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Letter to Editor

Internal Medicine Section

# Correspondence: Association of Serum Uric Acid Level with the Severity of Brain Injury and Patient's Outcome in Severe Traumatic Brain Injury

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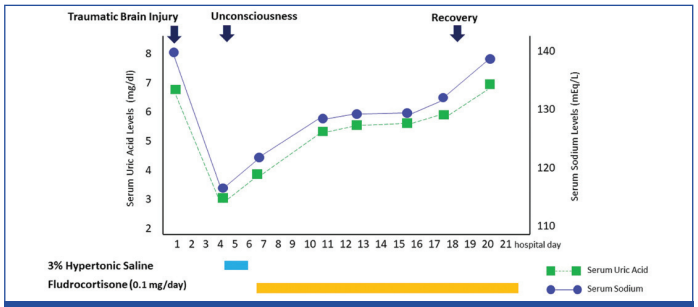
Dear Editor,

Uric Acid (UA) has validated its neuroprotective role in damaged brain. However, the relationship between serum UA levels and the severity of brain damage is controversial. I read with great interest the article "Association of Serum UA Level with the Severity of Brain Injury and Patient's Outcome in Severe Traumatic Brain Injury" by Hatefi M et al., [1]. They showed a strong relationship between UA levels and patients' outcomes in Traumatic Brain Injury (TBI). I recently experienced a coma patient with TBI who showed low UA and sodium levels, which may indicate an excess excretion or consumption of UA and sodium is a risk factor for the severity of TBI.

A 69-year-old male was admitted to the hospital because of head injury after a fall during clearing work. On arrival, his consciousness was clear. Radiological examination revealed traumatic subarachnoid haemorrhage and subdural haemorrhage. On the 4th hospital day, he became comatose, serum sodium and UA levels dropped to 115 mEq/L and 3.2 mg/dL, respectively. His plasma osmolality was 228 mOsm/kg $H_20$, whereas urine osmolality was 476 mOsm/kg $H_20$. The serum antidiuretic hormone concentration was detected as 1.5 pg/mL despite severe hyponatremia. His serum sodium level was increased to 122 mEq/L with 3% hypertonic saline drip at 20~30 mL/hour, resulted in slightly improving consciousness. His serum sodium and UA levels remained low even after adequate sodium infusion. Thus, fludrocortisone (0.1 mg/day) was stared considering the possibility of Central Sault Wasting Syndrome (CSWS). This resulted in the improvement in sodium and UA levels.

On the 21st hospital day, he recovered completely and was discharged with sodium and UA levels of 138 mEq/L and 6.8 mg/dL, respectively. The clinical course of the patient is illustrated in [Table/Fig-1].

TBI brings oxidative stress due to the structural and biochemical changes in the brain. UA is involved in almost 70% of the free radical scavenging reactions in the human. Thus, UA levels may influence the clinical course of TBI. Hatefi M et al., showed a significant positive linear relationship between increase in serum UA levels and improvement in the levels of consciousness in TBI patients [1]. In agreement with their report, the index patient showed low UA levels during unconsciousness and his condition improved as his UA levels were increased. Intriguingly, UA levels were related to sodium levels in the patient [Table/Fig-1].




**[Table/Fig-1]:** Clinical course; change in the patient's serum UA and sodium levels.

Brain damage interferes with normal neuroendocrine function and sympathetic nervous system of hypothalamus and pituitary, which could influence laboratory data such as serum sodium and UA levels [2]. CSWS brings an excess excretion of sodium and UA, although precise pathophysiology remains unclear. Since UA has numerous antioxidant properties including scavenging of free radicals, low UA levels could be related to poor outcomes in TBI patients. However, Liu H et al., indicated that UA levels were significantly lower in TBI patients that had a good outcome, and low serum UA level was an independent predictor of good outcome after TBI [3]. This contradicting report may be due to the difference of time for blood sampling. Following TBI, active antioxidant defence in the brain could result in consumption of UA in the serum, and thus, recovery from a decreased serum UA should be predictive of good clinical recovery.

In conclusion, the serum UA level is important in TBI patients as Hatefi M et al., mentioned because UA could act to attenuate neuronal loss in numerous TBI animal models. Therefore, further clinical studies are warranted to elucidate the effect of serum UA and sodium levels on TBI patients' outcomes.

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