after a very brief illness. Those who survived would recover completely without any neurological deficit. So far, I have treated 229 (2003 – 46; 2004 – 74; 2005 – 78; 2006 – 14; 2007-17) children and out of these 212 died giving a case fatality of around 93%.

To my surprise, >50% of the cases presenting this year gave a positive history of eating the beans of Cassia occidentalis, before falling sick. It is a highly prevalent weed called “Pnawad” in local language. Even the friends of the cases who had consumed the weed along with the patient but in smaller quantity also developed mild illness with raised SGPT but without full-blown encephalopathy.

As the public awareness about the poisonous effect of the weed is scant and even pediatricians are not aware of this possibility, there is an urgent need to create mass awareness about the weed and to take measures to reduce its density so that future outbreaks can be prevented.

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Cassia Poisoning Behind Mysterious Disease in Children in Uttarakhand

I would like to compliment ‘Indian Pediatrics’ for publishing and providing a solution to the recurrent annual outbreaks of a mysterious fatal disease affecting young children of western UP and Uttarakhand for last many years(1). This illness often labeled as ‘acute viral encephalitis’by the investigating agencies and ‘brain fever’ by lay media has been puzzling pediatricians and public health experts for over a decade.

I am practicing in Roorkee, adjacent to the district Saharanpur, the epicenter of these outbreaks for last 15 years. For last 6-7 years, I am getting cases of acute onset of encephalopathy during September to early December months, characterized by sudden onset of vomiting, mild fever, abnormal movements and at times seizures and rapid progression of unconsciousness. All these cases were hailing from rural areas and were 2-5 years old with a slight female preponderance.

These cases used to have hypoglycemia (30%), raised SGPT (100%), and prolonged PT I.N.R. more than 1.2 (95%) but with normal serum bilirubin levels. CPK was also found to be raised. The CSF examination was normal in all these cases without any pleocytosis. On the basis of the short clinical presentation and biochemical findings, we used to diagnose and treat them as cases of Reye syndrome. The cases had very bad prognosis and majority (>90%) would die within 48 hours of presentation after a very brief illness. Those who survived would recover completely without any neurological deficit. So far, I have treated 229 (2003 – 46; 2004 – 74; 2005 – 78; 2006 – 14; 2007-17) children and out of these 212 died giving a case fatality of around 93%.

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