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CASE REPORT

Mesial temporal sclerosis after status epilepticus due to milk alkali syndrome

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Summary Seizures induced by hypercalcemia are rare. A few case reports of seizures associated with hypercalcemia have been published, but none due to the milk alkali syndrome. This is the first report regarding seizures associated with calcium carbonate overuse. The two patients described in this article, who had no risk factors for developing epilepsy, suffered from status epilepticus probably induced by hypercalcemia. Subsequently, they both developed complex partial seizures, and were later found to have mesial temporal sclerosis on MRI. There are no reports linking hypercalcemia to mesial temporal sclerosis. While this may be a coincidence, there is reason to suspect that the development of persistent epilepsy, possibly due to mesial temporal sclerosis, was caused by prolonged seizures induced by hypercalcemia.

Introduction

Hypercalcemia is rarely a cause of seizure activity.[1] More commonly, it is associated with altered mental status and encephalopathy.[2] We report two cases in which the patient presented with status epilepticus without previous history of epilepsy. Status epilepticus was thought to be due to hypercalcemia. Evaluations for malignancy, hyperthyroidism and hyperparathyroidism were negative, and hypercalcemia was attributed to milk alkali syndrome. While the status epilepticus was controlled, the calcium level was promptly corrected and calcium supplements withheld, both patients developed epileptic seizure activity after the initial event. Both patients were later found to have mesial temporal sclerosis, diagnosed on MRI. We concluded that hypercalcemia due to milk alkali syndrome can induce status epilepticus, with subsequent persistent epilepsy. The relationship of hypercalcemia to the development of mesial temporal sclerosis remains unclear.

Case report 1

Patient 1 was a 54-year-old female with no significant past medical history, especially no history of previous

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seizures and epilepsy. She had a normal birth and normal early development. Specifically, there was no history of febrile seizures. She denied any other risk factors for epilepsy, including head trauma, infection and family history. There was, however, a history of alcohol abuse. She also admitted to take large doses of calcium carbonate for heartburn, prior to her presentation with status epilepticus.

The patient presented to a local emergency room after she was found unresponsive by her husband. While she was waiting for emergency services, a relative who is a registered nurse witnessed intermittent right-sided tonic clonic seizure activity with right-sided weakness. In the emergency room, urgent laboratory workup revealed a serum total calcium of 17.9 mg/dL (normal: 8.5—10.2 mg/dL) with acute renal failure. The patient was given phenytoin intravenously, and its level was brought into a therapeutic range. The total duration of seizure activity was uncertain, however, she definitely did not return to baseline after 30 min. While she was hospitalized, there was one single witnessed right-sided tonic clonic seizure with altered mental status lasting minutes, eventually requiring intubation. Due to recurrent seizures with no interictal recovery, the patient was diagnosed with status epilepticus.

During her 13-day hospitalization, her calcium levels returned spontaneously to normal, with complete resolution of her renal failure. Her calcium carbonate was withheld. The patient underwent an endocrinological evaluation which was unremarkable, with normal parathyroid hormone and vitamin D levels. A lumbar puncture performed to rule out infectious etiology was sterile, including normal cytology and a negative herpes virus PCR. The clinical evaluation and laboratory studies found no evidence of malignancy. A CT of the head demonstrated mild atrophy, attributed to previous alcohol abuse. An MRI was not obtained at the outside hospital. An EEG performed 12 days later demonstrated left hemispheric suppression, with intermittent paroxysmal discharges on the left. The patient had no further seizures at that time and was discharged on phenytoin.

Subsequently, over the next few months she had recurrent events of "phasing out", sometime with an aura of a scintillating scotoma in her right visual field. The events were associated with confusion, aphasia and occasionally right arm dystonia. She was evaluated at our epilepsy clinic, and was found to have a therapeutic level of phenytoin (12.4 mg/l) and a normal calcium level (9.9 mg/dL). She was having frequent complex partial seizures, with recurrent episodes of confusion and aphasia, some of which were witnessed in the clinic. An urgent EEG was quickly obtained, showing her to be in complex partial status epilepticus, with left temporal sharp theta bursts followed by slowing of the background activity. Phenytoin was increased and she was admitted for better control of her seizures. Upon reaching a higher therapeutic level of phenytoin (19.2 mg/l), the patient recovered and was back to baseline within 12 h. Later, lamotrigine was added for better seizure control, but she is still having about one complex partial seizure per month.

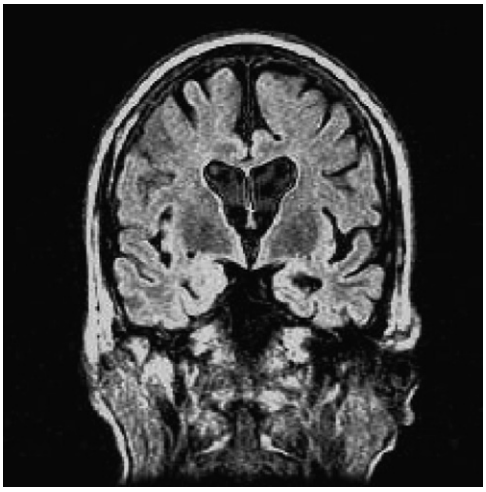

Figure 1 FLAIR brain MRI of patient 1. Volumetrics of the hippocampi. Right: 3.09, left: 3.16 mm^3.

MRI of the brain using an epilepsy protocol, performed 2 months after the initial presentation, demonstrated mild asymmetry with a prominent left temporal horn and left hippocampal atrophy (see Fig. 1). Neuropsychological assessment conducted 3 months following the first seizure demonstrated generally mild deficits in verbal encoding and retrieval, executive functions (cognitive flexibility, ability to maintain cognitive set) and visuospatial skills. These findings were noted in the context of low average to average intellect, and functioning within normal limits across other cognitive domains.

Case report 2

The second patient was a 41-year-old woman, with a history hypothyroidism and hypoparathyroidism, who had undergone a thyroidectomy for a benign mass. She was treated for years with calcium acetate and magnesium supplements. Otherwise, she had a normal birth, with reportedly normal developmental milestones, but a history of significant learning difficulties. There was no history of febrile seizures, head trauma or infection. There was no family history of epilepsy.

Without obvious precipitating factor, she was found on the floor having continuous generalized