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CASE REPORT
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SEVERE HYPONATREMIA AND METABOLIC ACIDOSIS IN A TODDLER WITH STEROID – RESISTANT NEPHROTIC SYNDROME FOLLOWING ACUTE GASTROENTERITIS

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ABSTRACT

Background

Steroid-resistant nephrotic syndrome (SRNS) accounts for 3–15% of childhood nephrotic syndrome and is associated with significant morbidity, progression to chronic kidney disease, and susceptibility to complications. Intercurrent infections, such as gastroenteritis, may precipitate severe fluid and electrolyte derangements, including hyponatremia and metabolic acidosis, which can be life-threatening in pediatric SRNS.

Case Presentation

We report a 3-year-old male with SRNS and underlying minimal change nephropathy who presented with acute gastroenteritis, complicated by severe hyponatremia (Na^+ 120 mEq/L) and metabolic acidosis (HCO_3^- 13.9 mEq/L). The child developed vomiting and watery diarrhoea without fever or oliguria. Laboratory investigations confirmed critical electrolyte disturbances despite preserved renal function. Management involved prompt intravenous fluids, cautious electrolyte correction, intravenous hydrocortisone, and broad-spectrum antibiotics, followed by supportive therapy. The child's condition stabilized by Day 5, allowing transition to oral therapy, including alternate-day prednisolone, enalapril, atorvastatin, calcium carbonate with vitamin D, zinc, folic acid, nitazoxanide, loperamide, co-trimoxazole prophylaxis, and probiotics.

Discussion

This case underscores the complexity of SRNS with minimal change nephropathy during intercurrent infections. Severe hyponatremia and metabolic acidosis compounded by diarrheal losses and hypoalbuminemia highlight the need for vigilant monitoring. Notably, corticosteroids were continued without additional immunosuppressive agents, reflecting individualized clinical decision-making in real-world practice, which may diverge from guideline recommendations advocating calcineurin inhibitors in SRNS.

Conclusion

Children with SRNS are particularly vulnerable to electrolyte crises during infections. Early recognition and correction of hyponatremia and metabolic acidosis are crucial to prevent fatal complications. This case emphasizes the importance of tailored therapeutic strategies and highlights the gap between guidelines and real-world management in pediatric SRNS.

INTRODUCTION

Nephrotic syndrome is a kidney disorder characterized by swelling, proteinuria, hypoalbuminemia, and hyperlipidemia. It occurs when the kidney's tiny filtering units, the glomeruli, are damaged, allowing protein to leak from the blood into the urine. This leads to fluid accumulating in the body's tissues, causing oedema, and can increase the risk of blood clots and other serious complications[1,2] .

Corticosteroid therapy is effective in achieving remissions of symptoms in more than 85% of patients and are classified as steroid-sensitive nephrotic syndrome (SSNS). However, approximately 3 – 15 % of children develop steroid-resistant nephrotic syndrome (SRNS), which is associated with higher morbidity, progression to chronic kidney disease, and increased susceptibility to complications[3-5].

Amongst the common complications of NS, infections are particularly frequent due to loss of immunoglobulins in the urine, impaired complement activity, and long-term immunosuppression[6,7]. Intercurrent infections can lead to severe fluid and electrolyte disturbances, especially in children, along with increasing the risk of relapse. While acute gastroenteritis is a common childhood illness, its impact in the setting of SRNS can be profound, owing to overlapping mechanisms of intravascular volume depletion, altered renal handling of electrolytes, and hypoalbuminemia-related fluid shifts.

Electrolyte imbalances, though often under-reported in the literature, may pose immediate life-threatening risks. Severe hyponatremia can precipitate neurological complications such as seizures, cerebral oedema, and coma, while metabolic acidosis can compromise cardiovascular stability and worsen renal function[8,9]. The dual occurrence of these abnormalities in a child with SRNS and underlying MCN presents unique diagnostic and therapeutic challenges that warrant reporting.

Minimal change nephropathy (MCN) is the most common histopathological lesion observed in childhood nephrotic syndrome. Importantly, MCN typically shows an excellent response to corticosteroids, with more than 90% of cases achieving remission[10,11]. The occurrence of steroid resistance in MCN is therefore uncommon and often prompts consideration of alternative or adjunct immunosuppressive therapies. This makes each reported case of SRNS with underlying MCN of significant clinical interest, particularly when complicated by additional systemic derangements.

In this case report, we describe a toddler with steroid-resistant minimal change nephropathy who presented with acute gastroenteritis, complicated by severe hyponatremia and metabolic acidosis. This case underscores the importance of close monitoring for electrolyte derangements in pediatric SRNS and emphasises the need for timely, individualized management strategies during intercurrent illnesses.

CASE PRESENTATION

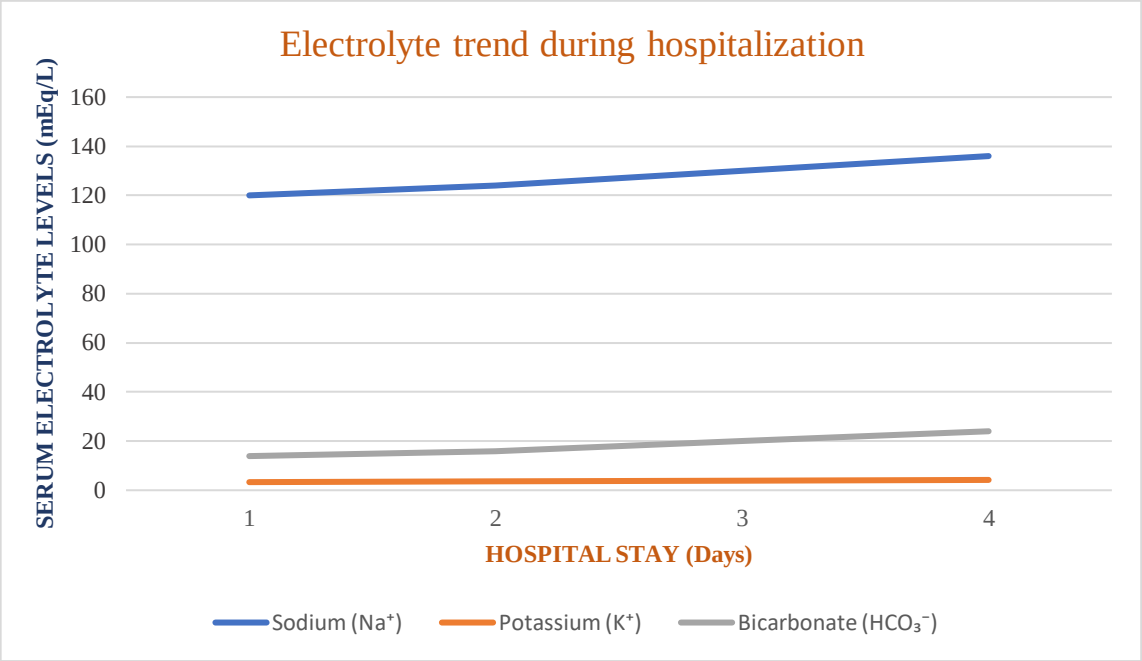
A 3-year-old developmentally normal male was admitted with a 5-day history of non-projectile, non-bilious vomiting of sudden onset and watery loose stools. There was no fever, abdominal pain, lethargy, respiratory difficulty, or oliguria. The patient was a known case of SRNS on treatment, with underlying minimal change nephropathy (MCN).

On admission, the child’s weight was 11.6 kg and height 88 cm. He was afebrile (98.6°F) with a pulse rate of 84/min, respiratory rate 24/min, and an elevated blood pressure of 146/104 mmHg (average 114 mmHg). Oxygen saturation was 98% on room air, and systemic examination of the cardiovascular, respiratory, abdominal, and neurological systems was unremarkable.

Laboratory investigations revealed a low serum urea of 5 mg/dL and creatinine of 0.1 mg/dL, values that are within the expected range for age and consistent with preserved renal function[12]. The electrolyte profile showed critical abnormalities: serum sodium was 120 mEq/L, indicating severe hyponatremia; potassium was mildly reduced showing mild transient hypokalaemia at 3.3 mEq/L; chloride was 84 mEq/L; and bicarbonate was 13.9 mEq/L, confirming a metabolic acidosis. Stool analysis showed mucus, occult blood positivity, 8–10 RBCs/HPF, and 4–5 pus cells/HPF, consistent with infective diarrhoea[13]. These findings emphasized that the main metabolic derangements were related to electrolyte and acid–base imbalance rather than renal dysfunction.

Table 1. Laboratory Investigations at Admission

Test	Result	Reference Range	Interpretation
Serum Urea	5 mg/dL	15–40	Low (dilutional)
Serum Creatinine	0.1 mg/dL	0.4–1.3	Low (normal renal function)
Sodium (Na^+)	120 mEq/L	134–145	Severe Hyponatremia
Potassium (K^+)	3.3 mEq/L	3.4–5.4	Mild Hypokalaemia
Chloride (Cl^-)	84 mEq/L	92–102	Low
Bicarbonate (HCO_3^-)	13.9 mEq/L	22–30	Metabolic Acidosis
Stool Occult Blood	+++	Negative	Positive for infection
RBC (HPF)	8–10	<5	Abnormal



During hospitalization, the patient was treated with intravenous fluids and careful electrolyte correction, along with hydrocortisone, ciprofloxacin, metronidazole, and amikacin. Supportive measures included ondansetron for vomiting, pantoprazole for gastric protection, and intravenous magnesium sulphate. By Day 5, the child was stabilized and transitioned to oral therapy. At discharge, he was prescribed alternate-day prednisolone along with enalapril, atorvastatin, calcium carbonate with vitamin D, zinc, folic acid, nitazoxanide, loperamide, co-trimoxazole prophylaxis, and probiotics[14,15]. This treatment course demonstrates how the acute crisis was managed stepwise, with initial intravenous stabilization followed by tailored oral maintenance therapy.

Table 2: Treatment Timeline

Day of Admission	Intervention	Outcome
Day 1	IV fluids and electrolyte correction; Hydrocortisone (IV) ;	Stabilized hydration
Day 1–3	IV antibiotics (ciprofloxacin, metronidazole, amikacin)	Resolution of diarrhoea
Day 2–4	Ondansetron (IV); Pantoprazole (IV, PPI); Magnesium sulphate (IV); supportive care	Symptom relief
Day 5	Transition to oral therapy	Tolerated feeds
Discharge	Oral steroids (Prednisolone), Enalapril, Atorvastatin, Calcium carbonate + Vit D, Zinc Sulphate, Folic acid, Loperamide, Nitazoxanide, Co-trimoxazole, Saccharomyces boulardii	Stable

DISCUSSION

This case illustrates the dual complexity of managing SRNS (MCN subtype) and electrolyte crisis during intercurrent gastroenteritis. Severe hyponatremia (Na^+ 120 mEq/L) and metabolic acidosis (HCO_3^- 13.9 mEq/L) were precipitated by diarrheal electrolyte losses, compounded by hypoalbuminemia-related fluid shifts. Prompt diagnosis and rebalancing of these electrolytes were critical to prevent neurological and cardiovascular complication.

Although the child had SRNS, low-dose corticosteroids were continued. This aligns with standard practice in some SRNS protocols, both to maintain immune suppression and to prevent adrenal insufficiency in children on long-term steroids. Stress-dose steroids may also be required during acute illness[16]. Furthermore, some patients with SRNS may still demonstrate partial responsiveness to steroids, and therefore low-dose or alternate-day corticosteroids are sometimes maintained as part of long-term management.

Despite a diagnosis of SRNS, corticosteroids were continued, likely reflecting the need for stress-dose coverage in a child on chronic steroid therapy, as well as the possibility of partial responsiveness to steroids. Notably, no additional immunosuppressive agent was prescribed, which diverges from current guidelines that recommend calcineurin inhibitors as first-line therapy in SRNS[3,4] .

This divergence underscores the gap between guideline-directed management and real-world practice, particularly in resource-limited settings or where treatment is individualized for minimal change nephropathy, as MCN may still show late responsiveness to steroids[17,18]. This case therefore not only emphasizes the need for vigilant electrolyte monitoring in SRNS with intercurrent illness, but also highlights the reality of individualized, resource-influenced therapeutic decision-making in pediatric nephrology [19,20].

CONCLUSION

Children with SRNS are vulnerable to electrolyte crises during intercurrent infections. Severe hyponatremia and metabolic acidosis require rapid diagnosis and management to prevent life-threatening complications. Additionally, the persistence of corticosteroid therapy without escalation to other immunosuppressants in this case reflects individualized clinical decision-making for SRNS with MCN, underscoring the gap between guideline recommendations and real-world practice.

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