after 30 days of infection when antigen is no longer detected. The presence of viral antigen and a fourfold rise in antibody titre in seven children indicated a current infection. However, six children testing positive for virus antigen did not have detectable virus specific antibody probably due to maternal antibody masking the IgG response in the infants(8).

It is concluded that RSV is an important etiological agent causing bronchiolitis and bronchopneumonia. Exposure to RSV was virtually universal since almost all children above one year had antibodies against RSV. Early viral diagnosis by rapid tests like enzyme immunoassay could help in reducing the unnecessary use of antibiotics.

REFERENCES

1. Mishra PK, Chaudhary RS, Jain A, Pande A, Mathur A, Chaturvedi UC. Viral etiology of acute respiratory infections in children in North India. J Trop Pediatr 1990,36: 24-27.
2. Aggarwal SC, Bardoloi JNS, Mehta S. Respiratory syncytial virus infection in infancy and childhood in a community in Chandigarh. Indian J Med Res 1991, 59:19-25.
3. Chatterjee R, Suttaone B, Chattopadhya D, *et al.* Techiques in the etiological diagnosis of lower respiratory tract infections in Children. J Commun Dis 1988, 20: 226-231.
4. Masters HB, Bate BJ, Wren C, Lauer BA. Detection of respiratory syncytial virus antigen in nasopharyngeal secre tion by Abbot diagnostic enzyme immunoassay. J Clin Microbiol 1988, 26: 1103-1105.
5. Gardner PS. Respiratory syncytial virus infections. Postgrad Med J 1973, 49: 788-791.
6. Haq F, Rahman M, Nagar M, *et al.* Acute lower respiratory tract infection due to virus among hospitalized children in Dhaka, Bangladesh. Rev Infect Dis 1990,8(Suppl): 5982-5991.
7. Parrot RH, Lim HW, Brandt CD, Chanock RM. Respiratory syncytial virus in infants and children. Prev Med 1974,3: 473-480.
8 Meddens MJM, Herbrink P, Lidman J, Van Dijk WC. Serodiagnosis of respiratory syncytial virus infection in children as measured by respiratory syncytial virus specific immunoglobulin G, M, A with enzyme-linked immuno-sorbent assay. J Clin Microbiol 1990, 28:152-155.

Cerebritis in Typhoid Fever

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Among, the various complications of typhoid fever, shock and encephalopathy are commonly seen. Cerebritis as a complication of typhoid fever has not been reported in children to the best of

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our knowledge. A child with culture proven typhoid fever who had cerebritis in addition to cardiovascular collapse is being described.

Case Report

An 8-year-old boy presented with continuous fever for 15 days. There was no associated cough, bleeding from any site, alteration of sensorium, seizures, bladder and bowel complaints. On examination, the child was toxic, and had peripheral circulatory failure with a BP of 60 mm Hg systolic and a pulse rate of 120/minute. The regular, low volume heart sounds were not muffled. There was no clinical evidence of heart failure or pneumonia. Spleen was palpable 2 cm below costal margin and liver edge was just palpable. Central nervous system examination was within normal limits. A provisional diagnosis of enteric fever with shock (circulatory collapse) was made. The investigations revealed a hemoglobin of 9 g/dl, TLC 3100 cells/mm^3 (DLC P78 and L22). Peripheral smear revealed toxic granules in leukcocytes. Widal test was positive with a TO 1:800 and TH 1:800. Blood culture grew *Salmonella typhi.* The Child was administered injection Ciprofloxacin and dopamine infusion was started to combat shock. He showed steady improvement with the above medication and BP stabilized by the 5th day. However, on the 8th day of hospitalization, the child developed recurrent seizures characterized by orofacial twitchings which persisted for 72 hours and were controlled with dilantin and luminal. There was no alteration of sensorium, cranial nerve palsies, focal neurological defects or meningeal signs and CSF examination was normal. Bolus intravenous contrast enhanced CT of head showed grossly normal posterior fossa structures (Cerebellum and brain stem); the basal and sylvian cisterns, and 4th, 3rd and lateral ventricles were normal. Ventricular level and supra ventricular cuts showed almost symmetrical frontal and high parietal white matter hypodensities with associated minimal gyral enhancement with no significant mass effect which were suggestive of bilateral frontal and high (convexity) parietal cerebritis *(Fig. 1)*. With the evolution of cerebritis on ciprofloxacin therapy, ceftriaxone was started to achieve a better CNS penetration. The child improved on ceftriaxone therapy and was discharged two weeks later. Repeat CT scan revealed normal brain parencyma and the child is clinically well after one year of follow up.

Discussion

The cerebral and meningial complications of typhoid fever have been studied in detail in children in the last few years. Well known neuropsychiatric manifestations of typhoid fever include

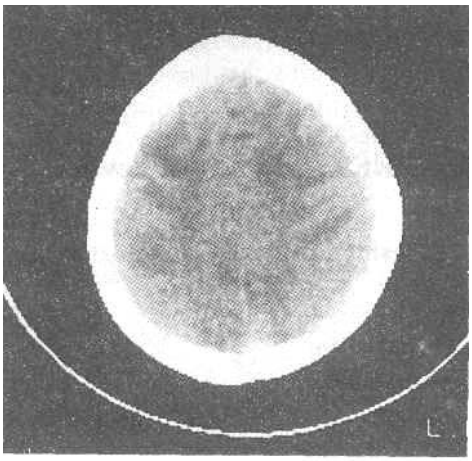

Fig. 1. CT scan showing cerebritis.

toxic confusional states (57%), meningismus (5%), meningitis (0.2%), focal neurological defects such as hemiplegia (0.5%), convulsions (1.7%), optic neuritis, perceptive deafness (0.5%) and peripheral neuropathy (0.7%), optic neuritis, perceptive deafness (0.5%) and peripheral neuropathy (0.7%)(l). The incidence of these complications have been reported to vary depending on age and drug resistance(2). Cerebritis as a complication has not been reported in literature. In the above described patient with serologically and bacteriologically proven typhoid fever, CT scan showed evidence of cerebritis. Cerebritis usually manifests initially in white matter with vascular congestion and edema so that ill defined areas of low density and mass effect in the white matter are noted on enhanced CT scans(3). With the progress of the disease process, however, contrast CT scan may show patchy enhancement mostly in the region of grey matter reflecting breakdown of blood brain barrier(4). Encephalitis denotes a diffuse inflammation of brain and CT scans in cases of encephalitis do not reveal localized areas of enhancement as seen in this patient(3). The presence of bilateral edema in the temporoparietal regions favor cerebritis and not post ictal edema which manifests unilaterally.

The exact -pathogenesis of CNS involvement in typhoid has not been clearly elucidated. The typhoid endotoxin is believed to have an affinity for the basal cranial structures to produce a picture described as typhoid encephalitis characterized clinically by cortical and bulbar manifestations(5). Post mortem histology in fatal cases has revealed congestion, diffuse edema and perivenous lymphocytic infiltrations(5).

REFERENCES

1. Osuntokum BO, Bademosi O, Ogunremi K, Wright SG. Neuropsychiatric manifestations of typhoid fever in 959 patients. Arch Neurol 1972, 27: 7-13.
2. Ramanan A, Pandit N, Yeshwanth M. Unusual complications in a multidrug resistant Salmonella typhi outbreak. Indian Pediatr 1992,29:118-120.
3. Haaga JR. Infectious processes of the brain. *In:* Computed Tomography of the Whole Body, 2nd edn. Missouri, CV Mosby Company, 1988, pp 120-123.
4. Enzmann DR, Britt RH, Yeager AS. Experimental brain abscess evolution: Computed tomographic and neuropathologic correlation. Radiology 1979,133:113-122.
5. Seth CK, Puri RK. Neurological complications of typhoid fever. Indian J Child Health 1963,12: 335-341.