

Dengue Shock Syndrome in New Born – A Case Series

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Four cases of dengue shock syndrome were seen during an epidemic of dengue fever. Three cases recovered following appropriate management.

Key words: *Dengue, Neonate, Shock.*

Dengue fever is an acute febrile illness caused by four serotypes of Dengue virus and in older children characterized by biphasic fever, myalgia, arthralgia, rash and leucopenia. Dengue hemorrhagic fever (DHF) is characterized by hemoconcentration, abnormality of hemostasis and in severe cases by a fluid & protein losing shock syndrome (Dengue Shock Syndrome, DSS). This arthropod born virus is transmitted by a day time biting mosquito *Aedes aegypti*. There is no cross protection between the 4 dengue serotype but there is cross reaction(1). The disease usually establishes a pattern of epidemic activity every 2-5 years. DHF and DSS are usually confined to children with a modal age at hospitalization of 4-6 years(2). Only 9% of the cases in a recent epidemic in Delhi were infants and the youngest child was 3-month-old(3). We are reporting 4 cases of DSS in neonatal period seen during recent

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epidemic of dengue fever in Rajasthan (August-November, 2001).

Case 1

A 28-day-old term neonate was admitted with fever, refusal to feed and excessive cry for two days. There was no history of rash, convulsion or bleeding from any site. On examination the heart rate was 150/min, respiratory rate 48/min, temperature 38.5°C per axilla with normal perfusion. Neonatal reflexes were poor. The neonate was treated with a presumptive diagnosis of neonatal septicemia with parenteral cefotaxime and amikacin. Investigation revealed hemoglobin 13 g%, total leukocyte count 6300/mm³, DLC (N24L70M6), platelet count 1 Lakh/mm³ and negative serum C-reactive protein. The following day baby was in shock. As there was ongoing dengue epidemic the case was investigated for it. Investigation revealed hemoglobin 14g%, platelet count 36000/mm³, SGOT 1386U/L, SGPT 1858 U/L, Stool for occult blood positive and positive serum IgG and IgM for dengue. Mother also had positive IgG and IgM for dengue. The shock was managed with appropriate fluid and dopamine. Baby improved within 72 hours and was discharged after 10 days.

Case 2

A four day old term male neonate was admitted with fever, lethargy and refusal to feed for two days. There was no history of rash, convulsion or bleeding from any site. Maternal history was non-contributory. On examination baby had normal heart rate, respiratory rate, temperature and peripheral perfusion but was lethargic. The neonate was managed with a presumptive diagnosis of neonatal septicemia. Initial investigations were normal except for a positive C-reactive protein. The next day baby was in shock with bleeding per rectum. Investigation revealed

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platelet count 50,000/mm³, prothrombin time 18 seconds and normal PTTK. Serology for dengue (IgG and IgM) was positive. Chest X-ray revealed right side pleural effusion. He was treated with appropriate fluid management and platelet rich plasma. Baby improved and discharged after 7 days.

Case 3

A 34 week premature neonate presented on day seven of life with shock, rectal bleeding, progressive distension of abdomen and increasing pallor. The baby had thrombocytopenia and weakly positive IgG and negative IgM for dengue. Five day later IgG and IgM for dengue were positive. Despite aggressive management of shock baby deteriorated and expired on day 20 of life.

Case 4

A full term baby was admitted on day four of life with history of delayed cry after birth, seizures and unconsciousness. On examination baby had altered sensorium with subcostal and intercostals retraction, bilateral crepitations and wheezing. The baby had a positive serum C-reactive protein. On day 11 baby developed shock. Investigation revealed low platelet count and positive IgM for dengue. There has history of fever in last trimester in mother and investigation showed IgM and IgG for dengue positive in mother serum. Baby improved with supportive management.

Discussion

Dengue virus infection results in a spectrum of disease. On one end is dengue fever characterized by fever, myalgia, arthralgia, leucopenia and lymphadenopathy, while on the other end is DHF characterized by hemorrhage and shock syndrome(4). The disease and particularly severe DHF/DSS form has a predilection for pediatric age group

with a modal age at hospitalization of 4-6 years(2).

According to WHO criteria, for defining DHF the following must all be present (a) fever, (b) hemorrhagic tendency (c) thrombocytopenia (d) evidence of plasma leakage, manifested by either a rise in the hematocrit equal to or greater than 20% above average for age, sex and population or signs of plasma leakage such as pleural effusion, ascites and hypoproteinemia. For defining DSS all of the above four criteria for DHF plus evidence of circulatory failure manifested by rapid and weak pulse, narrow pulse pressure (less than 20 mmHg), hypotension for age or cold, clammy skin must be present(2).

DHF/DSS occurs with higher frequency in two immunologically defined groups: children who have experienced a previous dengue infection, and infants with waning levels of maternal dengue antibody. Various studies reported its rarity in early infancy and neonatal period(2).

In 1996 epidemic in Delhi, 23% cases of DHF/DSS was in 0-3 years age group and the youngest child was 3-months-old(3). Another study of the same epidemic reported a four months old infants as the youngest patient(5). In Calcutta epidemic in 1990, no case of DHF/DSS was reported in infancy(6). Recently, we came across four cases of DSS in newborn period. Presence of IgM antibodies in a neonate can be detected by day 5 of life in postnatally acquired illness(2).

DHF/DSS is uncommon in children below 1 year who are usually exposed to infection by dengue virus for the first time. However, if the mother is previously infected by dengue virus and hence has already developed antibody against that virus the infant may have placentally transmitted antibodies and may develop DHF after the first infection by

dengue virus of antigenically different type(6). So presence of shock and hemorrhagic manifestation seen during neonatal period can be attributed to passively transferred circulating antibodies from the mother. This can be explained on the basis of immune enhancement theory also. According to this theory DHF/DSS occurs as a result of enhanced replication of virus in presence of preexisting antibody against another dengue serotype(6). Most cases have been noted to occur when dengue type 2 infects either (1) a baby with maternal antibody against dengue or (2) a child with serological evidence of having been infected during the previous 5 years with a heterogenous dengue serotypes(6-8).

Some authors have suggested that viral virulence is a risk factor for DHF/DSS independent of pre-infection antibody status(8). Therefore, cases of DHF/DSS in earlier age group could also be due to increased virulence of virus during the present epidemic.

In Delhi epidemic the cause of low incidence of DHF/DSS in infants may be because the epidemic was controlled in a very short time and thus secondary infection rate was low, but 2001 epidemic in Rajasthan lasted for about 4 months and thus mother infected during earlier period of epidemic gave birth to babies who were subsequently infected in later period of epidemic and developed DHF/DSS. One must think for DHF/DSS in a neonate presenting with short duration pyrexia, bleeding disorder and shock.

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