

## **Are These Cases of Heat Stroke?**

In Gorakhpur and adjoining areas, Japanese Encephalitis (JE) occurs round the year both in epidemic as well as endemic forms. The peak of JE epidemic occurs at the end of rainy season (September to November). This year, quite unexpectedly, several children with acute encephalopathic illness were admitted to the B.R.D. Medical College, Gorakhpur during May and June. Certain clinical features of these patients were distinctly different from those of patients with JE, which are being highlighted here.

Thirty patients (20 M and 10 F; age distributed in *Table I*) presented with short history of high fever, altered sensorium and generalized convulsions. Preceding gastrointestinal symptoms as diarrhea (10 patients), colicky abdominal pain (4 patients), abdominal distension (2 patients) and gastrointestinal hemorrhage (4 patients) were reported in 20 cases (66.5%). Examination revealed hyperpyrexia with varying degree of coma in all the patients. The core temperature ranged from 39.5-41.5°C. None of the patient had meningeal signs, involuntary movements or decerebrate posturing. Focal neurologic deficit such as hemiparesis was noted in one patient only.

Nine patients had low hemoglobin with polymorphonuclear leucocytosis in 16 blood biochemistry including liver enzymes was normal in all with negative smears for malaria parasites. The cerebrospinal fluid (CSF) findings are detailed in *Table I*.

The CSF and sera at the peak of illness were sent to the National Institute of Virology, Pune, where hemagglutination, complement fixation, MAC Elisa tests against Japanese Encephalitis, West Nile and Dengue antigens were reported as negative. The therapy in all patients included lowering temperature by aggressive cooling and control of infection, seizures and brain edema. Eleven patients (36.7%) recovered fully, while 7 (23.3%) succumbed. Twelve patients (40%) had prolonged convalescence, of which 3 had behavioral changes on follow up. Unfortunately, none of the parents agreed for autopsy of the deceased.

The timing of the illness and the age group involved are strikingly in contrast to JE(1). Preceding gastro-intestinal symptoms, hyperpyrexia and absence of meningeal irritation, involuntary movements, focal seizures and decerebrate posturing were other remarkable features. Only one patient had hemiparesis while CSF pleocytosis was absent in 24 (80%). Virological studies proved their non flavi viral encephalopathy. Other cases of acute encephalopathy like Reye syndrome and cerebral malaria were excluded.

**TABLE I**—Age and CSF Findings Compared with JE Cases(1).

Age	Present series % (No.)	JE Cases %	C.S.F.	Present series % (No.)	JE Cases %
3 mo - 3 yr	50.0 (15)	02.9	Proteins <40	83.3 (25)	70.6
4 - 6 yr	23.4 (7)	28.9	(mg/dl) >40	16.7 (5)	29.4
7 - 10 yr	16.7 (5)	49.2	Cells >50	6.7 (2)	17.1
11 - 15	10.0 (3)	19.0	( $\times 10^6/l$ ) 5 - 50	13.3 (4)	56.5
			<5	80.0 (24)	26.4

Nevertheless, the outcome of the illness was same as in JE(1).

The year 1995 had an unusually intense summer and heat induced encephalopathy may be a possibility in these patients. Heat hyperpyrexia may occur in children during high ambient temperature, particularly if they are not acclimatized and have water deficiency. Hyperpyrexia (core temperature beyond 40 °C) and deranged sensorium varying from delirium to deep coma may be associated with convulsions. Features of disseminated intravascular coagulation, hepatic, renal and cardiac damage may complicate heat stroke. Young children and neonates may develop neurologic symptoms at relatively lower temperature (39.4°C)(2). Preceding history of mild to moderate vomiting and diarrhea may follow sudden development of the illness, closely simulating 'acute encephalopathy of childhood' (3). Examination of cerebrospinal fluid is

unremarkable unless meningeal infection is associated. However, whether our patients had heat hyperpyrexia or some other acute encephalopathic illness can not be ascertained with certainty.

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