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TOXIC DISORDERS

ACUTE ISONIAZID NEUROTOXICITY

An increased incidence of acute isoniazid (INH) neurotoxicity correlating with a resurgence of tuberculosis (TB) in New York City is reported from the Children's Medical Center of Brooklyn and the Department of Emergency Medicine, State University of New York, Health Science Center at Brooklyn. Nine patients receiving INH prophylaxis for TB between 1991 and 1994 developed refractory seizures, metabolic acidosis, vomiting, and/or coma after accidental or suicidal ingestion of toxic doses of INH (14 - 99 mg/kg). Eight patients were adolescents and one was a 5-day-old infant. Symptoms began within 45 - 150 min (aver, 90 min). IV pyridoxine controlled seizures. (Shah BR et al. Acute isoniazid neurotoxicity in an urban hospital. <u>Pediatrics</u> May 1995;95:700-704). (Reprints: Binita R Shah MD, Box 49, Department of Pediatrics, Children's Medical Center of Brooklyn, State University of New York, Health Science Center at Brooklyn, 450 Clarkson Ave, Brooklyn, NY 11203).

COMMENT. In children receiving prophylactic treatment for tuberculosis who present with an acute onset of seizures refractory to anticonvulsants, isoniazid toxicity should be suspected and pyridoxine administered intravenously. Pyridoxine reverses the depletion of GABA caused by INH and restores the balance of inhibitory and excitatory neurotransmitters in the brain. If the amount of INH ingested is known, the dose of pyridoxine is limited to a gram-for-gram replacement, and is given in 15 to 30 minutes. Multiple excessive doses of pyridoxine may result in sensory loss and should be avoided. INH inhibits phenytoin metabolism and may lead to phenytoin toxicity. Diazepam can be used to supplement the specific anticonvulsant effect of the pyridoxine in INH induced seizures that are severe or prolonged.

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