Hyponatremia after traumatic brain injury

Hyponatremia is frequent in patients suffering from traumatic brain injury, subarachnoid hemorrhage, or following intracranial procedures, with approximately 20% having a decreased serum sodium concentration to <125 mmol/L. The pathophysiology of hyponatremia in neurotrauma is not completely understood, but in large part is explained by the syndrome of inappropriate secretion of antidiuretic hormone (SIADH). The abnormal water and/or sodium handling creates an osmotic gradient promoting the shift of water into brain cells, thereby worsening cerebral edema and precipitating neurological deterioration. Unless hyponatremia is corrected promptly and effectively, morbidity and mortality increases through seizures, elevations in intracranial pressure, and/or herniation. The excess mortality in patients with severe hyponatremia (<125 mmol/L) extends beyond the time frame of hospital admission, with a reported mortality of 20% in hospital and 45% within 6 months of follow-up. Current options for the management of hyponatremia include fluid restriction, hypertonic saline, mineralocorticoids, and osmotic diuretics. However, the recent development of vasopressin receptor antagonists provides a more physiological tool for the management of excess water retention and consequent hyponatremia, such as occurs in SIADH [1].

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Kleindienst A, Hannon MJ, Buchfelder M, Verbalis JG. Hyponatremia in Neurotrauma: The Role of Vasopressin. J Neurotrauma. 2016 Apr 1;33(7):615-24. doi: 10.1089/neu.2015.3981. Epub 2015 Dec 23. Review. PubMed PMID: 26472056.