REFERENCES

1. Bar-Maor JA, Eitan A. Determination of the normal position of the anus (with reference to the idiopathic constipation). J Pediatr Gastroenterol Nutr 1987; 6: 559-561.
2. Reisner SH, Nitzan M. Determination of anterior displacement of the anus in the newborn infants and children. Pediatrics 1984; 73: 216-217.
3. Leape LL, Ramenofsky ML. Anterior ectopic anus: A common cause of constipation in children. J Pediatr Surg1978; 13: 627-630.
4. Genc A, Taneli C, Tansug N, Kasirga E, Yilmaz D, Kucukoglu T, *et al*. Evaluation of the location of the anus by a modified technique in the neonate. J Pediatr Surg 2002; 37: 80-82.
5. Upadhyaya P. Mid-anal sphincteric malformation, cause of constipation in anterior perineal anus. J Pediatr Surg 1984; 19: 183-186.

A Child with Guillain-Barré Syndrome Caused by Acute Hepatitis A Infection

To our knowledge, 20 patients with Guillain-Barré Syndrome (GBS) secondary to acute hepatitis A virus (HAV) infection have been reported in the literature. Only one of them was a child(1). We report another such case in a 6 year old boy who presented with pain and weakness in legs and difficulty in walking. His past medical history was unremarkable except for a jaundice two weeks back. Vital signs and anthropometry was in normal limits. Neurological examination revealed reduced power (2/5) and hypotonia in all extremities. Deep tendon reflexes (DTR) were absent. Other systemic examination was normal. Transaminases (SGOT: 1000 IU/dL, SGPT: 900 IU/dL) and bilirubine levels (total/direct bilirubin: 12/10 mg/dL) were elevated. Anti-HAV Ig M was positive and anti-HAV Ig G was negative in blood and cerebrospinal fluid (CSF); markers for hepatitis B virus (HBV) were negative. Cerebrospinal fluid revealed a protein concentration of 82 mg/dL, glucose of 41 mg/dL and no cells. Electromyograpy and nerve conduction velocity revealed a severe motor polyneuropathy associated with axonal damage in muscles and nerves in all extremities.

The patient was treated with 0.5 g/kg intravenous immunoglobulin (IVIG) for five days. He was discharged from the ward on the tenth day of admission. Physical examination at the end of the first month showed a 4/5 of the muscle power and positive DTR. Laboratory studies revealed normal liver function tests. The serum anti-HAV Ig M and anti-HAV Ig G were positive.

Guillain-Barré Syndrome occurring in the course of acute hepatitis caused by hepatotrophic viruses like cytomegalovirus (CMV), Epstein-Barr Virus (EBV), HBV and HAV have been reported previously(1-4). In patients who develop GBS after an acute viral hepatitis, demonstration of the specific viral antibodies in the CSF may confirm the central nervous system involvement(5).

Kocabas E,
Yildizdas D,
Çukurova University Faculty of Medicine,
Department of Pediatrics,
Adana/TURKEY

Correspondence:
Dr. Dinçer Yildizdas,
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, Bujanouer 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 Hopital, 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 econnomic 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