

Correspondence:

Dr. Dinçer Yildızdas,

Çukurova University Faculty of Medicine,

Pediatric Intensive Care Unit,

01330, Adana/Turkey.

E-mail: rdy90@hotmail.com

REFERENCES

1. Azuri J, Lerman-Sagie T, Mizrahi A, Bujanou Y. Guillain-Barre syndrome following serological evidence of hepatitis A in a child. *Eur J Pediatr* 1999; 158: 341-342.
2. Glaze DG. Guillain-Barré Syndrome. *In: Feigin RD, Cherry JD, editors. Textbook of*

Pediatric Infectious Diseases 4th ed. Philadelphia: WB Saunders; 1998. p 470-482.

3. Guillain-Barre Syndrome. *In: Behrman RE, Kliegman RM, Jenson HB, editors. Nelson Textbook of Pediatrics. 16th ed. Philadelphia: WB Saunders; 2000. p 1892-1893.*
4. Tabor E. Guillain-Barré Syndrome and other neurologic syndromes in hepatitis A, B and non-A, non-B. *J Med Virol* 1987; 21: 207-216.
5. Breuer GS, Morali G, Finkelstein Y, Halevy J. A pregnant woman with Hepatitis A and Guillain-Barré Syndrome. *J Clin Gastroenterol* 2001; 32: 179-180.

Outbreak of Killer Brain Disease in Children

With reference to the viewpoint(1), we wish to share our experience of Acute Encephalopathy Syndrome in Bangalore(2) and Reye's Syndrome in Bangalore(3).

An ICMR study was conducted during the period of October 1986 to December 1986 on 269 cases of Acute Encephalopathy admitted to Vani Vilas Children Hospital. Out of these 124 were diagnosed as having Reye's Syndrome (RS). This report was the largest series from a single centre/City in India.

One hundred and twenty four cases of Reye's syndrome admitted to Vani Vilas Children Hospital, bangalore were investigated. Clinical, biochemical and epidemiological details were obtained. The median age was five years, with no difference in sex ratio. This disease was frequent in winter months. Cases clustered in certain

congested localities of the city among lower socio economic strata. Aspirin and varicella could not be associated as preceding factors. The clinical and biochemical features of the patients were suggestive of Reye's Syndrome. Histopatho-logical evaluation was done in 104 liver biopsy specimens and 102 brain specimens (post mortem). Virological studies for influenza and arbovirus were negative. Mortality was high (78%). During this period CT scan was not available and hence brain CT was not done in any of these cases.

We share this experience of the largest published series, so that the astute pediatrician keeps these conditions in mind under mysterious outbreaks of Killer Brain Diseases. Off late for reasons not known, incidence of RS has decreased, though sporadic cases are reported. It is surprising to note that the expert team did not carry out the investigations for RS. It is very unfortunate that so many children died without a proper workup.

I entirely agree with Dr. Jacob John's

view. Periodic sensitization of Pediatricians is necessary to be aware of the rare, but not uncommon disease of RS especially during epidemics. We personally feel IAP should constitute an expert group to investigate such outbreaks.

**D.G. Benakappa,
Asha Benakappa,**
*601, First Cross, 30th Main,
Banashankari III Stage,
Bangalore 560 085, India.*

REFERENCES

1. John TJ. Outbreak of killer brain disease in children. Mystery or missed diagnosis? *Indian Pediatr* 2003; 40: 863-869.
2. Benakappa DG, Prasad SR, Shastry MSC and S Geogre. Acute Encephalopathy Syndrome in Bangalore. *Indian J Pediatr* 1983; 50: 121-127.
3. DG Benakappa, Das S, Shankar SK, Rama Rao BSS, George PS, Aswath PS, *et al.* Reye's syndrome in Bangalore. *Indian J Pediatr* 1991; 58: 805-810.

Reply

The letter from Drs. Benakappa and Benakappa is interesting and important. They had admitted 269 cases of acute encephalopathy during 3 months in 1986, of which at least 124 were diagnosed as Reye's syndrome. This illustrates the high incidence of Reye's syndrome in one region at one period of time. Would it be wrong to call it an outbreak? For a spurt in incidence there must have been some

common factor, such as a preceding infection or exposure to a toxin. Unfortunately, research to investigate such causative factors are seldom undertaken partly because our institutions are by and large not well equipped for such research and partly because the very diagnosis is often contested and the opportunity for such targeted research is often lost. If Drs Benakappa and Benakappa could do liver biopsies on 104 children with acute encephalopathy, others who see large numbers of cases should be encouraged and emboldened to do liver biopsies on at least a small proportion of cases to confirm or exclude Reye's syndrome by examining the tissue for microvesicular fat in hepatocytes. The spurt in incidence is unpredictable and often nonrecurring in the same place. If IAP can tie up with the Indian Council of Medical Research, a research protocol can be developed and it may then be applied anywhere when the incidence of Reye's syndrome is found to be of outbreak proportions. Research is also needed to investigate the cases of encephalopathy not diagnosed as Reye's syndrome. It will be worthwhile for IAP to develop consensus on diagnostic criteria and treatment modalities for acute encephalopathy syndromes including Reye's syndrome. Early detection of cerebral edema and its rapid treatment can drastically reduce case fatality.

T. Jacob John,
*439, Civil Supplies Godown Lane,
Kamalakshipuram,
Vellore, TN, 632 002
E - Mail: vlr_tjjohn@sancharnet.in*