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Case Report

Complete remission of severe nephrotic syndrome: a case report of outpatient therapy

Kadek Nova Adi Putra*

Department of Internal Medicine, Wangaya Public Hospital, Denpasar, Bali, Indonesia

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***Correspondence:**
Dr. Kadek Nova Adi Putra,
E-mail: novaadie27@gmail.com

ABSTRACT

Nephrotic syndrome is characterized by massive proteinuria, hypoalbuminemia, generalized edema, and hyperlipidemia, often resulting from primary glomerular diseases or secondary systemic conditions. Severe hypoalbuminemia and dyslipidemia can lead to life-threatening complications, necessitating timely and effective treatment. Here, this report presents a case of a 21-year-old male presenting with generalized edema, severe hypoalbuminemia (0.6 g/dl), and marked hyperlipidemia (LDL 501 mg/dl, triglycerides 401 mg/dl), consistent with nephrotic syndrome. Despite the recommendation for hospitalization, the patient opted for outpatient care. Management included high-dose oral corticosteroids, angiotensin-converting enzyme (ACE) inhibitors, dual lipid-lowering therapy (statin and fibrate), diuretics, nutritional support, and close biochemical monitoring. Over 24 weeks, the patient demonstrated complete clinical and laboratory remission. Proteinuria resolved by week 8, serum albumin normalized by week 16, and lipid profile returned to baseline by week 24. No significant complications were observed during follow-up. This case highlights the potential for successful outpatient management of severe nephrotic syndrome in a compliant and closely monitored patient. It underscores the importance of individualized care, adherence, and interdisciplinary collaboration in achieving favorable outcomes outside a hospital setting.

Keywords: Nephrotic syndome, Poteinuria, Albumin, Dislipidemia, Edema

INTRODUCTION

Nephrotic syndrome is a clinical constellation of signs and symptoms arising from disruption of the glomerular basement membrane, leading to massive proteinuria, hypoalbuminemia, generalized edema, and hyperlipidemia. This condition may result from primary kidney disorders (such as membranous nephropathy, minimal change nephropathy, or focal segmental glomerulosclerosis) or secondary causes, including systemic lupus erythematosus (SLE), congenital infections, diabetes mellitus, neoplasms, or the use of certain medications.[1]

Nephrotic syndrome can affect individuals of all ages but is more commonly observed in children. Annually, the incidence of nephrotic syndrome in adults is approximately 3 per 100,000 individuals.[2,3] The most common cause of nephrotic syndrome in adult patients is focal segmental glomerulosclerosis, whereas in children, the predominant cause is minimal change disease. Severe hypoalbuminemia and dyslipidemia can lead to various complications, such as pleural effusion (due to decreased oncotic pressure), arterial or venous thrombosis (as a result of urinary loss of anticoagulant proteins including antithrombin III, protein C, and protein S), and hypothyroidism (caused by the urinary loss of protein-bound thyroid hormones).[3]

This case report explores the successful outpatient management of a patient with severe hypoalbuminemia and hyperlipidemia secondary to nephrotic syndrome. The goal of this discussion is to review the therapeutic strategies, challenges, and outcomes involved in treating such a condition outside the hospital setting.

CASE REPORT

A 21-year-old male presented with complaints of generalized body swelling that had been progressively worsening over the past two months. The edema initially appeared in both lower extremities and subsequently spread to the eyelids, abdomen, and upper limbs. The patient reported a weight gain of 6 kilograms within this period. Associated symptoms included foamy urine for the past three months and pruritus, predominantly affecting the lower limbs.

The patient had a history of a similar episode at the age of 9, which resolved following several months of treatment. There was no significant family history of related illnesses. The patient was an active smoker, frequently consumed alcohol, and worked as a construction laborer having migrated from Kupang. On physical examination, vital signs were within normal limits. Notable findings included bilateral palpebral edema, pitting edema of both lower limbs, and minimal abdominal distension.

Laboratory investigations revealed: WBC 13.40×10³/µl, Hb 14.2 g/dl, HCT 38.4%, PLT 588 × 10³/µl; liver enzymes: SGOT 24 U/l, SGPT 31 U/l; renal function: urea 17 mg/dl, creatinine 0.9 mg/dl; electrolytes: sodium 138 mmol/l, potassium 4.2 mmol/l, chloride 95 mmol/l; serum albumin 0.6 g/dl. Urinalysis showed proteinuria +4 and hematuria +3. Lipid profile revealed LDL 501 mg/dl, triglycerides 401 mg/dl, and HDl 52 mg/dl.

The patient was advised to undergo a renal biopsy and inpatient care, but he refused and opted for outpatient management. A high-dose corticosteroid regimen was initiated with methylprednisolone 16 mg three times daily for two months, followed by a tapering schedule. Additional medications included captopril 12.5 mg twice daily, simvastatin 20 mg once daily, fenofibrate 300 mg once daily, furosemide 40 mg once daily, spironolactone 50 mg once daily, vitamin albumin three times daily, alongside a high-calorie, adequate-protein, and low-sodium diet.

Monitoring included serial evaluations of renal function, liver function, urinalysis, lipid profile, serum albumin, and complete blood counts at each follow-up visit. Clinically, the edema progressively improved, and by the eighth week, the patient achieved complete remission, prompting a gradual tapering of corticosteroids up to the 24th week. Serum albumin normalized by the 16th week, and lipid profile normalized by the 24th week. At the final follow-up, the patient was asymptomatic, with all laboratory parameters within normal limits.

Table 1: Laboratory investigations.

Blood analysis	Day 1	Week 4	Week 8	Week 12	Week 16	Week 24
Hemoglobin (g/dl)	14.2	14				
HCT (%)	38.4	37.9				
WBC (10^3/ul)	13.40	9.6				
Platelets (10^3/ul)	588	410				
SGOT (U/l)	24	36	35	37	37	37
SGPT (U/l)	31	33	34	35	36	36
BUN (mg/dl)	17	20	19	20	22	24
Serum creatinine (mg/dl)	0.9	0.9	0.8	1.0	1.0	0.9
Na/K/Cl	138/4.2/95	136/3.9/97	-ve	-ve	-ve	-ve
Proteinuria	+4	+1	-ve	-ve	-ve	-ve
Albumin (g/dl)	0.6	1.4	2.2	3.6		
Random blood sugar (mg/dl)	112	182	147	142	134	129
HDL (mg/dl)	43	40	41	43	42	43
LDL (mg/dl)	501	407	363	262	192	132
Triglyceride (mg/dl)	401	304	274	199	162	121

DISCUSSION

Nephrotic syndrome is a clinical syndrome characterized by massive proteinuria leading to hypoalbuminemia, hyperlipidemia, edema, and various other complications.[1] The diagnostic criteria for nephrotic syndrome include proteinuria ≥3.5 g/day (+3 to +4 on dipstick), serum albumin ≤3.0 g/dl, edema, and dyslipidemia.[4] In the case described above, the patient presented with clinical signs and symptoms highly characteristic of nephrotic syndrome. Physical examination revealed edema involving nearly the entire body. Massive urinary protein loss resulted in a significant reduction of serum protein levels, particularly albumin, leading to decreased intravascular oncotic pressure and subsequent fluid shift into the interstitial space, causing edema. Hyperlipidemia occurred as a compensatory response by the liver to the reduced serum protein levels by increasing lipoprotein

synthesis, which in turn elevated blood lipid levels, especially low-density lipoprotein (LDL) and triglycerides.[1,5]

Albumin serves several essential functions within the body. One of its primary roles is maintaining oncotic pressure within the blood vessels, thereby preventing the extravasation of fluid into the interstitial tissues. Approximately 80% of the plasma colloid osmotic pressure is attributed to albumin. In addition to this, albumin acts as a carrier for a wide range of endogenous and exogenous substances, possessing a large transport capacity despite its relatively low affinity. It functions as a temporary storage medium and transport vehicle for these various compounds. Hypoalbuminemia can lead to several serious complications, including pleural effusion, ascites, and thromboembolic events.[6]

Complete blood count examination revealed thrombocytosis ($588\times10^3/\mu l$), raising suspicion of an early thromboembolic event in this patient. In nephrotic syndrome, glomerular damage results not only in albumin loss but also in the loss of essential hemostatic proteins, including antithrombin and protein S, both of which play critical roles in maintaining hemostatic balance. The depletion of these proteins disrupts the coagulation system's equilibrium, shifting the hemostatic balance toward a prothrombotic state, thereby increasing the tendency for thrombus formation. This prothrombotic condition substantially elevates the risk of thrombosis in patients with nephrotic syndrome.[7]

Urinalysis revealed severe proteinuria (+4), while serum albumin was markedly low at 0.6 g/dl. The lipid profile demonstrated significant elevations, with an LDL level of 401 mg/dl and triglycerides at 381 mg/dl. In contrast, renal and liver function tests remained within normal limits. This presentation is uncommon in adults, as extreme protein loss is typically accompanied by impaired renal function, ideally a renal biopsy should be performed as the gold standard to evaluate the underlying cause of nephrotic syndrome.[1,4,5] The patient was also advised to undergo inpatient treatment, with an initial plan including albumin transfusion, administration of diuretics, and corticosteroid therapy; however, the patient declined and opted for outpatient management.

The management of nephrotic syndrome includes the administration of high-dose corticosteroids, renin-angiotensin system (RAS) inhibitors, lipid-lowering agents, diuretics, and non-pharmacological interventions. Corticosteroids serve as immunosuppressants aimed at suppressing the inflammatory response within the glomeruli. The Kidney Disease: Improving Global Outcomes (KDIGO) guidelines recommend the use of corticosteroids (prednisone or prednisolone) to achieve remission in patients with nephrotic syndrome, at a dose of 1 mg/kg per day (maximum 80 mg), with a treatment duration ranging from a minimum of 4 weeks to a maximum of 16 weeks during the initial phase, followed by a tapering regimen extending up to 24 weeks. Once remission is achieved, KDIGO advises reducing the glucocorticoid dose to 5–10 mg/week or less, with a total exposure period of at least 24 weeks. Corticosteroid therapy has been shown to achieve complete remission in approximately 80% of adult patients with nephrotic syndrome.[4] In this patient, oral methylprednisolone was administered at a dose of 16 mg three times daily during the initial 8 weeks, and following the achievement of remission, the dose was gradually tapered over a 6-month period.

Renin-angiotensin system (RAS) inhibitors, including angiotensin-converting enzyme inhibitors (ACEIs) and angiotensin receptor blockers (ARBs), are the antiproteinuric agents of choice in nephrotic syndrome. ACEIs and ARBs can reduce proteinuria by approximately 40–50%, depending on the dosage, particularly when patients adhere to dietary sodium restriction. RAS inhibitors exert their effect by modulating the action of angiotensin II. ACEIs inhibit the conversion of angiotensin I to angiotensin II, while ARBs block angiotensin II receptors. Angiotensin II induces constriction of the efferent arteriole, thereby increasing glomerular capillary pressure and promoting excessive protein filtration into the urine. By reducing the production and blocking the effects of angiotensin II, RAS inhibitors lower intraglomerular pressure and consequently diminish protein leakage into the urine. There is currently insufficient evidence to support the superiority of ACEIs over ARBs, or vice versa, in this regard. Although combining ACEIs and ARBs may provide additive antiproteinuric effects, this strategy carries a heightened risk of acute kidney injury (AKI) and hyperkalemia.[4] Furosemide was administered to manage edema, and in combination with spironolactone, it was expected to reduce the risk of hypokalemia.

Hyperlipidaemia is also known to worsen the progression of glomerular damage, partly due to the rapid development of atherosclerosis in the kidney's blood vessels. In this case, the administration of dual antilipidemic therapy (a statin and a fibrate) was indicated due to the patient's markedly elevated LDL and triglyceride levels, as well as to prevent further glomerular injury. This combination was considered particularly because the patient declined inpatient care, with the aim of achieving a more rapid reduction in lipid levels and thereby minimizing the risk of glomerular damage. Prolonged dyslipidemia in nephrotic syndrome is also a recognized risk factor for various complications, including accelerated atherosclerosis, myocardial infarction, stroke, chronic kidney disease, and thrombosis.8 The administration of dual antilipidemic therapy has been shown to effectively correct lipid profiles; however, regular monitoring of liver function is necessary, as the combination of statins and fibrates increases the risk of liver injury.[9]

The patient was also prescribed oral vit-albumin supplementation and advised to follow a high-calorie, adequate-protein, and low-sodium diet. A high-calorie,

adequate-protein diet (0.8 g/kg body weight per day) has been shown to reduce urinary protein excretion without causing malnutrition in patients.[4]

A low-sodium, adequate-protein diet is recommended to help manage edema and prevent worsening hypoalbuminemia, although this must be balanced against the need to minimize proteinuria.

The patient demonstrated a good response to the prescribed therapy, achieving complete remission by the second month of treatment. Significant clinical improvement was observed after the initiation of outpatient management. Serum albumin levels gradually returned to normal, and proteinuria decreased markedly. Hyperlipidemia also improved with the combination of statin and fibrate therapy, although routine monitoring was required for potential adverse effects, such as myalgia or liver injury. The patient's edema resolved, and no significant complications occurred during the follow-up period.

CONCLUSION

An important aspect of outpatient management is ensuring patient adherence to the prescribed treatment regimen. The patient received detailed counselling regarding the importance of medication compliance, dietary restrictions, and the necessity of regular follow-up visits. Although outpatient management in this case was successful, it also carried inherent risks.

In the presence of severe hypoalbuminemia, the patient was at risk of developing complications such as pleural effusion or thrombosis, which would require prompt intervention but could be delayed due to the patient's refusal of inpatient care. Additionally, the risk of infection associated with high-dose immunosuppressive therapy needed to be carefully monitored and managed.

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REFERENCES

1. Tapia C, Bashir K. Nephrotic Syndrome. 2023. Treasure Island (FL): StatPearls Publishing. 2025.
2. Maslachah A, Pramudya D, Pranawa, Widodo, Mardiana N, Tjempakasari A. Inadequate treatment in a patient with minimal change disease (MCD) nephrotic syndrome: a case report. Bali Med J. 2023;12(2):2026-9.
3. Hull RP, Goldsmith DJ. Nephrotic syndrome in adults. BMJ. 2008;336(7654):1185-9.
4. Kidney Disease: Improving Global Outcomes (KDIGO) Glomerular Diseases Work Group. KDIGO 2021 Clinical Practice Guideline for the Management of Glomerular Diseases. Kidney Int. 2021;100(4S):S1-6.
5. Siddall EC, Radhakrishnan J. The pathophysiology of edema formation in the nephrotic syndrome. Kidney Int. 2012;82(6):635-42.
6. Gounden V, Vashisht R, Jialal I. Hypoalbuminemia.: StatPearls Publishing. 2023. Treasure Island (FL): StatPearls Publishing. 2025.
7. Kerlin BA, Ayoob R, Smoyer WE. Epidemiology and Pathophysiology of Nephrotic Syndrome–Associated Thromboembolic Disease. Clin J Am Society Nephrol. 2012;7(3):513-20.
8. Agrawal S, Zaritsky JJ, Fornoni A, Smoyer WE. Dyslipidaemia in nephrotic syndrome: mechanisms and treatment. Nat Rev Nephrol. 2018;14(1):57-70.
9. Sobukawa Y, Hatta T, Funaki D, Nakatani E. Safety of Combined Statin and Fibrate Therapy: Risks of Liver Injury and Acute Kidney Injury in a Cohort Study from the Shizuoka Kokuho Database. Drugs Real World Outcomes. 2024;11(2):317-30.

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