

changes in the previously observed cerebral lesions⁵. The clinical neurologic significance of these findings as related to B12 deficiency has not yet been determined.

Other features of neurologic recovery

This patient demonstrated early neurologic improvement which continued to six months follow-up. Of note the patient demonstrated changes in his gait, general mood and behaviour. His vibration sense exhibited a marked improvement. On presentation he had reduced vibration sense to the level of his sternum but at six months follow-up he could sense vibration in his feet once again. Most notably however, the patient regained function for daily activity which included a marked improvement in his severely impaired gait observed on his initial presentation to the clinic.

CONCLUSION

Vitamin B12 deficiency continues to demonstrate a wide variety of neurologic symptomatology. We present here findings of a novel episodic dysosmia and document reversal of cervical cord MRI findings with B12 therapy. In patients with diffuse neurologic changes Vitamin B12 deficiency should be considered on the differential diagnosis, particularly in metabolically and nutritionally vulnerable populations. Despite the presence of macrocytic anemia in our patient we should caution against the dismissal of Vitamin B12 deficiency upon its absence. Normal Vitamin B12 level does not rule out its

deficiency as well. Furthermore Vitamin B12 and its metabolites should be investigated in any white matter disease.

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TO THE EDITOR

A Case of Amiodarone-Associated Myoclonus Responsive to Levetiracetam

Amiodarone, a class III antiarrhythmic, is infrequently associated with neurotoxicity. Reported adverse events include peripheral neuropathy, tremor, ataxia, parkinsonism and cognitive impairment.¹ We report a case of a patient with amiodarone-associated myoclonus.

A 90-year-old man with mild dementia presented with involuntary jerks and impaired gait. Past medical history was significant for chronic renal insufficiency, coronary artery disease, aortic valve replacement, and automated defibrillator placement. His chronic renal insufficiency was first diagnosed as a young adult, but his creatinine, although elevated, had remained stable for years without the need for hemodialysis. Two years prior to presentation, the patient was prescribed the antiarrhythmics digoxin and amiodarone. One year after initiating amiodarone, he developed fluctuating, often repetitive, action-induced jerks in his trunk and limbs. Repeated attempts to taper amiodarone from 200 to 100 milligrams (mg) daily resulted in recurrence of cardiac arrhythmias but provided significant improvement in the jerks, which quickly reemerged once the higher dose was reinstated. The Naranjo score² was 10, indicating amiodarone was the definite cause of the myoclonus.

On neurological examination, the Montreal Cognitive Assessment score was 19/30 (normal ≥ 26). Cranial nerve examination was normal except for decreased hearing bilaterally. Sensory examination was intact except for decreased vibratory sensation to the left knee. Coordination testing was normal. Biceps, triceps, and patellar reflexes were 3+ (hyperreflexic) bilaterally. Remaining reflexes were normal with flexor plantar responses. The movement examination showed a low-amplitude postural tremor and myoclonus in the upper extremities with posture and action. Myoclonus was not present at rest, was not induced by sound or touch, and was not associated with alteration of consciousness. The patient could not stand without using his arms to support himself and was unable to transfer to his walker without assistance. While walking, several episodes of negative myoclonus occurred requiring the patient to stop and grip the walker to prevent falling.

Electroencephalogram testing was normal. Magnetic resonance imaging of the brain was remarkable for a right cerebellar lacune and age-related white matter disease and volume loss. Serum tests including thyroid screen and liver function tests were normal. The creatinine level was 1.9 (0.66-1.25 normal range). A brief trial of clonazepam (0.5 mg) was not tolerated due to excessive drowsiness. Levetiracetam was then initiated. After one month of 125 mg twice daily, the patient's family noted improved ability to stand and transfer to the walker. He was titrated to 250 mg twice daily. Myoclonus was

significantly reduced; four months later he was able to stand up from a chair without using his hands and was able to ambulate with the walker without significant disruption. The increased dose was associated with fatigue, limiting further titration.

To our knowledge, this is the first case report of amiodarone-associated myoclonus with symptomatic improvement following initiation of levetiracetam. Amiodarone-associated myoclonus is a very rare phenomenon, which we believe was only described once before in a case series from 1992 that reported two patients who developed myoclonus after being exposed to amiodarone.³ It is worth noting that one of these patients also abused alcohol. Interestingly, a 12-year retrospective study of 707 patients exposed to amiodarone did not report the presence of myoclonus,¹ and a thorough review on the etiology, pathophysiology, phenomenology and treatment of myoclonus did not identify amiodarone as a cause of myoclonus.⁴ Our case thus reinforces the association between amiodarone and myoclonus, and provides anecdotal evidence of effective treatment. Our patient's exquisite response to levetiracetam is fortunate and likely spared him from the increasing risk of side effects that may be encountered when polytherapy is necessary for complete symptomatic control. However, caution is still

advised when using amiodarone and levetiracetam in combination, as there is an increased risk for the development of psychosis.⁵

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TO THE EDITOR

Lithium Induced Diabetes Insipidus, Trauma and the Shrunken Brain

A 53-year-old female with past history of bipolar disorder, treated with lithium carbonate, presented to hospital after being hit by an automobile travelling 60 km/hr. Upon arrival to emergency, her Glasgow Coma Scale was 13. She was verbalizing incoherent words. Initial trauma survey and computed tomogram (CT) chest/abdomen/pelvis were negative for acute injury. The CT-head demonstrated only a very small right lateral cortex subarachnoid hemorrhage. She was kept for observation. Her initial sodium and lithium levels were 141 mmol/L and 1.06 mmol/L, respectively. Initially, her urine output had not been accurately monitored. The next day she had worsening confusion and was admitted to the Neurosurgical Service. Repeat CT-head demonstrated significant bilateral subdural hypodense collections. Repeat serum sodium was reported as >180 mmol/L. Repeat serum creatinine was 142 mmol/L, significantly elevated from the initial at 88 mmol/L. Serum urea was also elevated, indicating pre-renal acute renal failure. Urine output was measured at 500 to 900 mL/hr while in the ICU.

A diagnosis of lithium induced diabetes insipidus was made and she was transferred to the Surgical ICU, where her serum sodium was carefully corrected over the next week. DDAVP was administered; however, there was no reduction in urine output or osmolality, indicating a nephrogenic source for the diabetes insipidus. With eventual normalization of all serum and urine output, her neurological status returned to her pre-accident baseline.



Figure 1: Initial Uninfused CT Head. Uninfused axial CT scan image at time of admission showing a small right lateral cortex hyperdensity, (arrow) consistent with suspected traumatic subarachnoid hemorrhage