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Original Research Paper

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A CASE OF OSMOTIC DEMYELINATION SYNDROME AFTER THE CORRECTION OF SEVERE HYPONATREMIA IN ACUTE GASTROENTERITIS

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ABSTRACT Osmotic demyelination syndrome (ODS), previously known as central pontine myelinosis is an uncommon disorder, characterized by non-inflammatory demyelination involving the pons and other areas of the central nervous system, which may occur for instance as a consequence of an overly rapid correction of hyponatremia. Here we report a case of ODS in a patient with acute gastroenteritis leading to hyponatremia, for which he was given hypertonic saline correction. Also, we observed additional risk factors for the development of this syndrome in our patient which could have been prevented.

KEYWORDS :

CASE PRESENTATION

48-year-old male presented to us with chief complains of difficulty in speech, difficulty in walking, unable to take food and altered sensorium in form of not talking to or recognizing and communicating with relatives for 2 weeks. Patient had a medical history of chronic alcohol abuse and hypertension. On general examination, he was fairly built and nourished male with no signs of dehydration, cachexia and significant abnormalities. His blood pressure was 150/90 mmHg. Other vital signs were normal. On central nervous system examination, there was hyperreflexia in all limbs, with increased tone. Power was normal with no focal neurological deficit or stroke like features. His pupils were bilaterally equal and normally reacting to light with a normal fundus examination. There was no history of fever, altered bowel and bladder habits, headache, seizures, or syncope. No meningeal signs could be elicited on examination. His GCS was 14/15. Rest systemic examination was normal.

Biochemical assessment performed at admission revealed hyponatremia (115 mmol/L) and hypokalemia (3.0mmol/L). His renal function tests showed blood of urea 53 mg/dl and serum creatinine of 1.74 mg/dl. Other blood investigations were normal. The patient was promptly admitted to the intensive care unit of the hospital and he was given 3% hypertonic saline correction along with K-lactate supplementation. Patient underwent MRI brain study 2 days later in view of persistent neurological symptoms which showed typical appearance of osmotic demyelination syndrome.

On further questioning the relatives they gave a history of an episode of acute and severe gastroenteritis 20 days back for which he was admitted in a local health care facility and given symptomatic treatment and then discharged. No investigations were done at that time.

On repeat investigations, serum sodium was persistently on lower side but hypertonic saline was stopped due to findings of ODS. Further investigations were done which showed urinary sodium loss(59mEq/L), serum osmolality of 277 mosmol/kg and urine osmolality of 637 mosml/kg water. Additional tests for TSH and total protein- serum albumin was done which turned to be normal. Patient was advised for fluid restriction and strict fluid input- output monitoring was done. Supportive treatment was continued especially considering his alcohol abuse factor and hypertension. His serum sodium levels were frequently monitored which were still on lower side. We started him on oral Tolvaptan and fluid restriction was removed after discussing the case with nephrologist. His sodium levels started to rise gradually. He was also given oral potassium chloride for hypokalemia. Patient gradually improved sensorium wise and now could recognize and communicate with relatives. Also, he was able to move around with little support, could go to washroom and was able to eat food by himself. Physiotherapy was given to the patient as a part of the supportive treatment which was continued. Before discharge, serum sodium was 133 mmol/L and serum potassium was 4.2 mmol/L. Other investigations, including his renal function tests were normal. He was discharged successfully with only complain of residual dysarthria. Prognosis was explained to the relatives and patient was asked for follow up after 7 days.

DISCUSSION

Here we like to discuss the factor that patient developed ODS despite the recommended rate of sodium correction. Also, associated hypokalemia and medical history of chronic alcohol abuse could predispose such unfortunate pathology even with slow correction. ODS usually has irreversible or only partially reversible recovery in some cases. In this patient we had exceptionally good recovery if not complete. Prompt identification and correction of electrolyte abnormalities could have prevented a simple gastroenteritis turning into a rare and fatal syndrome. This abstract should give more impetus to clinicians and researchers to unravel the mechanisms of exact underlying pathology and thus its preventive and treatment measures.

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