## Phenytoin induced status epilepticus

*To the Editor*

I read the interesting case report by Al-Khulaif and Shujaa[1] on the phenytoin induced status epilepticus. The possibility that antiepileptic drugs might aggravate epilepsy must always be borne in mind by the clinician. Seizure aggravation might include increase in the frequency or severity of existing seizures, emergence of new types of seizure, or the occurrence of status epilepticus. The pathophysiology of seizure aggravation is poorly understood including non-specific effects such as those associated with sedation, drug-induced encephalopathy, and paradoxical or inverse pharmacodynamic effects. Risk factors for worsening of seizures were found to be epileptic encephalopathy, polytherapy, high frequency of seizures, and cognitive impairment.[2] I have 3 comments on the aforementioned study.

First, Al-Khulaif and Shujaa[1] stated that the status epilepticus in the studied patient is attributed to phenytoin toxicity. They explained that from a biochemical point of view in terms of saturation of the hepatic hydroxylation system. However, such an explanation seems questionable considering that the studied patient was taking 300 mg phenytoin per day, which is still within the therapeutic range of 200-500 mg per day. I presume that phenytoin poisoning, whether intentional or unintentional, must not be overlooked in the studied patient though absent history of witnessing the event does not definitely exclude it. Epileptics are more prone to have poisoning and/or suicidal attempt because of increased emotional instability and poor impulse control.[3]

Second, apart from the phenytoin toxicity induced seizure as stated by Al-Khulaif and Shujaa,[1] the poor compliance of the studied patient to antiepileptic drugs could additionally contribute to development of status epilepticus in a way nearly similar to an abrupt withdrawal of antiepileptic drugs.[4,5]

Third, considering that the serum phenytoin concentration in the studied patient was mildly-moderately elevated (30.3 mg/l), I presume that Al-Khulaif and Shujaa[1] totally depend upon native body clearance to relevantly lower phenytoin concentration, which was fortunately effective. However, in high serum phenytoin concentrations, 3 sessions of 4 hour long combinations of activated charcoal hemoperfusion and high flux hemodialysis could result in considerably reduced half-life during these measures of around 7-13 hours compared to the native half-life wavering between 40-100 hours.[6] Hemodiaperfusion with activated charcoal seems to be an effective tool for forced lowering of highly toxic phenytoin plasma concentration and should be strongly considered in critical circumstances like poisoning or inadequate iatrogenic dosing.

***Mahmood D. Al-Mendalawi***
*Department of Pediatrics*
*Al-Kindy College of Medicine, Baghdad University*
*Baghdad, Iraq*

### *Reply from the Author*

No reply was received from the author.

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