Sir,

A 32-year-old woman was brought to the emergency room ~28 h after ingesting an unknown amount of carbamazepine (probably 150–200 tablets containing 200 mg each). She had been taking carbamazepine 400 mg twice daily for the past 10 years for symptomatic epilepsy related to cerebral arteriovenous malformation. She was unconscious but responding to noxious stimuli. She had shallow irregular respiration at 18 breaths/min. Her blood pressure was 130/86 mmHg and heart rate was 82 beats/min. Her pupils were 4 mm in size bilaterally and sluggishly reacting to light. There was no discernible focal neurological defect. Motor examination revealed generalized hypotonia and depressed deep tendon reflexes. Both plantars were flexor. Primary detoxification at admission was performed with gastric lavage and charcoal instillation. She needed intubation and mechanical ventilation because of respiratory insufficiency. Serum carbamazepine level at admission was 2.567 µg/ml (therapeutic level 4–10 µg/ml). She was managed conservatively but there was no improvement in her neurological status. A CT scan of the head ruled out any bleeding from the cerebral arterio-venous malformation. Repeat carbamazepine level performed 58 h after ingestion was 22.58 µg/ml. As there was no improvement in her sensorium and blood carbamazepine levels remained persistently high, haemodialysis was performed for 4 h. After 2 h of haemodialysis, her sensorium improved markedly, and the carbamazepine level at this time was 12.26 µg/ml. She was weaned off the ventilator and extubated within 40 h of her arrival in hospital. A psychiatry evaluation was performed after stabilization and an underlying depressive disorder was diagnosed. She was restarted on carbamazepine 400 mg twice daily. Fluoxetine 20 mg twice daily was also added and she was discharged 5 days after admission.

There is no antidote for carbamazepine overdose. Primary gut decontamination with activated charcoal is commonly used, as charcoal adsorption decreases carbamazepine absorption from gut, thereby reducing plasma half-life [1]. For pharmacodynamic and pharmacokinetic reasons, there are several difficulties in the management of carbamazepine overdose. Gastrointestinal absorption of carbamazepine is unpredictable and peak plasma concentrations may be seen from 2 to 18 h after a single dose. The plasma half-life ranges from 21 to 55 h in normal volunteers and 8 to 19 h in chronically treated epileptic patients [2]. Carbamazepine has high plasma protein binding (70–80%); hence alkaline diuresis, haemodialysis and peritoneal dialysis are thought to have limited effects in removing carbamazepine from plasma [3]. A few reports in the 1980s showed efficacy of haemoperfusion in reducing the plasma levels of carbamazepine by 25–50% in intoxicated patients [1]. Since then, haemoperfusion has been considered to be the treatment of choice in severe carbamazepine overdose. Combined haemodialysis and haemoperfusion resulted in a 50% reduction in the blood levels of carbamazepine in one study [3]. More recently, plasma exchange was successfully used in treating carbamazepine overdose [4]. However, none of these methods is accepted as the gold standard for treatment of carbamazepine overdose. In our patient, haemodialysis was performed, as facilities for haemoperfusion were not available when this patient was admitted. Serum levels of carbamazepine fell by >50%, and the patient regained consciousness within 2 h of the start of haemodialysis. Lee et al. [5] had studied the effect of haemodialysis on removal of carbamazepine in four uraemic patients. They showed that the clearance of the drug by dialysis was twice the normal plasma clearance after oral administration of 500 mg carbamazepine. However, there are no published reports of carbamazepine overdose treated with haemodialysis alone. Haemodialysis is simple, cheap and more commonly and easily performed than haemoperfusion. It may be a good therapeutic option in removing carbamazepine from the circulation in patients with severe carbamazepine overdose.

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