Bilateral Asterixis Associated with Supratherapeutic Carbamazepine and Ammonia Levels

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INTRODUCTION

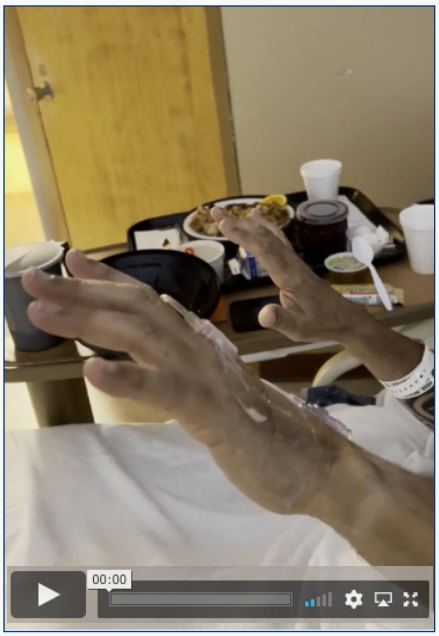
We present a case and a video demonstrating asterixis in a patient who developed toxicity on carbamazepine (**Video 1**), and resolution after withholding the medication (**Video 2**).

CASE PRESENTATION

A 65-year-old man with a 10-year history of complex partial epilepsy and hypertension, was admitted to the hospital for dysarthria, dizziness, and gait ataxia, which began gradually approximately 1 week prior to presentation. His seizures had been well controlled on carbamazepine 800 mg in the morning, 600 mg in the evening, and phenobarbital 64.8 mg twice daily. His examination revealed scanning dysarthria, slowed saccades, mild bilateral dysmetria in the arms, no nystagmus, and bilateral hand asterixis (**Video 1**). Given the subacute onset of his brainstem/cerebellar symptoms, stroke was initially suspected, and ruled out with an MRI brain, which showed few scattered white matter hyperintensities and no acute findings. His blood work revealed a normal comprehensive metabolic profile, including LFTs, complete blood count, and carbamazepine level of 26 (therapeutic range 4–12 UG/ML), phenobarbital level 23 (15–40 UG/ML), and ammonia 57 (range 2–50 UMOL/L). After two days of holding carbamazepine, the level fell within the therapeutic range and his asterixis resolved (**Video 2**).

Click to view video 1

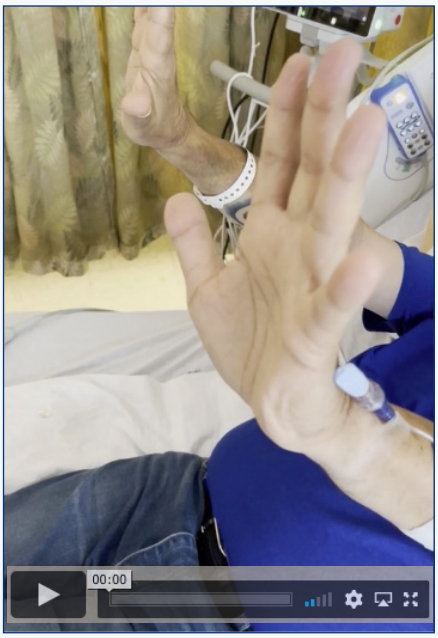
[0:14, https://vimeo.com/772119716]



Video 1. This video, taken at initial presentation, demonstrates asterixis (repeated, brief and rapid loss of sustained wrist extension (negative myoclonus) followed by immediate corrections). Note that the slow handflexions are not asterixis; only the fast flexions are asterixis.

Click to view video 2

[0:10, https://vimeo.com/772129122]



Video 2. This video, taken 2 days after holding carbamazepine, shows that the patient is able to maintain extension of his wrists, and the previously seen rapid flexion-movements are notably absent.

DISCUSSION

Asterixis is a "clinical sign that describes the inability to maintain sustained posture with subsequent brief, shock-like, involuntary movements"[9] caused by electrical silence for under 1/10 second in the tonically contracting muscle. Asterixis may be seen in any limb and may cause falls if the legs are involved. Bilateral asterixis points to a toxic or metabolic encephalopathy, which in this case is likely due to elevated ammonia and supratherapeutic carbamazepine levels. Most previous reports of patients taking carbamazepine in therapeutic doses and developing asterixis found elevated serum ammonia.[3,4,5,6] There are a few other reports of patients developing asterixis after having a therapeutic dose of carbamazepine added to their regimens in which carbamazepine levels in serum were elevated; however, serum

ammonia levels were not provided.[7] One case reported a patient developing hyperammonemia, agitation and aggressive behavior without developing asterixis.[8] The cause of the supratherapeutic CBZ level after 10 years of stable intake is unknown.

While an asymptomatic transient elevation of liver enzymes in patients taking carbamazepine is common[2], instances of carbamazepine-induced asterixis and hyperammonemia have been rarely reported.

References

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