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Acute Severe Hyponatremia in Patient with Psychogenic Polydipsia, Learning Disability and Epilepsy

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Abstract : Introduction: The diagnosis and management of severe hyponatremia in neuropsychiatric patients present a significant challenge to physicians. Several factors contribute, including diagnostic shadowing and attributing abnormal behavior to intellectual disability or psychiatric conditions. Hyponatraemia is the commonest electrolyte abnormality in the inpatient population, ranging from mild/asymptomatic, moderate to severe levels with life-threatening symptoms such as seizures, coma and death. There are several documented fatal case reports in the literature of severe hyponatremia secondary to psychogenic polydipsia, often diagnosed only in autopsy. This paper presents a case study of acute severe hyponatremia in a neuropsychiatric patient with early diagnosis and admission to intensive care. Case study: A 21-year old Caucasian male with known epilepsy and learning disability was admitted from residential living with generalized tonic-clonic self-terminating seizures after refusing medications for several weeks. Evidence of superficial head injury was detected on physical examination. His laboratory data demonstrated mild hyponatremia (125 mmol/L). Computed tomography imaging of his brain demonstrated no acute bleed or space-occupying lesion. He exhibited abnormal behavior - restlessness, drinking water from bathroom taps, inability to engage, paranoia, and hypersexuality. No collateral history was available to establish his baseline behavior. He was loaded with intravenous sodium valproate and leveritircaetam. Three hours later, he developed vomiting and a generalized tonic-clonic seizure lasting forty seconds. He remained drowsy for several hours and regained minimal recovery of consciousness. A repeat set of blood tests demonstrated profound hyponatremia (117 mmol/L). Outcomes: He was referred to intensive care for peripheral intravenous infusion of 2.7% sodium chloride solution with two-hourly laboratory monitoring of sodium concentration. Laboratory monitoring identified dangerously rapid correction of serum sodium concentration, and hypertonic saline was switched to a 5% dextrose solution to reduce the risk of acute large-volume fluid shifts from the cerebral intracellular compartment to the extracellular compartment. He underwent urethral catheterization and produced 8 liters of urine over 24 hours. Serum sodium concentration remained stable after 24 hours of correction fluids. His GCS recovered to baseline after 48 hours with improvement in behavior -he engaged with healthcare professionals, understood the importance of taking medications, admitted to illicit drug use and drinking massive amounts of water. He was transferred from high-dependency care to ward level and was initiated on multiple trials of anti-epileptics before achieving seizure-free days two weeks after resolution of acute hyponatremia. Conclusion: Psychogenic polydipsia is often found in young patients with intellectual disability or psychiatric disorders. Patients drink large volumes of water daily ranging from ten to forty liters, resulting in acute severe hyponatremia with mortality rates as high as 20%. Poor outcomes are due to challenges faced by physicians in making an early diagnosis and treating acute hyponatremia safely. A low index of suspicion of water intoxication is required in this population, including patients with known epilepsy. Monitoring urine output proved to be clinically effective in aiding diagnosis. Early referral and admission to intensive care should be considered for safe correction of sodium concentration while minimizing risk of fatal complications e.g. central pontine myelinolysis.

Keywords : epilepsy, psychogenic polydipsia, seizure, severe hyponatremia
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