Case Reports

Phenytoin Induced Dyskinesia

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Drug induced movement disorders are commonly produced by neuroleptics and antiparkinsonian drugs but may be observed occasionally due to anticonvulsants too(l). There are occasional case reports of phenytoin, phenobarbitone, carbamazepine, primidone and ethosuximide producing dyskinesias in Engligh literature(2-5). We report an uncommon case of phenytoin-induced chorea and ballismus.

Case Report

An 11-year-old boy (boy weight 29 kg) was admitted in December 1996 with the chief complaints of involuntary movements of face, tongue and limbs of two days duration. The child was diagnosed to have tubercular meningitis three months back and he had undergone ventriculoperitoneal (VP) shunt drainage operation for hydrocephalous at that time. He was on four drugs antitubercular regimen (Isoniazid, Rifampicin, Pyrazinamide and Streptomycin) since then alongwith oral prednisolone (dose 1 mg/kg per day). He was also taking 150 mg of phenytoin (dose 5 mg/kg per day) for generalized tonic clonic seizures which he had 3 months ago. On enquiry parents revealed mild swaying while walking and slurring of speech. There was no history of fresh seizure, altered sensorium or visual symptoms. General examination revealed a thin built boy with pulse 76/min regular, BP 108/70 mm Hg, no pallor or significant lymphadenopathy. Central nervous system examination showed normal mentation, mild dysarthria, bilateral gaze evoked horizontal nystagmus, no papilledema, involuntary movements involving face, tongue and upper extremities, depressed deep tendon reflexes and flexor plantar response. The child had repeated twitching and grimacing movements of the face that changed constantly in character and location. He was unable to hold out his tongue for any length of time and when asked to protrude it he shot it out and then jerked it back. There was abnormal vocalization and difficulty in maintaining phonation. There were bilateral abrupt involuntary rapid and forceful swinging or flinging movements of upper limbs at irregular intervals. These used to disappear during sleep. The boy had bilateral mild finger-nose ataxia and was unable to perform tandem walking. Rest of the systemic examination was normal. With fresh clinical findings of chorea, bilateral ballismus and bilateral cerebellar signs, phenytoin toxicity was suspected. An increase in hydrocephalus due to nonfunctioning of VP shunt or infarct due to tubercular vasculitis in basal ganglia and/or brainstem or cerebellum were the other possibilities considered for the develop-

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Manuscript Received: July 28,1997;
Initial review completed: September 3, 2997;
Revision Accepted: September 30,1997

ment of fresh symptoms. Routine hematological and biochemical profile were normal. Magnetic resonance imaging (MRI) of brain did not show any evidence of increased intracranial pressure or infarct in brain, basal ganglia, brainstem or cerebellum. The plasma phenytoin level was reported to be in toxic range of 32 µg/ml (normal 10-20 µg/ml). The antitubercular treatment was continued and the dose of phenytoin was reduced as per the nomogram. The involuntary movements disappeared within three days. Repeat plasma phenytoin level estimation was done after 1 week which was found to be 14.2 µg/ml and patient was discharged from the hospital.

Discussion

Phenytoin is one of the most commonly used anticonvulsants in India due to easy affordability and availability though it is now regarded as a second line drug in most countries. This drug has been reported to induce orofacial chorea, ballismus and dystonia(4). Even acute athetosis and encephalopathy have been reported in a child due to phenytoin toxicity(3,5). These involuntary movements have occurred in some patients with high plasma phenytoin levels as was found in the present case where the plasma phenytoin levels was in toxic range. The other predisposing factor for development of phenytoin-induced dyskinesia may be a pre-existing lesion in the basal ganglia. The hemiballismus seen during second stage of tuberculous meningitis is usually associated with hemiplegia on opposite side due to destruction of subthalamic and subputaminial area by basal exudates and vasculities. In our case there was no limb weakness and normal MRI scan of brain ruled out any structural lesion in the basal ganglia(6). In the present case the boy was on anti-tubercular treatment since three months making good recovery. Moreover the toxic level of phenytoin in blood at the time of presentation and disappearance of involuntary movements within 3 days of reducing the dose of phenytoin virtually rules out the possibility of these abnormal movements as part of disease process of tubercular meningitis. In animal experiments phenytoin has been shown to possess dopamine receptor blocking properties which can lead to dyskinesias(l). Irrespective of the mechanism of phenytoin induced dyskinesia, the drug interaction between phenytoin and antitubercular drug might have played a key role in producing toxic range of plasma phenytoin level in the present case. Isoniazid (the antitubercular drug) is known to interact and raise the plasma phenytoin level(7). Hence periodic plasma anticonvulsant drug monitoring is recommended in patients receiving concurrent anticonvulsant and antituberculous therapy to avoid such adverse complications.

Acknowledgement

We wish to thank Dr. (Mrs) P.M. Pai, Dean, Seth G.S. Medical College and K.E.M. Hospital, for allowing us to publish this case report.

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