CASE REPORT

Osmotic Demyelination Syndrome Secondary to Colonic Pseudo-Obstruction and Resistant Hypokalaemia: A Case Report

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Abstract

Osmotic demyelination syndrome (ODS) commonly occurs following overly rapid correction of chronic hyponatremia; however, other precipitants are described. A 63-year-old female was admitted with abdominal distension and found to have severe hyponatraemia and hypokalaemia. Hypertonic saline was initially administered to correct sodium levels within recommended limits. She subsequently developed colonic pseudo-obstruction and an unexpected rise of serum sodium by 16 mmol/L occurred on day four in the absence of any further hypertonic saline therapy. A few days later she became drowsy and magnetic resonance imaging showed changes consistent with ODS. This case highlights how severe hypokalaemia precipitating colonic pseudo-obstruction, followed by rapid change in sodium from the intestinal third spacing of fluid and repeated phosphate enemas contributed to ODS. Early recognition of compound electrolyte disturbances and strict monitoring of fluid balance are essential to avoid such complications.

Keywords: osmotic demyelination syndrome (ODS), hyponatremia, hypokalaemia, Ogilvie syndrome, colonic pseudo-obstruction

Background

Osmotic demyelination syndrome (ODS) is a potentially fatal neurological condition associated with overly rapid corrections of chronic hyponatraemia. When serum sodium levels decrease, an immediate adaptive response is triggered to prevent cerebral oedema. The brain releases osmotic molecules to adjust to the changes in tonicity, and these changes usually occur within two days. When serum sodium levels are corrected too rapidly after these adaptive changes have occurred, pontine and extrapontine myelinolysis can result. A meta-analysis of 11 cohort studies involving 26,710 patients confirmed that correction rates exceeding 8-12 mmol/L within 24 hours are associated with a

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significantly increased risk of ODS (odds ratio, 3.16; 95% CI, 1.54-6.49). However, some patients developed ODS despite correction within recommended limits.[1] A recent case series demonstrates the wide range of clinical circumstances leading to demyelination syndrome even when sodium correction followed recommended guidelines. The contributory factors described include hypokalaemia, malnutrition and alcoholism.[2] Sri Lankan data on ODS is scarce. We describe a case of ODS which occurred in the background of hypokalaemia and colonic pseudo-obstruction.

Case presentation

A 63-year-old lady with a past medical history of diabetes mellitus, dyslipidaemia and hypertension presented to a medical ward with generalized body weakness of five days' duration. She also complained of regurgitation and bloating of the abdomen for one week, and her bowels had not opened for two days. She was taking metformin 500mg bd, atenolol 25mg bd, losartan 50mg bd, atorvastatin 20mg nocte and hydrochlorothiazide 25mg mane. On examination, she was alert with a GCS of 15/15 and her abdomen was mildly distended with reduced bowel sounds. Her volume status appeared to be normal. Blood investigations revealed a serum sodium of 102 mmol/L and a serum potassium of 2.1 mmol/L. Hydrochlorothiazide and losartan were omitted, and a bolus of 3% NaCl, 100 mL, was administered over 30 minutes, followed by a 40 mmol potassium chloride (KCl) correction. The repeat electrolytes were Na+ 106 mmol/L and K^+ 2.7 mmol/L, and a second bolus of 3% NaCl, 100 mL, was administered with 20 mmol KCl. The repeat sodium increased to 109 mmol/L. Thereafter, no further hypertonic saline was given. By day four, her abdomen was getting progressively distended, and bowels had not opened for the last four days. An X-ray of the abdomen (Figure 2) showed gaseous distension of the colon with dilated small bowel loops. She was administered sodium phosphate enemas several times. Contrast-enhanced CT Abdomen was reported as colonic pseudo-obstruction with mild prominence of the distal ileal loops. On day four, her electrolytes were repeated, and Na^+ had rapidly and unexpectedly risen to 125mmol/L. On days three and four of the hospital stay, only 0.9% saline was given as a maintenance fluid. Over the next few days, she had persistent and resistant hypokalaemia with ongoing paralytic ileus. By day eight, her level of consciousness started to deteriorate, and it was suspected she had developed osmotic demyelination. MRI brain with contrast confirmed demyelination in the pontine and extra-pontine regions (Figure 3). Axial FLAIR MRI brain showed a symmetrical central pontine hyperintense lesion involving the basal pons, with relative sparing of the peripheral pons, in keeping with central pontine myelinolysis. By this time, she required ventilatory support due to low GCS and poor respiratory effort. A course of IV methylprednisolone and IV immunoglobulins was given; however, there was no improvement in her clinical condition, and sadly she succumbed to ventilator-associated pneumonia twenty-seven days after admission. A summary of her investigations is illustrated in Figure 1 and Table 1.

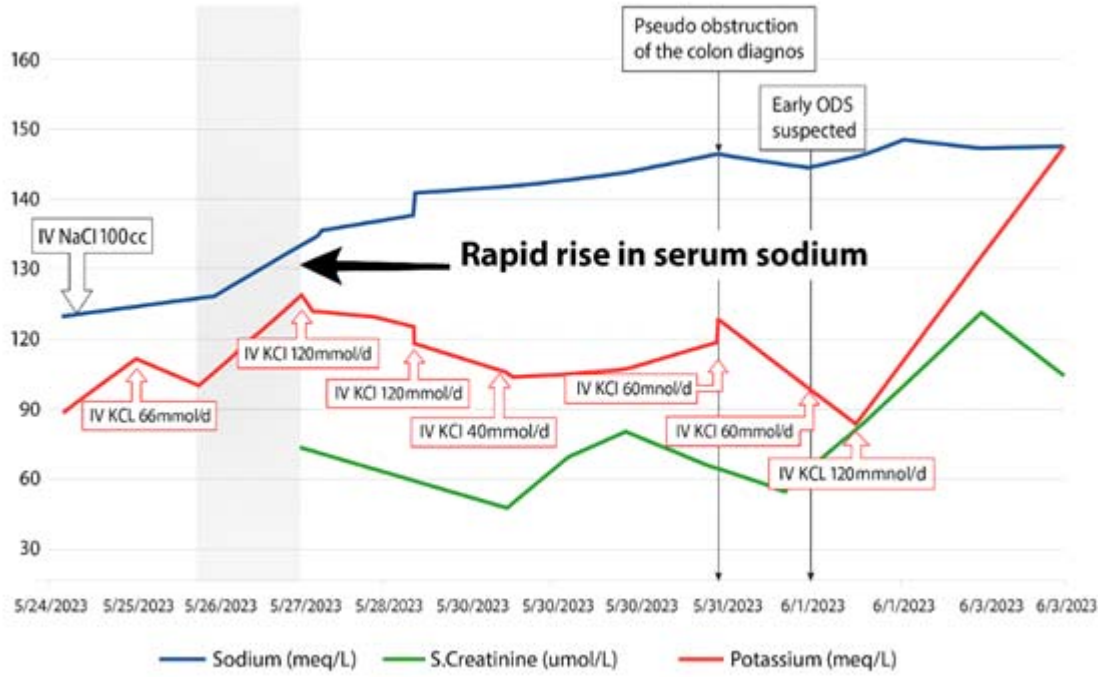

Figure 1. Trends in serum sodium, potassium, and creatinine levels during the first 11 days of hospitalization.

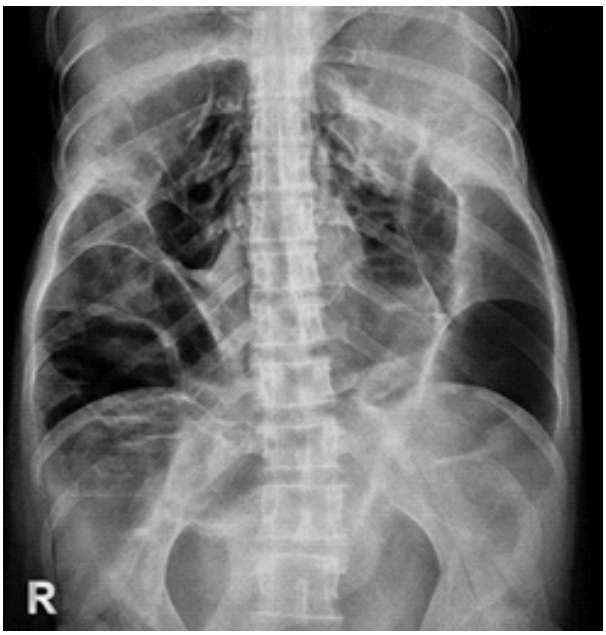

Figure 2. Abdominal X-ray showing dilated large bowel loop consistent with acute colonic pseudo-obstruction.

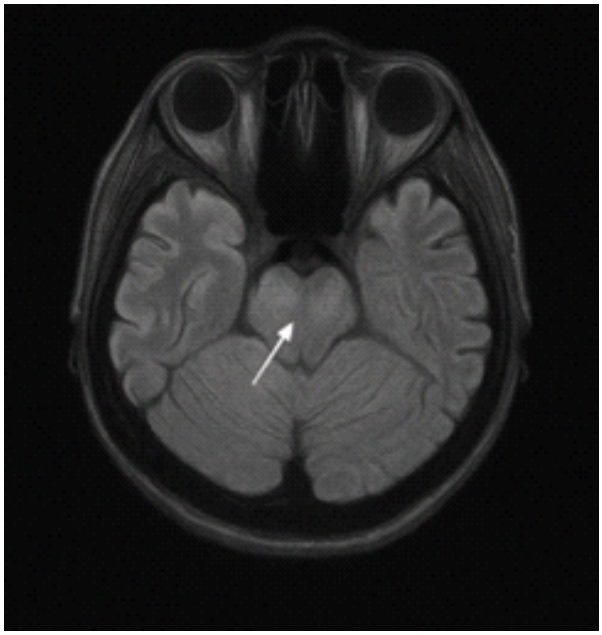

Figure 3. Axial FLAIR MRI brain showed a symmetrical central pontine hyperintense lesion (arrow) involving the basal pons.

Table 1. The changes in serum electrolytes over time

Day	*Serum Na+ (mmol/L)*	*Serum K+ (mmol/L)*
0	102	2.1
1	106	2.7
2	109	2.9
3	110	2.8
4	125	2.5
8	128	3.0

Discussion

The most common aetiology of ODS in clinical practice is rapid correction of serum sodium exceeding recommended limits of > 8mEq/L in 24 hours and is particularly seen when hyponatremia has been there for 48 hours or more. The mechanism of myelin loss in ODS is still poorly understood. One suggestion is that the increase in serum sodium produces an osmotic endothelial injury that releases myelinotoxic factors and initiates vasogenic oedema and apoptosis.[3] Another explanation is that astrocytes expel inorganic ions and organic osmolytes such as taurine, myoinositol and glutamine within 24-48h, thereby preventing cerebral oedema but leaving the tissue osmotically fragile.[4] A subsequent rise in serum sodium forces water efflux, provoking oligodendrocyte shrinkage, cytoskeletal disruption and myelin splitting.[4]

Our patient was initially admitted due to hyponatremia and hypokalaemia, likely induced by hydrochlorothiazide. The rapid rise in sodium from day three to day four was not due to iatrogenic administration of hypertonic saline but to the coexisting conditions. She concurrently had severe and resistant hypokalaemia leading to colonic pseudo-obstruction. Intestinal obstruction causes third-space fluid shifting, in which intravascular fluid is redistributed to the extravascular compartment, leading to hypovolaemia. Typically, this is also associated with hyponatremia, and careful correction of intravascular dehydration is important in these cases; otherwise, sodium levels can rise.

Symptomatic treatment for intestinal obstruction often involves the use of enema, such as sodium phosphate enema. Phosphate enemas induce osmotic diarrhoea, resulting in a state of hypernatraemic dehydration due to disproportionate water loss and an increase in serum sodium levels.[5,6,7] Additionally, toxins which accumulate secondary to bowel obstruction can hinder the kidney's ability to excrete sodium.[6] A systematic review on adverse effects of sodium phosphate enemas describes hypernatraemia as one of the main metabolic side effects.[7] We believe the rise of sodium by 16 mmol/L seen on day four of admission was due to these processes.

Profound hypokalaemia (<2.5 mmol/L) further compromises volume regulation. Potassium depletion suppresses Na^+/K^+ ATPase activity and impairs NKCC1 mediated regulatory volume increase, thereby hindering rapid accumulation of osmolytes. In rat models, the threshold sodium shift required to trigger demyelination falls by approximately 50% in the setting of severe hypokalaemia.[8] More recent reviews continue to highlight hypokalaemia as a modifiable co-risk.[4] Our case adds to the limited Asian data set and underscores the need for vigilant potassium replacement when correcting sodium. Regional predilection for the central pons, basal ganglia, and thalami is attributed to their high oligodendrocyte density, limited astrocytic glycogen reserves, and relative paucity of aquaporin-4 water channels.[9] Following the initial osmotic injury, microglial activation and release of pro-inflammatory cytokines propagate secondary demyelination over 2-6 days, explaining the latency between biochemical insult (day 4) and neurological deterioration (day 8) observed in this patient.[4,9]

Alternative causes of encephalopathy, including Wernicke's syndrome, acute stroke and sepsis-associated encephalopathy, were considered in our patient. However, the temporal relationship of the neurological deficits with the metabolic insult and the typical MR imaging findings favoured the diagnosis of ODS. The absence of osmolality measurements is acknowledged as a limitation; however, the timeline and imaging findings strongly support ODS. Evidence for disease-modifying therapy in established ODS remains anecdotal. Case series suggest a possible benefit from early plasmapheresis or immunoglobulin.[10] Neither intervention altered the course in our patient, consistent with reports that delayed initiation (>7 days) is rarely effective.

Conclusions

ODS can develop via indirect mechanisms even when guideline-compliant sodium correction is followed. Severe hypokalaemia and bowel dysmotility should alert clinicians to the risk of abrupt, uncontrolled rises in serum sodium. Comprehensive electrolyte monitoring, judicious use of fluids and phosphate enemas alongside early radiological evaluation of ileus are critical preventive steps.

Key points

- Osmotic demyelinating syndrome can occur in situations other than iatrogenic over correction of serum sodium.
- Co-existent hypokalaemia can increase the risk of developing ODS.
- Hypokalaemia leading to pseudo-obstruction of the bowel can cause rapid changes in serum sodium, due to fluid shifts and administration of sodium phosphate enemas.

Authors' contributions

Concept and design: DL, KG, PJ

Literature review: TPP, VWV

Compilation of manuscript: DL, TPP, VWV, KG

Manuscript editing and proofreading: DL, PJ

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References

1. Suppadungsuk S, Krisanapan P, Kazeminia S, Nikravangolsefid N, Singh W, Prokop LJ, *et al*. Hyponatremia Correction and Osmotic Demyelination Syndrome Risk: A Systematic Review and Meta-Analysis. *Kidney Medicine*. 2025; **7**(3):100953. doi: 10.1016/j.xkme.2024.100953
2. Yu Z, Wang C. Osmotic demyelination syndrome: Case report and literature retrospect. *Medicine*. 2024; **103**(51): e41031. doi: 10.1097/MD.0000000000041031
3. DeLuca GC, Nagy ZS, Esiri MM, Davey P: Evidence for a role for apoptosis in central pontine myelinolysis. *Acta Neuropathol* 2002; **103**: 590-98. doi: 10.1007/s00401-001-0508-2
4. Klavansky D, Marquez DL, Vijayan M, Reynolds AS. Osmotic Demyelination Syndrome Associated with Uremia and Elevated Serum Osmolality. *Neurocrit Care*. 2024; **40**(3): 1218-22. doi: 10.1007/s12028-024-01956-w
5. Beraldo DO, Duarte SBCP, Santos RB, Mendes CG, Silveira MP, Neto AS, *et al*. Pontine Myelinolysis Caused by Hypovolemic Hypernatremia. *Case Reports in Nephrology*. 2020; **2020**: 1-4. eCollection 2020. doi: 10.1155/2020/4079098.
6. Quarrie I, Whipple GH. Renal function influenced by intestinal obstruction. *J Exp Med*. 1919; **29**(4): 397-419.
7. Mendoza J, Legido J, Rubio S, Gisbert JP. Systematic review: the adverse effects of sodium phosphate enema. *Aliment Pharmacol Ther*. 2007; **26**(1): 9-20. doi: 10.1111/j.1365-2036.2007.03354.x
8. Sugimoto T, Central pontine myelinolysis associated with hypokalaemia in anorexia nervosa., *J Neurol Neurosurg Psychiatry* 2003. doi: 10.1136/jnnp.74.3.353.
9. Verbalis JG, Goldsmith SR, Greenberg A, *et al*. Diagnosis, evaluation, and treatment of hyponatremia: expert panel recommendations. *Am J Med*. 2013; **126**(Suppl 1): S1-S42. doi: 10.1016/j.amjmed.2013.07.006
10. Kumon S, Usui R, Kuzuhara S, Nitta K, Koike M: The improvement of the outcome of osmotic demyelination syndrome by plasma exchange. *Intern Med*. 2017; **56**: 733-6. doi: 10.2169/internalmedicine.56.7491