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# Dementia following traumatic brain injury complicated by acute on chronic renal failure: A case report

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## Introduction:

Dementia is a neurological condition that is characterized by progressive loss of motor, cognitive, memory, and autonomic functioning.[1] MRI serves as a helpful diagnostic tool, as different neurodegenerative diseases present with distinctive findings on imaging. For example, Alzheimer's disease, the most common neurodegenerative disease, is characterized by brain atrophy starting in the entorhinal cortex and hippocampus.[2]

Currently, the greatest risk factor for dementia is age, with the majority of cases taking place in patients older than 65.[1] Traumatic brain injury (TBI) is a less common risk factor that can also predispose to dementia.[3,4] Currently, the leading cause of TBI is falls.[5] Classification of TBI as mild, moderate, or severe is based on the patient's Glasgow Coma Scale (GCS).[6] Traditionally, GCS of 14-15 is mild, 9-13 is moderate, and less than 9 is severe.[7] Many patients with mild TBI fully recover, but occasionally, patients may experience symptoms that persist following the TBI. Persistent symptoms are more likely if the patient has a prior history of TBI, but rarely, one instance creates permanent deficits.[6] These cases are more likely with elderly patients or if the patient has a pre-existing comorbidity, such as chronic renal disease.[6]

When kidney function is reduced, blood urea nitrogen (BUN) and creatinine are not cleared from the bloodstream as effectively. The accumulation of uremic toxins can lead to encephalopathy and can increase the likelihood of dementia.[8-10] Compared to chronic renal disease, acute kidney injury is more likely to lead to encephalopathy since the brain has less time to respond to accumulation of toxic metabolites.[9,10] Acute kidney injury induces an inflammatory response through activation of the innate immune system, which is thought to worsen accumulation of toxic metabolites through increased oxidative stress.[10] Initially, acute kidney injury presents as fatigue and loss of concentration, which can progress to seizures and coma if left unaddressed .[10] We present a patient with post traumatic dementia who later experienced acute on chronic renal failure.

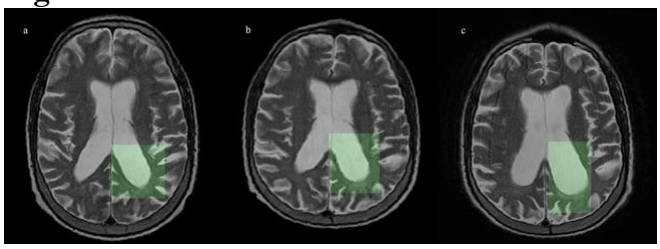
## Case description:

In December 2016, a 58 year-old male with a history of chronic renal disease and primary hypertension presented to the emergency room following mild TBI. Computerized tomography (CT) scan of the brain without contrast demonstrated small left parietal subdural hematoma, which was managed conservatively. After the TBI, the patient began to experience right quadrantanopia, right hemi neglect, and memory/cognitive impairment. He was asymptomatic prior to the TBI. These symptoms worsened throughout the following year. Magnetic resonance imaging (MRI) without contrast was performed in October 2017, which demonstrated mild hydrocephalus ex vacuo, especially in the left occipital horn (Figure 1a).

The patient began to experience decreased appetite and fatigue in July 2018. Basic metabolic panel was ordered (BMP) by his primary care physician, which revealed an elevated BUN of 78 and elevated creatinine of 7.45 compared to his baseline values of 40 and 2.5, respectively. The patient's bicarbonate was low at 18 with an elevated anion gap of 18. He was taken to the emergency room and admitted to the intensive care unit (ICU). On

admission, systolic blood pressure was elevated at 194. Per the patient's wife, since he began experiencing memory deficit, he was taking his anti-hypertensive medications inconsistently.

**Figure 1**: **MRI Brain**



a) Left, MRI in 2017, b) middle, MRI in 2018, c) right, MRI in 2021. Left occipital horn highlighted in green box

The patient was started on continuous intravenous (IV) amlodipine, hydralazine, and carvedilol plus normal IV saline. MRI without contrast was ordered, which demonstrated increased hydrocephalus ex vacuo compared to the MRI from 2017 (Figure 1b). After five days, the patient's anion gap and blood pressure stabilized, BUN remained elevated at 75, and creatinine remained elevated at 5.80. He was diagnosed with acute on chronic renal failure and was discharged from the ICU with plans for renal transplantation. The patient underwent hemodialysis until November 2018, when he received the kidney transplant. There were no post-operative complications, and creatinine decreased to 1.30.

Following the renal transplantation, the patient's appetite and energy level improved, but his memory, visuospatial, and cognitive impairment persisted. He has continued to follow up with his neurologist. Montreal Cognitive Assessment (MoCA) score from December 2018 was 13/30. Repeat MRI without contrast from August 2021 demonstrated more prominent hydrocephalus ex vacuo compared to the MRI from 2018, again primarily in the left occipital horn (Figure 1c). Based on the hydrocephalus ex vacuo being most noticeable in the left occipital horn, adjacent to the initial subdural hematoma, the patient's neurologist diagnosed the patient with dementia due to TBI. Although the patient was asymptomatic before the TBI, the neurologist also concluded that his prior chronic renal disease made him more susceptible to neurocognitive deficits following a single instance of brain trauma. Since 2021, the patient has developed a rolling tremor of the distal right upper extremity, bradykinesia, and shuffling gait. His pre-existing symptoms have worsened since onset in 2017, and both creatinine and blood pressure are stable at this time.

## Conclusion:

In conclusion, dementia is a neurological disease process with a variety of risk factors. While our patient did not demonstrate any symptoms of dementia prior to the TBI, his history of chronic renal disease likely made him more vulnerable to post traumatic neurocognitive decline. Among dementia patients, clinical presentation and age of onset are vast, but a common theme is that it is a slowly progressive condition with no definitive cure.

## Author Contributions:

Both authors contributed to the conception or design of the work and the acquisition, analysis, critical revision of the case report. Both authors approval the final version of the manuscript to be published.

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