Journal of Neurology & Neurophysiology

Ramtinfar, J Neurol Neurophysiol 2016, 7:6
DOI: 10.4172/2155-9562.1000406

Commentary Open Access

Brain-Kidney Cross-Talk in Neurotrauma

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Received date: December 16, 2016; **Accepted date:** December 21, 2016; **Published date:** December 28, 2016

Citation: Ramtinfar S (2016) Brain-Kidney Cross-Talk in Neurotrauma. J Neurol Neurophysiol 7: 406. doi:10.4172/2155-9562.1000406

Commentary

Acute Kidney Injury (AKI) plays a significant role in determining the prognosis of patients with head trauma. Head Trauma itself could lead to AKI by vasoconstrictation of kidneys induced by sympathetic hyperactivity, the expected response after brain damage.

Mannitol, an inevitable medication in neurotrauma setting to lower the ICP and prevent further edema and herniation, increases the risk of AKI by nephrotoxicity [1].

Accompanied rhabdomyolysis, sepsis and prescription of other nephrotoxic medications are all factors that make the patient with head injury extremely prone to AKI.

We study this subset of patients as patients who are suffering from severe head injury, but their management, it's much more than that. This is a real challenge because of setting teeming failures of many organs. Kidneys could be affected by inflammatory mediators released because of liver failure or respiratory failure.

Although AKI could be a direct effect of brain injury, it could affect the brain by itself. AKI causes brain endothelial damage and inflammation leading to brain edema and encephalopathy, worsening the outcome of patients. Imbalance of water-sodium, electrolyte abnormality and acute accumulation of uremic neurotoxins has been recognized as responsible mechanisms [2-4].

The incidence of AKI in neurotrauma has been reported from as low as 1.5% to as high as 23%. The wide reported range is because a different criterion has been used to make the diagnosis of acute kidney injury.

We preferred KDIGO criteria for diagnosis the AKI and to report the incidence; this diagnostic system includes both previously diagnostic systems described (AKIN and RIFLE).

Based on organization of Kidney Disease Improving Global Outcome (KDIGO), AKI is diagnosed by alterations in urine output in a six hour span of admission or serum creatinine within 48 h - 7 days of admission.

We have reported a high incidence of AKI (25.3%) in patients with severe type of traumatic brain injury. The subset of patients with AKI had shown significant higher rates of mortality, multi organ failure and poor neurologic outcome.

Although there is no definite therapy for early diagnosed AKI and the only available choice to date is resuscitative management, applying renal hygiene guidelines from the first moments of admission is always protective. Tight monitoring of volume issues and electrolyte imbalance especially potassium levels and observing the patient closely for early signs of uremia might be considered as the cornerstone of prevention of AKI. Finding new biomarkers to diagnose the at-risk population for renal injury or to detect the AKI in earlier stages will serve as a very great help to save and restore the kidney function [5].

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