



Role of Hemoperfusion in Phenytoin Poisoning

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Abstract

Suicidal ingestion of phenytoin can lead to severe neurological sequelae. Charcoal Hemoperfusion decreases phenytoin level. We present a lady with suicidal ingestion of phenytoin who responded to Charcoal Hemoperfusion.

Introduction

Phenytoin is commonly prescribed antiepileptic drug. However it has narrow therapeutic range and total serum level more than 20 ug/ml is associated with clinically relevant toxicity¹. Death has been reported at levels of 50-70ug/ml. Treatment recommendation beyond supportive care is unclear and effectiveness of extracorporeal elimination (charcoal hemoperfusion) is still under debate². From clinical point of view urgent lowering of phenytoin concentration may reduce the risk of dysrhythmia and neurological complication and shortens length of stay in hospital may therefore favorable³. We present a case of suicidal ingestion of phenytoin that was treated successfully with one session of charcoal Hemoperfusion resulting in both clinical improvement and normalization of serum phenytoin concentration.

Case History

A 24 years old female brought by relatives to emergency with complaints of sudden onset altered sensorium, involuntary head nodding and irrelevant talk for 24 hrs duration. There was no history of

fever, trauma. Relatives gives history suggestive of tablet consumption around 60 tablets. As Patient was known case of seizure disorder on Tab. Phenytoin. So the dose taken by her was approximately 6 gm.

On examination she was in altered sensorium with truncal ataxia suggestive of cerebellar toxicity. CT scan of brain was normal. She was started on supportive care. In view of cerebellar toxicity and nystagmus diagnosis of phenytoin poisoning suspected. Serum phenytoin level was found to be 76 ug/ml which was in toxic range. Patient's sensorium was deteriorated. Relatives counseled about hemoperfusion and charcoal hemoperfusion was done. The only complication noted is thrombocytopenia (platelet count 28,000). Two bags of platelet were transfused during session. With one session patient recovered neurologically. Her altered sensorium improved on 2nd day with serum phenytoin level 33.56 ug/ml. By the 8th day patients ataxia and nystgmus disappers with serum phenytoin level 5.6 ug/ml.

Discussion

Phenytoin follows zero order kinetics which are responsible for observed dose related toxicity⁴. The plasma half life of phenytoin has been shown to be dose dependent in human. In other words the time required for the plasma level to halve itself increases as concentration of drug increases. An explanation for the non exponential decline of phenytoin is that the biotransformation mechanism approaches saturation at high plasma level⁵. And in absence of displacing agent is bound tightly to plasma protein (87%-97%)⁶. The half life of phenytoin decreases as level decreases⁷. The role of charcoal Hemoperfusion can be explain by the fact that bound phenytoin has been found to dissociate from albumin in the presence of activated charcoal and subsequently becomes adsorbed to the activated charcoal⁸. We suggest that Hemoperfusion technique must be considered in patient with prolonged toxic concentration and neurological manifestation.

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