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RELAPSING NEPHROTIC SYNDROME COMPLICATED BY HYPOKALEMIA AND URINARY TRACT INFECTION: A CASE REPORT

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ABSTRACT
Relapse in adult nephrotic syndrome represents a clinically complex phase characterized not only by recurrent proteinuria but also by heightened vulnerability to metabolic and infectious complications. A 24-year-old male with a prior history of nephrotic syndrome presented with generalized edema, foamy urine, and dysuria. Urinalysis showed 3+ proteinuria, while laboratory evaluation revealed severe hypoalbuminemia (1.1 g/dL) and hypokalemia (2.9 mmol/L) with bacteriuria. A diagnosis of relapsing nephrotic syndrome complicated by moderate hypokalemia and urinary tract infection was established. The patient received step-down intravenous methylprednisolone, ceftriaxone, 20% albumin infusion, combined furosemide–spironolactone therapy, and cautious intravenous potassium replacement under continuous ECG monitoring. Within seven days, edema improved significantly and serum potassium normalized without complications. Infection-triggered relapse may worsen electrolyte imbalance through urinary losses and diuretic therapy. Early identification of hypokalemia and infection is essential to prevent cardiac and renal complications. Coordinated pharmacologic management allows safe correction while maintaining fluid balance. Prompt recognition and multidisciplinary management of relapse-associated infection and electrolyte disturbance are critical to improving outcomes in adult nephrotic syndrome.

Keywords: hypokalemia; relapsing nephrotic syndrome; urinary tract infection

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INTRODUCTION

Nephrotic syndrome (NS) is a clinical glomerular disorder characterized by massive proteinuria (≥ 3.5 g/24 hours), hypoalbuminemia, generalized edema, and hyperlipidemia resulting from increased permeability of the glomerular filtration barrier to plasma proteins (Tapia & Bashir, 2023). Although NS can occur at any age, adult presentations are most frequently associated with primary glomerular diseases such as minimal change disease, focal segmental glomerulosclerosis, and membranous nephropathy (Rovin et al., 2021). Nephrotic syndrome is an uncommon glomerular disorder, with an estimated annual incidence of approximately 2–7 cases per 100,000 population, although precise epidemiological data in adult populations are limited (Tapia & Bashir, 2023).

The pathophysiology of NS involves podocyte injury and disruption of the glomerular basement membrane, leading to excessive urinary protein loss. Resultant hypoalbuminemia reduces plasma oncotic pressure, promoting fluid translocation into the interstitial compartment and causing edema. Compensatory activation of the renin–angiotensin–aldosterone system further enhances sodium and water retention, exacerbating volume imbalance (Rovin et al., 2021; Tapia & Bashir, 2023). In addition to its hallmark manifestations, NS is associated with systemic complications including infection, electrolyte disturbances, dyslipidemia, and thromboembolic events (Busuioc & Mircescu, 2022).

Among infectious complications, urinary tract infection (UTI) is commonly observed in patients with nephrotic syndrome due to urinary loss of immunoglobulins and complement factors, which impair host defense mechanisms (Busuioc & Mircescu, 2022). Electrolyte disturbances such as hypokalemia may arise primarily from prolonged diuretic therapy or renal potassium loss related to tubular dysfunction in the context of treatment and secondary complications. Moderate hypokalemia can lead to significant neuromuscular weakness and potentially life-threatening cardiac arrhythmias (Castro & Sharma, 2025; Clase et al., 2020). Relapsing nephrotic syndrome represents an important clinical course characterized by recurrence of nephrotic-range proteinuria following remission. Relapse episodes are commonly precipitated by intercurrent infections or metabolic stress, creating a pathophysiological cycle in which inflammation, worsening glomerular permeability, and metabolic imbalance mutually intensify disease severity (Rovin et al., 2021). Recurrent relapses increase hospitalization risk and repeated exposure to immunosuppressive therapy, thereby heightening vulnerability to secondary complications (Busuioc & Mircescu, 2022).

The coexistence of severe electrolyte imbalance and infection during relapse presents a substantial therapeutic challenge. Hypokalemia may compromise renal tubular and cardiovascular stability, while persistent infection perpetuates inflammatory glomerular injury and protein loss. These interacting mechanisms can delay recovery, complicate fluid management, and increase the likelihood of adverse outcomes. Despite established clinical guidelines, reports describing integrated management of relapsing NS complicated simultaneously by urinary tract infection and moderate hypokalemia remain limited, particularly in young adult populations (Busuioc & Mircescu, 2022).

METHOD

This case report describes a young adult with relapsing nephrotic syndrome complicated by urinary tract infection and moderate hypokalemia. The case highlights the interconnected mechanisms underlying these complications and emphasizes the importance of early recognition and multidisciplinary management to optimize clinical outcomes.

RESULT

A 24-year-old man presented to the emergency department with progressive generalized body swelling that had developed over one week. The edema was initially noted around the eyes upon waking and gradually extended to the lower limbs, abdomen, and upper extremities. The swelling was persistent and progressively worsening. He also reported frothy urine, dysuria, and a sensation of incomplete bladder emptying, without visible hematuria. He denied shortness of breath, cough, chest pain, abdominal pain, nausea, vomiting, or fever. The patient had a prior diagnosis of nephrotic syndrome earlier in the same year, achieved complete remission following corticosteroid therapy approximately six months prior to current presentation. There was no history of nephrotoxic drug exposure, herbal medication use, or recent respiratory infection.

On examination, the patient appeared moderately ill but was fully conscious and hemodynamically stable. Blood pressure was 150/90 mmHg, pulse rate 86 beats per minute, respiratory rate 18 breaths per minute, and temperature 36.5 °C. Periorbital edema was evident, accompanied by bilateral lower limb swelling and mild abdominal distension. Cardiorespiratory examination was unremarkable. The abdomen was soft with normal bowel sounds and no organomegaly. Generalized edema combined with frothy urine suggested recurrent nephrotic activity. Laboratory investigations demonstrated hemoglobin 14.5 g/dL, leukocyte count 8.67 × 10^3/µL, hematocrit 42.8%, platelet count 315 × 10^3/µL, blood urea nitrogen 51 mg/dL, serum creatinine 1.1 mg/dL, marked hypoalbuminemia of 1.1 g/dL, serum sodium 138 mmol/L, moderate hypokalemia of 2.9 mmol/L, and chloride 103 mmol/L. Lipid profile revealed total cholesterol 755 mg/dL, low-density lipoprotein cholesterol 590 mg/dL, triglycerides 181 mg/dL, and high-density lipoprotein cholesterol 64 mg/dL. Urinalysis showed turbid yellow urine with 3+ proteinuria, leukocyturia, and

bacteriuria, consistent with urinary tract infection. Renal ultrasonography demonstrated normal renal size and parenchymal appearance without obstruction or structural abnormality. Urine culture was not performed due to resource limitations; empirical therapy was initiated based on clinical and urinalysis findings. Based on clinical course, the patient was classified as infrequently relapsing steroid-sensitive nephrotic syndrome. Concurrent urinary findings supported infection, while moderate hypokalemia was attributed to renal potassium loss and diuretic exposure, posing potential neuromuscular and cardiac risk.

Initial management included intravenous 0.9% sodium chloride administered at a controlled infusion rate. Empirical antimicrobial therapy consisted of ceftriaxone 1 gram intravenously twice daily for seven days. Immunosuppressive therapy was initiated with intravenous methylprednisolone 62.5 mg twice daily for three days, followed by gradual tapering and transition to oral corticosteroid therapy over eight weeks. Volume management included infusion of 20% human albumin (three vials total), combined diuretic therapy with furosemide 40 mg orally once daily and spironolactone 50 mg orally twice daily, and renin–angiotensin system blockade using captopril 25 mg orally twice daily. Gastrointestinal protection was provided with omeprazole 40 mg intravenously twice daily, while ondansetron 4 mg intravenously three times daily was administered for symptomatic relief. Moderate hypokalemia was corrected using intravenous potassium chloride 50 mEq infused at a controlled rate under continuous electrocardiographic monitoring with serial electrolyte reassessment.

During seven days of hospitalization, close monitoring of fluid balance, urine output, blood pressure, and electrolyte levels was maintained. Edema progressively resolved, abdominal distension subsided, and body weight decreased. Repeat laboratory testing demonstrated improvement of serum potassium to 3.7 mmol/L, increased serum albumin to 1.7 g/dL, and resolution of urinary infection markers. The patient was discharged in stable condition with recommendations for sodium restriction, adequate protein intake within tolerance, and continuation of tapering oral corticosteroids, antihypertensive therapy, and combined diuretics under outpatient nephrology follow-up.

DISCUSSION

Nephrotic syndrome (NS) is defined by heavy proteinuria (≥3.5 g/day), hypoalbuminemia, edema, and frequently hyperlipidemia resulting from disruption of the glomerular filtration barrier (Rovin et al., 2021). In adults with previously responsive disease, relapse is characterized by recurrence of nephrotic-range proteinuria after remission. According to KDIGO 2021, relapse frequency is categorized based on the number and timing of recurrences, and a relapse occurring more than six months after remission is generally considered an infrequent relapse rather than frequently relapsing disease (Rovin et al., 2021). In this case, the recurrence of significant proteinuria after a remission period exceeding six months fulfills criteria for an infrequent relapse.

Although relapse is well described in pediatric populations, relapse in adults often presents with greater clinical complexity due to higher cardiometabolic burden, infection susceptibility, and exposure to long-term immunosuppression (Faucon et al., 2025). This distinction is clinically relevant, as complications during adult relapse may significantly influence short-term morbidity and therapeutic strategy. The diagnosis of relapse was supported by the reappearance of marked proteinuria (+3 on dipstick urinalysis) in a patient with a documented history of nephrotic syndrome. Although urine dipstick testing is a useful screening modality, KDIGO 2021 recommends quantitative assessment using either a spot urine protein–creatinine ratio (UPCR) or 24-hour urine protein collection to more accurately define relapse and monitor therapeutic response (Rovin et al., 2021). Therefore, the absence of quantitative protein measurement represents a limitation in diagnostic precision and follow-up evaluation.

At the pathophysiological level, relapse reflects recurrent podocyte injury leading to increased glomerular permeability to plasma proteins. Structural alterations of the slit diaphragm and cytoskeletal dysregulation of podocytes including abnormalities involving nephrin- and podocin-associated signaling pathways are central mechanisms in the development of proteinuria (Fratila et al., 2024; Garg, 2018). Persistent proteinuria further aggravates renal injury through tubular toxicity and activation of the intrarenal renin–angiotensin–aldosterone system (RAAS), promoting sodium retention and intraglomerular hypertension (Fratila et al., 2024). These mechanisms plausibly explain the development of hypertension during relapse in this patient.

Hypokalemia was identified as a significant metabolic complication. In nephrotic syndrome, hypokalemia often arises from renal potassium losses enhanced by loop diuretic therapy and secondary hyperaldosteronism, which increase urinary potassium excretion by altering sodium delivery to the distal nephron and promoting kaliuresis (Chang et al., 2023). Clinically significant hypokalemia increases the risk of ventricular arrhythmias, muscle weakness, and characteristic electrocardiographic changes, necessitating appropriate correction. Importantly, in this patient, hypokalemia likely resulted from a synergistic interplay between relapse-associated RAAS activation and loop diuretic therapy. Severe hypoalbuminemia may lead to effective arterial underfilling, triggering secondary hyperaldosteronism. Concurrent loop diuretic administration increases distal sodium delivery, thereby enhancing aldosterone-mediated potassium secretion in the collecting duct. This RAAS–diuretic synergy provides a coherent mechanistic explanation for potassium wasting during relapse (Clase et al., 2020; Culver et al., 2024).

Potassium chloride (KCl) is the preferred formulation for potassium replacement due to its effectiveness in restoring both potassium and accompanying chloride deficits (McMahon & Bashir, 2023). In severe or symptomatic hypokalemia, intravenous administration with continuous ECG monitoring and controlled infusion rates is recommended to rapidly correct serum potassium while minimizing the risk of rebound hyperkalemia (Castro & Sharma, 2025). Intravenous administration is indicated in severe or symptomatic cases and requires careful infusion rate control and cardiac monitoring. In addition to potassium supplementation, evaluation and modification of precipitating factors such as adjustment of diuretic therapy are essential to prevent recurrence. The management approach applied in this patient is therefore consistent with established principles of electrolyte correction in kidney disease. In selected patients with severe hypoalbuminemia and refractory edema, intravenous albumin infusion combined with loop diuretics may enhance diuretic responsiveness by expanding intravascular oncotic pressure and improving renal perfusion (Kardalas et al., 2018; Rovin et al., 2021). In this case, administration of 20% human albumin alongside diuretic therapy was intended to optimize effective circulating volume and augment natriuresis. The addition of spironolactone, an aldosterone antagonist, may further counteract secondary hyperaldosteronism and mitigate potassium loss, particularly in patients receiving loop diuretics (Patibandla et al., 2023; Rovin et al., 2021).

Urinary tract infection (UTI) was managed with ceftriaxone therapy. Patients with nephrotic syndrome are predisposed to infection due to urinary loss of immunoglobulins and complement components, impaired opsonization, and potential exposure to immunosuppressive therapy. Infection is not only a recognized complication but may also serve as a trigger for relapse in glomerular disease (Kopp et al., 2020). The coexistence of relapse and UTI in this case suggests a bidirectional interaction, in which infection may amplify immune activation and cytokine-mediated podocyte injury, thereby sustaining proteinuria, while nephrotic-associated immune dysfunction increases infection susceptibility. Recognition of this interaction is crucial, as prompt infection control may contribute not only to resolution of systemic inflammation but also to stabilization of glomerular permeability (Rovin et al., 2021).

Ceftriaxone, a third-generation cephalosporin with broad Gram-negative coverage, is commonly used in hospitalized patients with moderate to severe UTI requiring parenteral therapy. Its pharmacokinetic profile permits once-daily dosing and provides adequate urinary and systemic antibacterial activity. Although empirical therapy may be clinically justified in symptomatic patients, microbiological confirmation is recommended whenever feasible to enable targeted treatment and antimicrobial stewardship (EAU Guidelines Office, 2023). Effective infection control is important not only for resolving the acute complication but also for eliminating inflammatory stimuli that may perpetuate podocyte injury and sustain proteinuria. Corticosteroids remain the first-line therapy for steroid-sensitive nephrotic relapse in adults according to KDIGO 2021 (Rovin et al., 2021). Glucocorticoids exert immunomodulatory effects, reduce inflammatory cytokine activity, and contribute to stabilization of podocyte structure, thereby decreasing proteinuria. In adult minimal change disease the most common steroid-responsive etiology—remission rates are high, although relapse is common. Re-initiation of corticosteroid therapy in this patient is therefore aligned with current evidence-based recommendations (Azukaitis et al., 2022).

Supportive therapy plays a crucial role in relapse management. RAAS blockade using angiotensin-converting enzyme inhibitors (ACE inhibitors) or angiotensin receptor blockers (ARBs) reduces intraglomerular pressure and proteinuria independent of systemic blood pressure reduction, thereby slowing glomerular injury progression (KDIGO Blood Pressure Work Group, 2021). Blood pressure control is a fundamental component of nephrotic syndrome management in adults. Diuretics are indicated for symptomatic control of edema through natriuresis and extracellular volume reduction; however, excessive diuresis may precipitate electrolyte disturbances, including hypokalemia, necessitating careful titration and laboratory monitoring(Ellison & Felker, 2017). In addition, evaluation of thromboembolic risk and management of dyslipidemia should be considered as part of comprehensive supportive therapy in nephrotic syndrome (Rovin et al., 2021).

Long-term outcomes vary according to histopathological subtype. Adult minimal change disease typically demonstrates high steroid responsiveness with favorable renal prognosis, whereas focal segmental glomerulosclerosis carries a higher risk of progression to chronic kidney disease (Azukaitis et al., 2022). In the absence of kidney biopsy, precise etiological classification in this case cannot be established. Therefore, conclusions regarding underlying histopathology must remain cautious, although the clinical course is compatible with steroid-sensitive disease. Overall, the therapeutic strategy implemented in this case is consistent with contemporary guideline-based management. Corticosteroids target immune-mediated podocyte injury; RAAS blockade reduces proteinuria and intraglomerular hypertension; diuretics provide symptomatic edema relief; potassium chloride corrects moderate hypokalemia; and ceftriaxone addresses infectious complications. Interpretation of this case should acknowledge the absence of quantitative proteinuria measurement and histopathological confirmation. This report underscores the importance of standardized monitoring, prompt complication management, and comprehensive supportive therapy in adult nephrotic syndrome relapse.

CONCLUSION

This case underscores that adult relapse of steroid-sensitive nephrotic syndrome may be complicated by moderate hypokalemia and infection, both requiring urgent recognition. Recurrent nephrotic-range proteinuria, marked hypoalbuminemia, potassium 2.9 mmol/L, and bacteriuria confirmed relapse with metabolic and infectious complications. Clinical stabilization was achieved through prompt corticosteroid re-initiation, renin–angiotensin system blockade, albumin-assisted diuresis, intravenous potassium replacement under ECG monitoring, and targeted antimicrobial therapy. For clinicians, early identification of relapse triggers and severe electrolyte disturbances, followed by integrated and carefully monitored management, is essential to prevent morbidity and optimize short-term outcomes in adult nephrotic syndrome.

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