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Opsoclonus Myoclonus due to Phenytoin Toxicity

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ABSTRACT

Phenytoin is one of the oldest and commonest antiepileptic drugs used in our country. It is known for its toxicity as neurological and cardiac manifestations. Opsoclonus myoclonus syndrome due to phenytoin toxicity is a rare incidence. We report a case of phenytoin toxicity in 18 year female patient who was taking phenytoin for symptomatic generalized tonic clonic seizures 1month back who now presented with opsoclonus, myoclonus and truncal ataxia.

Keywords: Phenytoin toxicity, opsoclonus, myoclonus

INTRODUCTION

Phenytoin has been used widely for generalized tonic clonic and focal seizures as it's cheap and easily available. Phenytoin is also notorious for many toxic effects due to its narrow therapeutic index. Toxicity can manifest as nausea, vomiting, nystagmus, ataxia, coma, seizures, hypotension, ventricular arrhythmias. Opsoclonus myoclonus syndrome (OMS) is a rare condition with incidence at 0.18 cases/ 1,000,000 person years.[1] It is characterised by a) opsoclonus which is involuntary, rapid, conjugate saccadic eye movements b)myoclonus which affects the trunk, limbs and c) cerebellar ataxia. Most frequent causes in adults is paraneoplastic, infections while toxic causes are uncommon.[2]

An 18 year old female patient was admitted in the emergency department with complaints of headache, fever and one episode of generalized tonic clonic seizure. Patient was evaluated with imaging first and then cerebrospinal fluid (CSF) examination. The CSF was consistent with acute bacterial meningoencephalitis and was managed with antibiotics and for symptomatic seizures she was started on phenytoin. Patient was discharged after 10 days of treatment and planned to continue phenytoin for next 3 months. After 1 month patient was readmitted in emergency with complaints of multiple episodes of vomiting. Her vitals were normal, there was gingival hypertrophy, neurological examination revealed exaggerated deep tendon reflexes while rest of the examination was normal. On second day of admission patient developed rapid, irregular eye movements, jerky movements of arms and truncal ataxia. On further examination there was slurring of speech, incoordination. In view of neurological abnormalities possibility of phenytoin toxicity was kept, drug was stopped and serum phenytoin levels were sent which came out to be high (> 40 ug/ml). After the withdrawal of drug patient shows gradual improvements of symptoms which resolved after 5 days and patient was discharged with advice of outpatient department follow up.

DISCUSSION

Phenytoin is absorbed and concentration peaks at 1.5 to 3 hours for immediate release and 4 to 12 hours for extended release formulation. Phenytoin has narrow therapeutic index between 10 -20 mg/dl. Phenytoin is a voltage gated sodium

channel blocker, prolonging the refractory period both in neuronal and cardiac tissues.[3] Phenytoin toxicity depends upon the duration of exposure, dosage and route of administration. The neurological toxicity is dose dependent while cardiac toxicity happens with parenteral administration. The neurotoxic effect varies from nystagmus, slurred speech, ataxia, vomiting and coma. Chronic toxicity by phenytoin is generally dose related as studies have shown that gradual accumulation of phenytoin over months to years leads to toxicity as a result of non-linear pharmacokinetics. But in our patient the exposure with drug was only for one month so there can be unpredictable relation between the serum concentration and its toxic effects. The pharmacokinetics may be not only related to drug dose but may be patient related too.

Opsoclonus myoclonus syndrome is also called as dancing eyes, dancing feet syndrome. It's a rare condition where there is degeneration of cerebellar neurons. Most common cause is the paraneoplastic syndrome due to cancer remote to central nervous system. OMS induced by toxic etiology is extremely rare. The pathophysiology for toxic etiology is unclear, it is thought to originate from dysfunction of omnipause neurons of the nucleus raphe interpositus [4] or disinhibition of fastigial nucleus in the cerebellum. [5] OMS has been seen with drugs like amitriptyline, cocaine, lithium, cefepime, cyclosporine and venlafaxine.

CONCLUSION

Due to the toxic effects of phenytoin it's important that the patients are followed up on regular basis and the serum levels can be monitored if there is any sign of adverse effects. The patients should also be made aware of the adverse effects so that they can identify them on time

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