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Abstract

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Outbreak of Dengue Fever in Delhi
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Abstract

An epidemic of illness with fever, severe aches, and lethargy lasting 5-10 days was recorded during September and October, 1988, in Delhi.

Eighteen affected adults presenting with upper gastrointestinal haemorrhage admitted to our gastroenterology ward. these patients were referred with upper gastrointestinal bleeding ascribed to ingestion of aspirin and non- steroidal anti-inflammatory drugs given for their pain and fever. The mean age of the eighteen patients (fifteen men, three women) was 35 year. On admission all of them had fever for 1-5 days and upper gastrointestinal bleeding was confirmed by aspiration. Four patients had feature suggestive of hypovolaemic shock and encephalopathy was present in eight. Petechiae distrusted all over the body were noted in eight patients. Except for a low haemoglobin (3.1-10.0 g/dl) no other haematological abnormality was found. Total leucocytes counts varied from 5400 to 11600 pl with a normal differential. Prothrombin times were prolonged by more than 3s in all patients except two. All had a rise in transaminases, but serum bilirubin and alkaline phosphatase levels were normal except in 1 patient whose bilirubin was 4.2 mg/dl. Endoscopy (not done in the eight patient with encephalopathy or the two in whom the bleeding stopped) consistently revealed multiple superficial erosions in oesophagus stomach and duodenum. No patient bled from the kidney or other site.

The triad of febrile illness with severe aching associated with upper gastrointestinal haemorrhage and encephalopathy during an epidemic of fever raised the suspicion of viral haemorrhagic fever, possibly due to dengue virus.

The patients were given supportive treatment (transfusion, H_2 receptor antagonises intravenous fluids), Seven of the eight patients with encephalopathy died; The other ten patients recovered. None of the patients with encephalopathy had nay abnormalities in serum electrolytes feature of cerebral oedema or focal neurological deficit. None was hypertensive. It was suspected that the encephalopathy might have been caused by punctuate cerebral haemorrhage but CSF was not examined.

Immediately after the first few admission of such patients, we asked for a proper surveillance study of the outbreak, with the serological assistance of the National Institute of Virology, Pune. Many sera were collected from victims of the epidemic in hospitals in Delhi and the National Institute of Virology confirmed our suspicion of Dengue virus infection. Seven of the first thirteen patients in our series in whom serological tests for viral haemorrhagic fever were available had high titres of complement fixing (CFI) and haemagglutination inhibiting (HI) antibody against all four strains of dengue virus indicating an anammestic response. In four of these seropositive patients IgM antibody (capture ELISA) against dengue virus type 2 was also present indicating that the epidemic was most probably Dengue-2. In three patients Dengue-virus like particles were isolated. In the remaining three patients tests for Dengue- Virus were inconclusive.

Mosquito-borne flavivirus-induced haemorrhagic fever in Children has been reported predominantly from the tropics. India is considered to be in the endemic zone for Dengue and Chickungunya virus. However, Dengue Haemorrhagic fever has been reported only once before from India during an epidemic in Calcutta. The Delhi epidemic of Dengue virus infection had some unusual features, noted in our series and by other physicians in the city. Haemorrhage from various organs has been reported in Dengue haemorrhage fever but upper gastrointestinal haemorrhage was the most important and life-threatening mode of presentation in this epidemic and was the most common reason for hospital admission. Encephalopathy almost always meant a fatal outcome. Diffuse punctate cortical haemorrhage may have been responsible for this fatal manifestation. Earlier reports suggested that Dengue haemorrhage fever is a disease of children and that adults are rarely affected. However, during the Delhi epidemic most of those affected were adults. Antibody-dependent enhancement in Dengue fever is the mechanism implicated in the haemorrhage manifestations. Low titre antibody from one Dengue virus infection enhances the disease process during a subsequent infection with another Dengue serotype. When this happens IgG antibody to group-specific antigen rises. During the first episode only IgM antibody specific to the infection serotype appears in the sera. The presence of high-titre IgG in seven of thirteen patients in our series indicates that most of our patients had had at least one Dengue virus infection previously and were thus susceptible to the haemorrhage disease during the current epidemic. During the epidemic 30% of the population of Delhi were affected and those contracting the disease for the first time are now at high risk of haemorrhage disease with or without encephalopathy during any subsequent epidemic.