, 4-pint FFP and 1-pint PRBC transfusion was done. During the course in the hospital, he was treated with antibiotics (INJ Piperacillin tazobactam, C. Doxycycline), inotropes, colloids, steroids (INJ Hydrocortisone-sepsis dose), PPI, antipyretic, IV Fluids and other supportive measures.

2nd Day Platelet:0.23 ALT / SGPT: 4168.0 IU/L AST / SGOT: 12060 IU/L Bilirubin indirect: 0.90 MG/DL Direct bilirubin: 2.43 mg/dl Globulin, Serum: 1.4 G/DL Total Bilirubin: 3.33 MG/Dl Total Protein: 3.9 G/DL, PT-CONTROL: 13.8 sec, PT-INR: 5.44, PT-TEST: 75.1 sec, Potassium: 5.1 MMOL/L Serum Creatinine: 2.7 MG/DL Sodium: 144 MMOL/L Urea: 43.0 MG/DL, the patient was started N-acetylcysteine (NAC) Loading dose: 150 mg/kg IV; mix in 200 mL of D5W and infuse over 1 hr followed by 50 mg/kg IV in 500 mL D5W over 4 hr, Then 100 mg/kg IV in 1000 mL D5W over 16 hr and other supportive measures.

3rd day / SGPT: 2301 IU/L AST / SGOT: 6235 IU/L, PT-INR: 2.1 platelet count also started improving. In subsequent days patient started improving and we were able to extubate him and stop the support. Later we shifted him to the ward.

III. DISCUSSION

The most prevalent arboviral illness in the world is dengue[v]. Dengue hemorrhagic fever affects more than 500,000 people annually and results in at least 12,000 fatalities. Three symptoms are seen in patients: hemorrhagic manifestations, signs of plasma leakage, and 50% platelet levels. Symptoms can start to show up 2–21 days after exposure, however the majority of individuals get sick in just 9 days[vi]. The patient first presents with fatigue, fever, headache, and muscle pains, and the illness

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can progress to multiorgan failure and hemorrhaging. Survival in severe infections depends on careful volume-replacement therapy to maintain blood pressure and intravascular volume[vii]. Dengue causes liver damage through multiple mechanisms viz direct viral effects on hepatocytes and Kupffer cells, immunologic hyperactivity via a T cell-mediated cytokine storm, and circulatory failure that leads to decreased hepatic perfusion[viii].

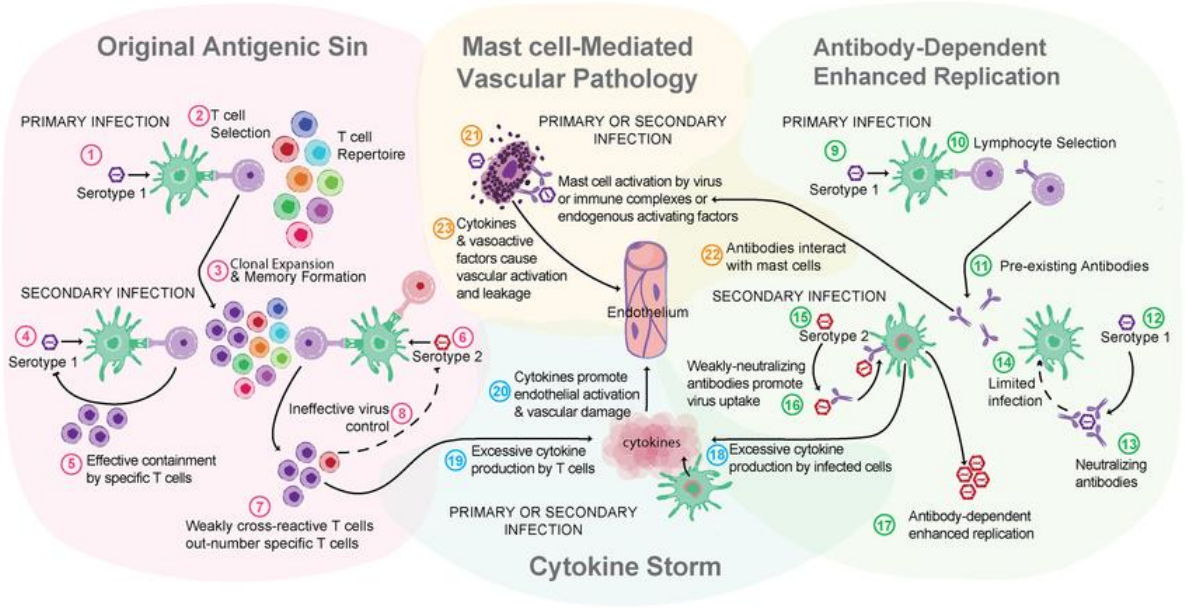

Fig 1: Dengue immune pathogenesis

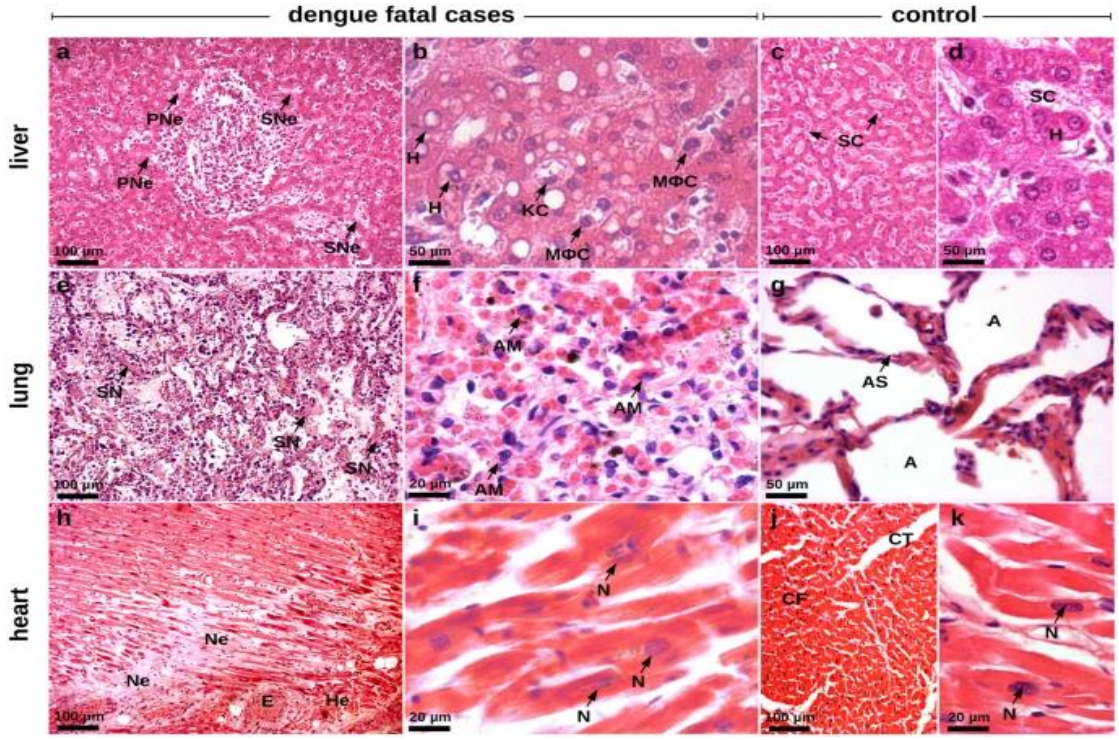

Fig 2 : Histological injuries in dengue fatal cases[ix]

From a liver aspect, dengue patients frequently report hepatomegaly with or without jaundice, along with abdominal pain, nausea, emesis, and anorexia. Transaminitis, hyperbilirubinemia, hypoalbuminemia, and increased INR are among the laboratory results[x]. These conventional signs were present in our patient, but the severity of her transaminitis made

her presentation distinct. Only 4% of cases of transaminitis have an increase in AST and ALT levels by 10 times the upper limit of normal. Elevated AST and ALT levels are present in 63%-97% and 45%-96% of dengue patients, respectively. 3,5 She most likely had a very severe liver injury. In addition to the direct hepatic consequences of dengue, she also had a serious hemodynamic deficit that raised the possibility of ischemic hepatitis. Moreover, liver injury decreases APAP metabolism and APAP-related hepatotoxicity can happen with multiple, small overdoses. Given her initial detectable APAP level, and that she received 3 doses of 650 mg in-house (to control fever before concern for APAP-related liver injury), this potentially contributed to hepatic injury.

Since there is no direct antiviral therapy, dengue treatment is supportive. Oral fluid intake is recommended, and blood products and intravenous fluids are administered as needed. Given that vascular leakage mixed with fluids predisposes patients to respiratory compromise, as happened with our patient, fluids should be used cautiously during the critical illness period[xi].

NAC is another element of therapy. By neutralizing free radicals, enhancing systemic hemodynamics, and maximizing tissue oxygen transport, NAC avoids hepatic damage. As was done with our patient, case reports advise providing NAC early in the course of ALF-associated DSS[xii].

Those who made it through the initial one to two days of the critical sickness phase reached the convalescent period shortly after the peak of the transaminitis, and they thereafter recovered with only supportive therapy; this was the case with our patient. Only one patient in that case series had the most severe case of dengue, called DSS, and that patient recovered without needing a transplant. Adult patients with DSS-associated ALF rarely respond to supportive treatment alone and remission rates are low.

IV. CONCLUSION

Acute liver failure in dengue shock syndrome has responded to N-acetylcysteine. A liver transplant can be explored in the uncommon event of deterioration during the critical illness period, with a newly reported successful transplant in dengue-associated ALF as evidence.

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