lower symptom-to-IST interval.

- The number of children with VSAA is much lower as compared to earlier studies from India (12% vs. 27-45%) [2-4]. The data from western countries is conflicting on response of VSAA to IST. Chandra, et al.[2], and Sharma, et al.[3], have reported lower response rates in VSAA as compared to SAA (33% vs. 54.5% and 25% vs. 68.7%), respectively. Similarly, children with higher neutrophil count were found to have superior response by Gupta, et al. [4].

- The median symptom-to-IST interval in the study was 2.5 months. This interval is nearly the same or less than diagnosis-to-IST interval in earlier Indian studies, except the one from Varanasi [2,4-5]. A quicker referral and early administration of IST in armed forces hospitals, as compared to ‘civilian institutions’ is possibly another reason for the superior outcome. Recent studies have documented that a shorter diagnosis-to-IST interval predicts better response by preventing irreversible damage to hematopoietic progenitor cells from auto-reactivated T cells.

The relapse rate observed (3%) is significantly less as compared to several Indian/Western pediatric series (10-33%). Although relapses can occur several years following IST, the median time to relapse in majority of the reports is 18-30 months. A prolonged duration and slow tapering of cyclosporine has been reported to be associated with a lower relapse rate. Although, the authors have mentioned the cyclosporine schedule in the treatment protocol, the median duration of administration of cyclosporine and cyclosporine dependence has not been cited. This information may help to explain the lower relapse rate.

It would be interesting to learn if such good results are replicated from other centers in India in the future.

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Eyelid Myoclonia with Absence Seizure: Precipitated by Carbamazepine Therapy

Jeavons syndrome (eyelid myoclonia with absence seizure) is a rare type of idiopathic generalized epilepsy [1]. We report a young girl who presented with this disorder after introduction of carbamazepine.

A nine year old girl, presented with two episodes of unprovoked seizures during sleep characterized by deviation of eyes and head to right and tonic clonic movement of all four limbs during sleep for two weeks before presentation. Antenatal and birth histories were uneventful. Maternal uncle had history of generalized seizures. Patient was a developmentally normal child. Physical examination was noncontributory. MRI brain showed no abnormality. Her first EEG showed generalized discharges. She was already on carbamazepine (CBZ) started by some private physician. CBZ was continued. Over next two months her academic performance deteriorated and she felt giddy on looking at television or sun. After three months, she presented with continuous eye blinking for three days. EEG showed absence status. She was diagnosed as having eyelid myoclonia with absence seizure. Status was controlled by intravenous benzodiazepines and sodium valproate. CBZ was stopped. Seizures stopped within 48 hours. She was discharged on sodium valproate and clonazepam. Only two brief episodes of eyelid myoclonia occurred in first
month of starting valparin and clonazepam. The child is asymptomatic for last 15 months.

Jeavons syndrome is a generalized epileptic condition clinically characterized by eyelid myoclonia with or without absences, eye closure-induced EEG paroxysms, and photosensitivity; in addition, rare tonic-clonic seizures may also occur [1]. It is more common in females between 2-14 years.

CBZ though a very useful drug can unmask or aggravate various types of seizures. Idiopathic generalized epilepsies e.g. absences, tonic, atonic, tonic-clonic, myoclonic etc. are known to be aggravated by CBZ [2,3]. It can even mask or reduce the beneficial effect of valparin or phenobarbitone [3]. With the use of CBZ in patients with GTCS, absences and myoclonic jerks can appear de novo, and generalized paroxysmal discharges can appear in various focal epileptic syndromes [4]. Menon, et al. [5] have described a similar case in an adult patient, although eye blinking in that case was not continuous.

Camphor Poisoning

Camphor is a commonly seen household item which can cause severe poisoning even when taken in small amounts in children. Neurotoxicity in the form of seizures can occur soon after ingestion. Camphor is used in many vaporized or topical cold medications, topical musculo-skeletal anesthetic preparations, moth repellants and in antimicrobial preparations. We report a child who presented with seizures due to ingestion of camphor used in religious ceremony.

A 3-year-old male child presented with history of consumption of camphor followed by two episodes of vomiting. He had accidentally consumed around two camphor cubes confusing it for sugar cubes. The family was using camphor for performing rituals in religious ceremonies. At admission, the child developed convulsions, with tonic posturing of limbs and uprolling of eyeballs. Interval between the onset of seizures and ingestion was around 30 minutes. Injection lorazepam was given to abort seizures. There were no other neurological deficits. Vital parameters were stable. Rests of the systems were normal. The child received stomach wash, ranitidine and intravenous fluids. He recovered completely within 24 hours and discharged after 2 days.

The easy availability of camphor in various forms put children at high risk of camphor poisoning. It is potentially fatal, even when taken in small doses in children [1]. It is remarkable for its rapidity of action. Camphor containing products can not exceed 11% of camphor as set by FDA, but in our country the concentration of camphor is not mentioned on the products.

Within 5-15 minutes patients commonly complain of mucous membrane irritation, nausea, vomiting and abdominal pain. Generalized convulsions are often the first sign of significant toxicity and can occur soon after ingestion [2]. Camphor induced seizures can occur after gastrointestinal, dermal or inhalation exposures [3]. One case of status epilepticus is reported following abdominal massage with camphor containing product [4]. Other CNS symptoms include headache, dizziness, confusion, agitation, anxiety, hallucinations, myoclonus, hyperreflexia and ataxia. CNS stimulation is followed by depression. Camphor can also cause hepatic and renal damage. Death is usually the result of respiratory failure or convulsions. Neurotoxicity is seen if ingestion of camphor is more than 50 mg/kg [5].

Treatment is primarily supportive with a focus on airway management and seizure control. Skin and ocular decontamination should be done by flushing with copious amounts of water. Patients with camphor inhalation