

Dengue Hemorrhagic Fever in Calcutta

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Calcutta has a long experience of recurrent epidemics of Dengue fever(1,2). Serological surveys carried out during 1960 by Sarkar *et al.*(3) and a re-survey in 1966(4) showed a high endemicity of Dengue in Calcutta. Calcutta experienced Dengue Hemorrhagic Fever (DHF) for the first time during 1963-65(5,6). During this epidemic, Dengue viruses 2 and 4 were isolated[^]). Dengue hemorrhagic fever is distinct from classical dengue fever (DF) and is characterized by an acute febrile illness followed by thrombocytopenia, hemorrhagic diathesis, dengue shock syndrome (DSS) and hemoconcentration. Another outbreak of DHF involving mostly children and young adults, due to Dengue virus type 3, was reported from different parts of Calcutta in 1983(2). The first epidemic of

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DHF/DSS has also been described from China recently(8).

In Calcutta a large number of children were affected with DHF/DSS and quite a number of them died in 1990 although no such case was reported earlier after 1963-65 outbreak. In view of the relative rarity and consequent inexperience of the medical community in diagnosing and managing this potentially fatal disease, particularly among children, the clinical presentations are being emphasized here so that pediatricians may be aware of the possibility of this disease beforehand.

Material and Methods

Seventy-two children from the Central, North-Eastern and Southern parts of Calcutta suspected to be suffering from DHF or DSS admitted to the Department of Pediatrics, Medical College hospitals, Calcutta from September to December, 1990 were included in the study.

A case was considered to have a dengue hemorrhagic fever (DHF), in presence of fever, hemorrhagic manifestations including a positive tourniquet test (except in case with shock), thrombocytopenia ($100,000/\text{mm}^3$ or less), hemoconcentration with hematocrit increased by 20% or more over the baseline value or objective evidence of increased capillary permeability. A diagnosis of dengue shock syndrome was considered in a patient of DHF who also showed hypotension or narrow pulse pressure (less than 20 mm Hg)(9). Serological studies on paired sera for dengue virus were performed using hemagglutination inhibition, complement fixation tests and virus isolation.

Results

Of 72 children, 24 were between 1 and 3 years old, 27 between 3 and 5 years and 21 between 5 and 7 years. No child aged below 1 year was admitted.

The clinical features included fever ranging from 38°C to 40.5°C for 1 to 7 days duration and association with headache, bodyache and malaise in 71 cases. Fever for 3-4 days was followed by bleeding manifestations including hematemesis and melena in 30 cases, purpura in 7 cases (9.7%); epistaxis, hematuria and hemoptysis were not observed. Thirty five (48.6%) children developed sudden circulatory failure manifested by cold extremities. Shock as evident by cold extremities, hypotension or narrow pulse pressure was observed in 30 (41.7%) patients. Hepatomegaly was present in 56 (77.8%) and splenomegaly in 6 (8.3%) patients. Ascites was seen in 27 (37.5%) and pleural effusion in 9 (12.5%) cases. Seventeen patients died.

The cause of death included bleeding in 8 (11.1%) patients, shock in 6 and bleeding and shock in 3 (4.2%) patients. Of 35 patients with bleeding, 24 survived and 11 died. Of 30 patients with shock, 21 survived and 9 died.

Serological tests for virus were performed in Virology Department, School of Tropical Medicine, Calcutta in 71 cases. One patient died before the blood sample could be collected. Thirty one (43%) cases were serologically positive for Dengue virus antibodies. Sera inhibiting 4 HA units of Dengue antigen in a dilution of 1/20 or more in HI tests or 1/4 or more dilution in CF test is considered positive serologically. The details of isolation, identification of dengue isolates as well as serological tests including their interpretations have been

followed as mentioned by Mukherjee *et al.*(2). With the paired samples, Dengue infection was considered to have been established when the convalescent sample showed either conversion or a four-fold or greater increase in HI and CF antibody titres against DEN2 (Dengue 2) antigen and with a significant difference from JE (Japanese encephalitis), WN (West Nile viruses) or both. Without such a difference it was considered as a flavivirus group reaction(2). From 2 cases, Dengue virus type 3 could be isolated. From the acute phase sera, 2 viral agents could be isolated in suckling mice which were identified as Dengue type 3.

Discussion

Fever, gastrointestinal and hemorrhagic manifestations were frequent manifestations. In the more severe cases of the disease, abdominal pain, hepatic enlargement and ascites were usually associated. Usually, on the fourth or fifth day of fever, shock frequently occurred suddenly associated with vomiting in more than half the cases. In certain cases, it was preceded by abdominal pain and petechiae. There was no alteration of consciousness in contradistinction from cases of viral encephalitis. Some cases of DSS survived when rapid and appropriate treatment was given. The severity of the disease and the frequency of gastrointestinal severe bleeding manifestations in those who died and those with severe form of the disease but who survived, were similar. Thrombocytopenia was present in 100% cases.

The modal age of hospitalized children with DHF/DSS was 4 years. There was no sex predilection. No child below one year was hospitalized. Some workers favor the "Immune enhancement" hypothesis, which

states that DHF/DSS occurs as a result of enhanced replication of virus in the presence of pre-existing antibody against another Dengue serotype. Most cases have been noted to occur when dengue type 2 infects either (i) a baby with maternal antibody against Dengue, or (ii) a child with serological evidence of having been infected during the previous 5 years with a heterologous dengue serotype(1,3,4,10).

So DHF/DSS was uncommon in children below one year who were exposed to infection by Dengue virus for the first time. If the mother is previously infected by Dengue virus and hence has already developed antibody against that virus the infant may inherit that antibody and may develop DHF after the first infection by Dengue virus of antigenically different type. No such case was, however, admitted to our hospital at that time.

The clinical picture manifested by children did not differ greatly from that described earlier for outbreak in the South-East Asia. This was also in agreement with observations of Kouri *et al.* (11). Encephalitic features as reported by Mukherjee *et al.*(2) were not experienced in any case.

Lymphadenopathy, generalized skin rash and arthralgia with or without swelling of the joint as reported by Sarkar *et al.*(7) were rare in our series. Tourniquet test which was one of the diagnostic criteria of DHF as suggested by the WHO was negative in most of our cases possibly due to associated features of shock.

REFERENCES

1. Verchere AM. Report on the epidemic of Dengue of 1872 as it appeared in Fort Williams, Calcutta. Indian Med Gaz 1879,14: 91-95.
2. Mukherjee KK, Chakravarti SK, Dey PN, Dey S, Chakraborty MS. Outbreak of febrile illness due to Dengue virus type 3 in Calcutta during 1983. Trans R Soc MedHyg 1987, 81:1008-1010.
3. Sarkar JK, Chatterjee SN. Survey of antibodies against arthropod-borne viruses in the human sera collected from Calcutta and other areas of West Bengal. Indian J Med Res 1962, 50: 833-841.
4. Chatterjee SN, Chakravarti SK, Chakraborty MS, Ray S, Pal SR, Sarkar JK. Re-survey of arbovirus antibody in the human sera after an interval of six years. Indian J Med Res 1969, 57: 1629-1635.
5. Sarkar JK, Chatterjee SN, Chakravarty SK. Hemorrhagic fever in Calcutta. Some epidemiological observations. Indian J Med Res 1964a, 52: 651.
6. Sarkar JK, Chatterjee SN, Chakravarti SK. Virological and serological studies of cases of haemorrhagic fever in Calcutta. Indian J Med Ref 1964b, 52: 684.
7. Sarkar JK, Chatterjee SN, Chakravarty SK. Three year study of mosquito borne hemorrhagic fever of Calcutta. Trans R Soc Trop Med Hyg 1967, 81: 725-735.
8. Fuxi Q, Qiugian C, Qiya H, *et al.* The first epidemic of dengue hemorrhagic fever in China. Am J Trop Hyg 1991, 44: 364-370.
9. World Health Organization Dengue hemorrhagic Fever: Diagnosis, Treatment and Control. Geneva, 1986, p 13.
10. White DO, Fenner FJ. Medical Virology, 3rd edn. Florida, Academic Press, 1986, p487.
11. Kouri GP, Guzman MG, Bravo JR, Triana Jr C. Dengue hemorrhagic fever/dengue shock syndrome: Lesson from the Cuban epidemic. Bull WHO 1989, 67: 375-380.