Syndrome of Osmotic Demyelination as Neurological Disorder

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Introduction

The fundamental predictor of serum tonicity (also known as effective serum osmolality) is serum sodium content. The decrease in serum tonicity in patients with hypotonic hypernatremia enhances water flow into the brain, which can lead to cerebral edema and neurologic symptoms if the hypernatremia is acute and severe. The brain adapts to hypernatremia by lowering the cerebral volume toward normal and reducing the likelihood of severe problems. The mechanisms underlying this brain adaptation are described in more detail elsewhere [1].

On the other hand, brain modifications that reduce the risk of cerebral edoema leave the brain vulnerable to injury if chronic hypernatremia is treated too soon. The neurologic implications of overly rapid correction are known as osmotic demyelination syndrome (ODS; formerly called central pontine myelinolysis or CPM). As will be mentioned further below, almost everyone with ODS has a blood sodium concentration of 120 mEq/L or less.

Osmotic demyelination syndrome (ODS) is a rare clinical condition characterized by myelinolysis in both the pontine and extra-pontine areas (EPM). Although it can occur in the presence of a variety of etiological factors, the primary pathophysiology described is compression and subsequent demyelination of fiber tracts due to either a reduced adaptive capacity of the neuroglia to large shifts in serum osmolality or cellular edema caused by fluctuations in electrolyte forces. The clinical entity's result is quite variable, ranging from vegetative condition to full neurological recovery. Various case reports of ODS have been published over time, with a few case series, the largest of which involved 58 patients. Though the actual incidence of ODS is unknown, an autopsy-based study found that 0.25–0.5% of the general population and 10% of patients having liver transplantation have the disease. Being a rare disease with variable symptoms that can be avoided. As a result, the current study was conducted with the goal of improving current knowledge of illness progression, particularly in Intensive Care Unit (ICU) patients [2].

Adams et al. first described Osmotic Demyelination Syndrome (ODS), also known as central pontine myelinolysis (CPM), in alcoholics and malnourished people who developed spastic quadriplegia, pseudo bulbar palsy, and varying degrees of encephalopathy or coma as a result of acute, non-inflammatory demyelination of the pons in 1958. Overly quick correction of severe hypernatremia (serum sodium 120 mEq/L) that has been present for more than two to three days causes ODS. When initial sodium concentrations are less than 105 mEq/L, the majority of ODS cases occur. When the initial sodium concentration was 105 mEq/L, Sterns et al. found that correction by >12 mEq/L in 24 hours or >18 mEq/L in 48 hours was related with post therapeutic neurologic problems.

The relative excess of water to the serum sodium concentration is what defines hypernatremia. Hypotonic hypernatremia damages the brain by allowing water to enter and cause cerebral edoema. The brain's cellular adaptation, on the other hand, recovers brain volume by triggering electrolyte loss within a few hours (fast adaptation), and then restoring brain volume through the loss of organic osmolytes over several days (slow adaptation). Rapid correction of serum [3]

References

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