Recovery After Fulminant Coxsackie B5 Meningoencephalitis in a 10-Year-Old Child

Coxsackie B5 infections are frequently associated with upper and lower respiratory tract infections, aseptic meningitis and carditis. Neurological complications are uncommon, except in neonates and infants.1 We report a child with Coxsackie B5 infection associated with fulminant meningoencephalitis, severe neurological manifestations and eventual complete recovery. A previously healthy 10-year-old girl presented to the Hospital with a 7-day history of headache and low grade fever. She was admitted following an episode of vomiting and a generalized convulsion lasting 20 minutes. Convulsions recurred during the day of admission and were both generalized and focal. The patient was confused and had definite nuchal rigidity. Blood pressure was 130/80 mmHg, and temperature was 38°C. General and neurological examinations were otherwise normal. White blood cell count on admission was 2.1 × 10^9/litre (56% neutrophils, 44% lymphocytes), and the ESR was 25 mm (after 1 h). Liver function tests, serum electrolytes, immunoglobulins and complement, T cell and neutrophil function tests were all normal. A brain CT scan was normal. CSF examination showed white blood cells less than 5/mm, no red blood cells, protein 0.15 g/litre, glucose 2.7 mmol/litre and negative bacterial culture. Electroencephalogram showed generalized slow wave activity suggestive of encephalitis.

The patient was treated supportively and with acyclovir for 10 days because of the possibility of Herpes encephalitis. On the third day of admission, her condition deteriorated. She remained in deep coma for 2 weeks during which repeat CSF examination was normal. She continued to have frequent focal seizures, and a repeat EEG showed generalized seizure activity with a focal predominance over the right frontal lobe. She gradually improved, and during the next 4 weeks regained full consciousness. After 8 weeks neurological examination was normal, and she was discharged on carbamazapine. During the following year she had one prolonged convulsion after discontinuing her medication. She continued on carbamazapine for a further 2 years.

Virological studies confirmed Coxsackie B5 infection as the cause of the meningoencephalitis. In serum a fourfold rise in complement fixing (CF) antibody was detected to enterovirus group antigen. Further analysis of the rise in antibody titre to enterovirus group antigen revealed a fourfold rise in neutralizing antibody titre to Coxsackie B5 and a twofold rise to B2, but there was a significant rise in IgM (ELISA) against B5 only in CSF and serum. Virus culture of several specimens was negative. CNS infection is one of the most important complications associated with enterovirus infections. Severe encephalitis is very rare, although fatal encephalitis has been reported in neonatal and childhood Coxsackie B infection.2,4 The present case of severe fulminant meningo-encephalitis due to Coxsackie B5 was associated with a favourable outcome. We do not believe that this was related to the use of acyclovir because acyclovir is effective in DNA viruses only.5

References

C-reactive Protein: The Test of Choice to Monitor the Inflammatory Response to Tissue Damage

Sir

The inflammatory response to tissue injury includes a change in the plasma concentration of several proteins that originate in the liver which are known as acute phase proteins. Quantitative measurement of acute phase proteins is a valuable indicator of the presence, extent and response of inflammation to treatment.1 Non-specific tests such as the ESR and plasma viscosity are sensitive to the cumulative effect of several plasma proteins. After tissue injury there is an increase in concentration of different acute phase proteins such as C-reactive protein (CRP), serum amyloid A protein (SAA) and α1-antichymotrypsin (α1-AT). Immunoassays for individual acute phase proteins are now available. C-reactive protein is striking