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 a 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 isipidus. With eventual normalization of all serum and urine output, her neurological status returned to her pre-accident baseline.

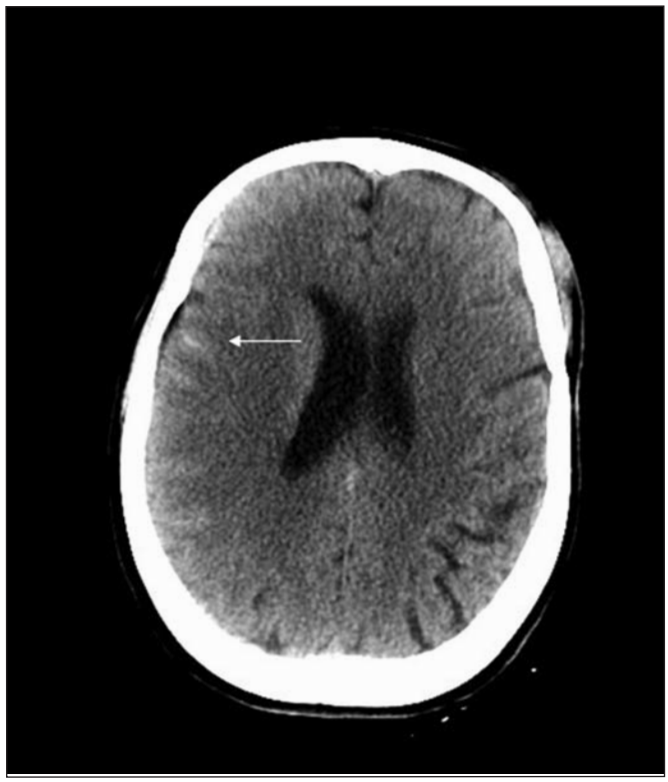

Figure 1: *Initial Uninfused CT Head. Uninfused axial CT scan image at time of admission showing a small right lateral cortex hyperdensity, (arrow) consistent with suspected traumatic subarachnoid hemorrhage*

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Magnetic resonance imaging (MRI) was conducted to assess for central pontine demyelination and was negative for any acute pathology. The patient was subsequently transferred back to Neurosurgery. She continues with hydorcholrothiazide, as per Endocrinology. Her urine output and serum sodium remain within normal limits. Consensus from Neurosurgery, Intensive Care, Nephrology, and Endocrinology was that she had lithium induced nephrogenic diabetes insipidus as the cause for the acute electrolyte, neurological, and imaging changes.

Figure 1 is an uninfused CT-head image acquired upon admission. On the right lateral cortical surface is a small hyperdensity indicating traumatic subarachnoid hemorrhage. Of note is the lack of subdural collections. Figure 2 demonstrates the repeat uninfused CT-head acquired 24 hours post-admission. This representative slice displays bilateral subdural hypodense collections with substantial decrease in cortical volume in comparison to Figure 2. Resolution of the right traumatic subarachnoid hemorrhage has occurred. The subdural collections are of CSF, density indicating they are extra-axial fluid collections in response to cortical volume changes.

Lithium carbonate is still a medication in common use for the treatment of mood disorders. Initial use in North America began in the 1970's for the treatment of various psychiatric disorders[1]. More recently, literature is emerging surrounding its use as a neuro-protective agent in acute brain injury[2]. However, one of the uncommon adverse effects of lithium is nephrogenic diabetes insipidus, which is always of concern and difficult to treat. Lithium has been shown to down-regulate aquaporin 2 water channel expression in the collecting duct of the nephron[3]. In addition, lithium induced naturesis leads to polyuria and increased fractional sodium excretion. This effect may be attributable to down-regulation of aldosterone[4].

Profound intravascular volume depletion with corresponding serum hypernatremia is common in untreated lithium induced diabetes insipidus. Such dramatic electrolyte changes can result in significant fluid shift. Our case demonstrates an interesting and novel view into the acute fluid changes intra-cranially with hypernatremia and the acute diuresis/naturesis associated with lithium induced diabetes insipidus, in the setting of normal serum lithium levels. Even literature in central diabetes insipidus is deficient in cases of acute subdural collection on neuroimaging[5].

This case outlines the importance and vigilance that one must maintain with monitoring electrolyte and fluid status in those patients on lithium chloride.

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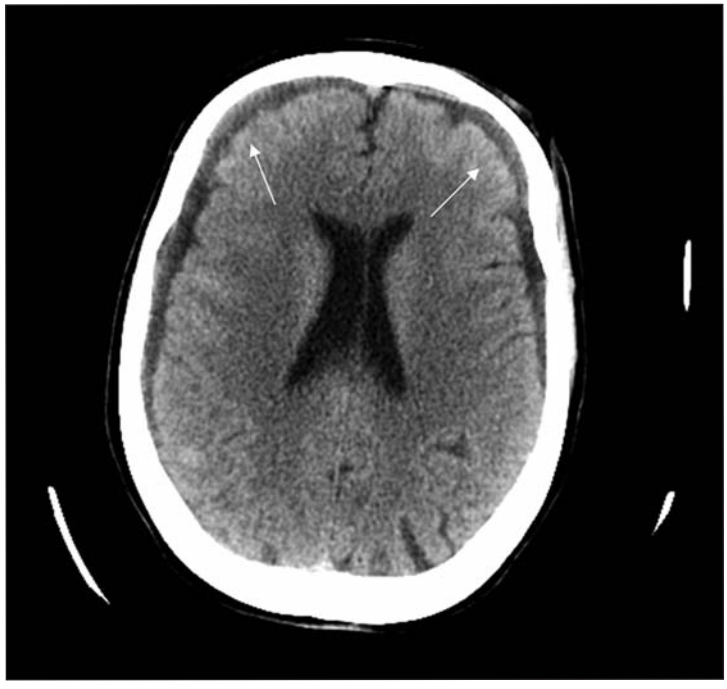

Figure 2: *24 Hours Post Admission uninfused CT Head. Axial image of uninfused CT-head 24 hours post injury. Image displays bilateral hypodense subdural collections with loss of cortical volume, consistent with extra-axial collections that developed in response to significant acute brain volume loss.*

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