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Complete Remission of Focal Segmental Glomerulosclerosis (FSGS) with Multiple Complications: A Case Report

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ABSTRACT

Background: The symptoms of nephrotic syndrome include severe proteinuria, hypoalbuminemia, hyperlipidemia, and edema. Renal biopsy is essential to identify the histological subtype, which guides therapy and prognosis. Early diagnosis and treatment are crucial to prevent the progression of kidney failure. We present a case of nephrotic syndrome secondary to focal segmental glomerulosclerosis (FSGS), complicated by severe anemia, hypercoagulability, and pressure ulcers.

Case Presentation: A 25-year-old woman presented with progressive generalized edema for four months, which ultimately restricted her mobility and resulted in immobilization and a prolonged bedridden state. She reported a decrease in urine output and foamy urine for two months. On admission, vital signs were stable. Physical examination revealed periorbital edema, pale conjunctiva, abdominal distension with striae and shifting dullness, non-pitting edema in both lower extremities and the left upper extremity, and a gluteal wound with erythematous macules. Laboratory findings revealed severe normocytic normochromic anemia, hypoalbuminemia, hypercholesterolemia, and nephrotic-range proteinuria. Peripheral smear demonstrated anisopoikilocytosis, leucocytosis with immature granulocytes, and giant platelets. The direct Coombs test was positive and the d-dimer level was increased. Echocardiography revealed mild pericardial effusion, while abdominal ultrasound demonstrated gallbladder sludge with free peritoneal fluid. Doppler ultrasound of the left upper extremity showed no abnormalities, with no evidence of venous thrombus. Renal biopsy confirmed **FSGS.** She was diagnosed with **nephrotic syndrome due to FSGS, complicated by severe anemia with a positive direct Coombs test,** hypercoagulability, and pressure ulcers. Clinical improvement was achieved through the use of high dose corticosteroids, immunosuppressive therapy, and antihypertensive agents. Complete remission was achieved within six weeks.

Conclusion: This case highlights the complexity of nephrotic syndrome caused by FSGS, where timely biopsy, immunosuppressive therapy, and supportive care were key to achieving remission and improving outcomes.

Keywords: *Nephrotic syndrome; Glomerulosclerosis, Focal Segmental; Nephrology*

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1. INTRODUCTION

A glomerular condition known as focal segmental glomerulosclerosis (FSGS) is characterized by segmental scarring that affects only a portion of the glomerular tuft and usually affects a fraction of glomeruli in renal biopsy specimens (1). It can be classified into primary, secondary, genetic, and undetermined forms, distinguished by their underlying etiology and clinical implication. Primary disease is often linked to circulating permeability factors, while secondary FSGS may develop in association with other conditions such as diabetes mellitus, systemic lupus erythematosus (SLE), malignancy, infections, or drug exposure. Estimating the true burden of FSGS is difficult because of variations in renal biopsy availability across regions. Reported incidence ranges from 0.2 to 1.8 per 100,000 population, and FSGS accounts for roughly 40% of nephrotic syndrome cases in adults. Around half of patients with persistent nephrotic-range proteinuria develop end-stage renal disease within three to eight years, and the disease is more common in men (2,3). Nephrotic syndrome is a clinical condition defined by heavy proteinuria exceeding 3.5 g per 24 hours, leading to hypoalbuminemia (<3 g/dL), but not necessarily accompanied by dyslipidemia, edema (4,5). The condition occurs when damage to the glomerular filtration barrier increases its permeability, leading to significant protein loss in the urine and predisposing patients to complications, including infections, thromboembolic events, hypovolemia, cardiovascular disorders, acute kidney injury, and anemia (5,6).

Despite advances in understanding FSGS, it remains a clinical challenge due to its heterogeneous presentation and various responses to therapy. Renal biopsy is crucial for confirming the diagnosis and guiding management and prognosis (4). Furthermore, complications such as severe anemia, hypercoagulability, and pressure ulcers may significantly affect outcomes but are often underrecognized. Here, we present a case of nephrotic syndrome caused by FSGS with multiple complications, highlighting both the diagnostic challenges and the need for prompt targeted therapy.

2. CASE PRESENTATION

A 25-year-old woman, came with complaint of generalized edema for four months before she was referred to the nephrology clinic at Dr. Soetomo General Hospital. The swelling affected her eyelids, more prominent in the morning and subsiding by midday. Over time, the edema gradually progressed from the feet to the thighs, becoming persistent and ultimately restricting her mobility, leading to prolonged bed rest for three months. Three months before admission, she developed a painful and itchy pressure ulcer in the gluteal area, along with intermittent fevers, progressive fatigue, and unintended weight gain. Her medical history revealed multiple hospitalizations over the past four months for suspected kidney infection, during which she received repeated albumin infusions and blood transfusions. She had no history of transfusion reactions and compatible blood was not difficult to obtain. Prior to the current admission, she had been taking oral methylprednisolone 32 mg twice daily, candesartan 8 mg once daily, furosemide 40 mg three times daily, and N-acetylcysteine 200 mg three times daily for four months. She denied having a history of autoimmune diseases, diabetes, heart disease, hypertension, or long-term usage of birth control. There was no family history of a similar illness. Her obstetric history included one miscarriage during her first pregnancy, followed by a spontaneous term delivery of a healthy child weighing 3.2 kg.

On examination, the patient appeared weak and compos mentis, with a body weight of 54 kg and a body mass index of 22.9 kg/m². The patient's vital signs were stable, and she reported a pain intensity of 5 on the Visual Analog Scale (VAS). She had pallor, periorbital edema, and generalized non-pitting edema involving both lower extremities and the left upper extremity. Striae were observed on the upper and lower extremities as well as the abdomen. A grade II pressure ulcer with granulation tissue and no necrotic slough was observed on the gluteal region. Cardiopulmonary examination was unremarkable, with normal heart sounds and vesicular breath sounds. Abdominal examination revealed shifting dullness, suggestive of ascites, without hepatosplenomegaly.

Initial laboratory investigations indicated severe normocytic, normochromic anemia with a hemoglobin level of 4.5 g/dL, leukocytosis with neutrophilia, and thrombocytosis. Due to the severe anemia, a Coombs test was performed, which demonstrated a positive direct result and a negative indirect result. Serum albumin was markedly reduced at 1.83 g/dL, with normal liver enzymes and bilirubin. Renal parameters showed elevated blood urea nitrogen (BUN) with preserved creatinine. Electrolytes were within normal limits. Inflammatory markers were elevated, including erythrocyte sedimentation rate (ESR), c-reactive protein (CRP) and procalcitonin suggesting a systemic infection. Coagulation studies were normal. Autoimmune markers (ANA, C3, C4) were normal, and viral serologies for hepatitis B, C, and HIV were negative. Lactate dehydrogenase (LDH) was mildly elevated. A peripheral blood smear revealed anisopoikilocytosis, leucocytosis with immature granulocytes, and giant platelets. The arterial blood gas had a pH of 7.49, indicating metabolic alkalosis. Urinalysis demonstrated heavy proteinuria (4+), trace hematuria, and protein to creatinine ratio (PCR) >300 mg/g. Nephrotic-range proteinuria was confirmed by a 24-hour urine collection at 3.36 g/day (Table 1). Imaging studies, including Chest X-ray showed cardiomegaly. A plain abdominal X-ray revealed prominent intestinal gas mixed with fecal material extending into the pelvic cavity, without any radio-opaque shadows along the urinary tract.

Table 1. Serial laboratory test of the patient

Lab Parameters (unit)	Result					
	Day 1	Day 2	Day 5	Day 8	Day 12	Reference value
Blood analysis						
Hemoglobin (g/dL)	4.5		9.9	9.7	10.4	11-14.7
Hematocrit (%)	15		31.5	31	32.4	35.2-46.7
Mean corpuscular volume (fL)	91.5		91	90.1	89	86.7-102.3
Mean corpuscular hemoglobin (pg)	27.3		28.6	28.2	28.6	27.1-32.4
Leukocytes (/µL)	13.360		7.67	8.67	15.72	11.3
Platelet (/µL)	686.000		265.000	198.000	393.000	150.000-450.000
Neutrophils (%)	65.9		66.6	71.5	62.9	39.8-70.5
Lymphocytes (%)	24.1		23.2	21.2	27.9	23.1-49.9
Erythrocyte Sedimenation Rate (mm/hr)	140				133	0-15
Peripheral blood smear	• **Erythrocytes:** Normochromic, normocytic with anisopoikilocytosis (including microcytes and ovalocytes), presence of polychromasia (+), no normoblasts observed. • **Leukocytes:** Appeared increased in number, predominantly segmented neutrophils; presence of immature granulocytes (+) (metamyelocytes) and atypical lymphocytes (+) (monocytoid); no blasts observed. • **Platelets:** Appeared increased in number, with giant platelets (+). **Interpretation:** Normochromic, normocytic anemia with anisopoikilocytosis; leukocytosis with immature granulocytes (+) and atypical lymphocytes (+); thrombocytosis					
Aspartate aminotransferase (U/L)	22		23	30		0-37
Alanine transaminase (U/L)	21		19	35		0-55
Total bilirubin (mg/dL)	0.45		0.29	0.32		0.2-1
Direct bilirubin (mg/dL)	0.13		0.13	0.09		<0.2
Blood urea nitrogen (mg/dL)	53		18	10		7-18
Serum creatinine (mg/dL)	0.87		0.51	0.5		0.6-1.3
Uric acid (mg/dL)	8					2.6-7.2
Albumin (g/dL)	1.83	2.09	2.12	2.22	2.57	3.4-5
Sodium (mmol/L)	144		148	142	142	135-144
Potassium (mmol/L)	4.1		3.4	3.9	3.5	2.5-5
Chloride (mmol/L)	108		106	103	104	98-107

Calcium corrected (mmol/L)	9.6				8.5-10.5
Random blood glucose (mg/dL)	103	99			<200
Hepatitis B surface antigen	Non- reactive				
HIV test	Non- reactive				
Lactate Dehydrogenase	IU/L	294			
Procalcitonin (ng/mL)	0.61		0.2		0.5-2 systemic inflammation
C-Reactive Protein (mg/dL)	0.5				0.3
ANA (mg/dL)	20.39				≥40
C3 (mg/dL)	135.14				90-207
C4 (mg/dL)	25.4				17.4-52.2
Direct Coombs Test	Positive				
Indirect Coombs Test	Negative				
High-Density Lipoprotein (mg/dL)		63		85	40-60
Low-Density Lipoprotein (mg/dL)		156	190	179	<100
Total Cholesterol (mg/dL)		269		319	<200
Triglycerides (mg/dL)		235		384	<150
Partial thromboplastin time (seconds)	10				11.5-15
Activated partial thromboplastin time (second)	24.6				28.6-42.2
Serum Iron (µg/dL)		34			35-150
Total Iron-Binding Capacity (µg/dL)		66			250-450
Ferritin (ng/mL)		2321.7			13-232
D-dimer (ng/m)		5.900	12.730	7.720	2.890	<500
Urine analysis					
pH	6.5			8	4.5-8
Protein	4+			2+	Negative
Leukocytes	Negative			Negative	Negative
Red blood cel	Trace			Negative	Negative
Glucose	Negative			Negative	Negative
Ketone	Negative			Negative	Negative
Nitrite	Negative			Negative	Negative
bilirubin	Negative			Negative	Negative
Protein to Creatinine Ratio (g/gCr)	>50			>50	<50
Albumin to Creatinine Ratio (mg/gCr)	>300			>300	<30

Protein excretion (g/24 h)		3.36	3.4	2.1
Protein concentration (g/L)		2.8	3	0.5
Urine volume (mL/24 h)		1200	1700	4200
Blood gas analysis				
pH	7.49			7.35-7.45
pO2 (mmHg)	103			80-100
pCO2 (mmHg)	42			35-45
HCO3 (mmol/L)	32			22-26
Base excess (mmol/L)	8.7			-2-2
SO2 (%)	98			94-100

Based on the patient's history, physical examination, and additional investigations, she was diagnosed with **nephrotic syndrome, severe normochromic normocytic anemia, suspected deep vein thrombosis (DVT) of the left upper extremity, cardiomegaly, and a grade II gluteal pressure ulcer, and** metabolic alkalosis. Initial management included bed rest, a 2100 kcal/day diet (0.8 g protein/kg body weight/day, sodium <2 g/day), fluid restriction, transfusion of packed red cells (1 bag daily), intravenous 20% albumin 100 mL over 4 hours, intravenous ceftriaxone 1 gram twice daily, intravenous furosemide 20 mg once daily, oral allopurinol 100 mg once daily, oral simvastatin 20 mg once daily, oral paracetamol 500 mg three times daily, candesartan 8 mg once daily, and oral spironolactone 25 mg once daily. Wound care was performed for the grade II pressure ulcer. The patient was advised to undergo blood test workups prior to receiving a blood transfusion (lipid profile, iron status, d dimer), abdominal ultrasound, echocardiography and doppler ultrasound of the left upper extremity.

On the second day of the hospitalization, the patient reported pain in the left hand and foot, with swelling noticeably reduced. The vital signs were stable and the body weighed 54 kg with a VAS pain score of 3. Laboratory tests showed hypoalbuminemia, dyslipidemia, low serum iron and Total Iron-Binding Capacity (TIBC), along with markedly elevated ferritin and D-dimer, suggesting anemia of chronic disease and a hypercoagulable state. Echocardiography revealed a mild pericardial effusion, while abdominal ultrasound demonstrated gallbladder sludge with free peritoneal fluid. Doppler ultrasound of the left upper extremity showed no abnormalities, with no evidence of venous thrombus. The patient's Anticoagulation and Risk Factors in Atrial Fibrillation (ATRIA) bleeding risk score was 3 (low risk). The Padua and IMPROVE VTE (venous thromboembolism) risk scores were both 3, indicating low risk of VTE and supporting consideration for pharmacologic prophylaxis. As part of the management, the patient received intravenous methylprednisolone 500 mg once daily for 3 days and subcutaneous **low molecular weight heparin** (LMWH) 0.4 mL every 12 hours, in addition to the previously prescribed medications.

After being admitted for five days, the patient's swelling had significantly improved, and she was able to sit upright comfortably. The vital signs were stable and the body weighed 52 kg with a VAS pain score of 3. Laboratory evaluation showed an increase in hemoglobin. Serum albumin remained low at 2.12 g/dL, and d-dimer levels were elevated, reflecting persistent hypercoagulability. Notably, 24-hour urinary protein excretion had risen to 3.4 g. The patient's intravenous methylprednisolone was gradually tapered to 62.5 mg once daily for 3 days, and oral cyclosporine 50 mg twice daily was newly initiated.

The patient was able to walk independently with minimal assistance on the twelfth day of the hospitalization. Vital signs were stable and body weight was 48 kg. She reported no pain (VAS 0). Laboratory tests showed an increase in hemoglobin to 10.4 g/dL and serum albumin to 2.57 g/dL, along with a decrease in d-dimer levels to 2.890 ng/mL. The lipid profile was elevated. Urinalysis revealed a reduction in proteinuria to +2, with 24-hour urinary protein excretion of 2.1 g. In view of these findings, a renal biopsy was subsequently planned to establish the underlying etiology and to guide further therapy and prognosis. The patient was planned for discharge with oral therapy consisting of methylprednisolone 16 mg twice daily and cyclosporine 50 mg twice daily.

At one month follow up after discharge, the patient was in good general condition and remained asymptomatic. Vital signs were stable with body weight was 45 kg. Laboratory tests showed hemoglobin 11.5 g/dL, stable renal function (BUN 18 mg/dL, creatinine 0.42 mg/dL), improved serum albumin (4 g/dL), and reduced proteinuria (24-hour urinary protein 0.2 g, PCR 0.3 g/gCr, albumin to creatinine ratio (ACR) >300 mg/gCr), despite persistently elevated lipid levels (total cholesterol 294 mg/dL, high-density lipoprotein (HDL) 85 mg/dL, low-density lipoprotein (LDL) 151 mg/dL, and triglycerides 255 mg/dL). Renal biopsy demonstrated 17 glomeruli, with two showing adhesions, one with sclerosis, and

mesangial cell proliferation in some glomeruli. Most tubules exhibited atrophy with mononuclear infiltration, and some showed thyroidization. Immunofluorescence was negative for IgA, IgM, IgG, C3, and C5. The biopsy was consistent with **FSGS with mesangioproliferative glomerulonephritis (Figure 1)**. The patient achieved complete remission and was transitioned to a maintenance regimen of methylprednisolone 16 mg once daily combined with cyclosporine 50 mg twice daily.

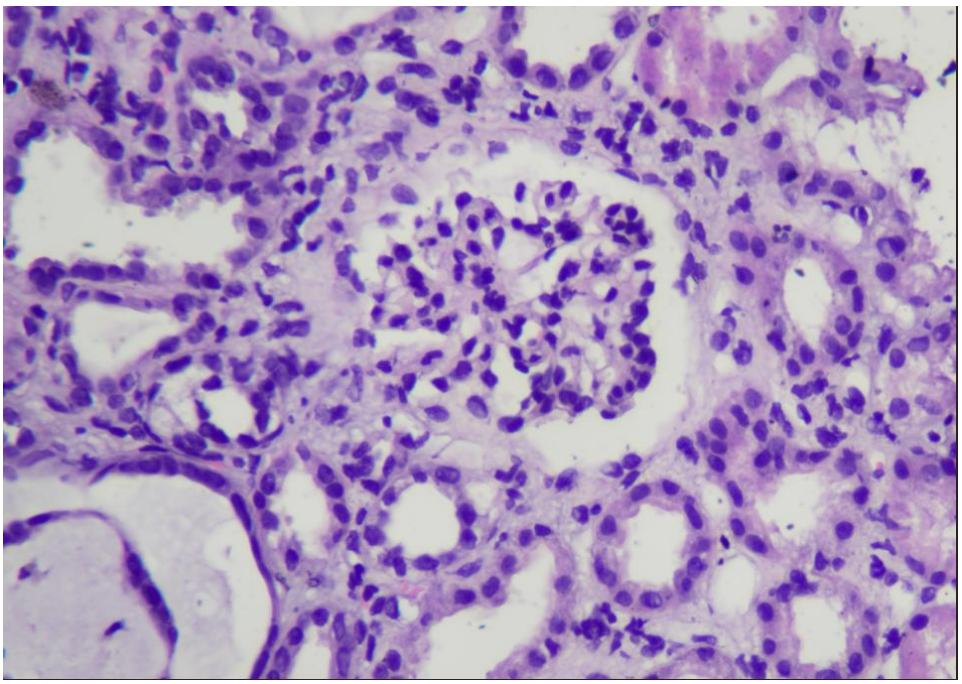

Figure 1. Renal biopsy showing segmental sclerosis with mesangial proliferation, tubular atrophy, and interstitial infiltration, consistent with focal segmental glomerulosclerosis.

The patient achieved complete remission within six weeks and was transitioned to a maintenance regimen of methylprednisolone 16 mg once daily combined with cyclosporine 50 mg twice daily. This approach was selected to maintain remission while reducing overall steroid exposure, with close monitoring for potential adverse effects from both medications.

3. DISCUSSION

Focal segmental glomerulosclerosis (FSGS) remains a challenging case of nephrotic syndrome in adults due to its heterogeneous presentation and various responses to therapy. This case is unique in that the patient not only developed multiple severe complications but also ultimately achieved complete remission. This case is notable for the presence of multiple severe complications followed by complete remission. The majority of complications are caused by massive urine protein losses, consequently controlling proteinuria is the most effective treatment strategy (7). This case illustrates several features that are rarely described in the literature. In this case, our patient is a young adult woman presented with progressive generalized edema, heavy proteinuria, and complications including severe anemia, a hypercoagulability state, and the occurrence of pressure ulcers, illustrating the systemic impact of nephrotic syndrome beyond the kidney.

The FSGS can be classified as primary, secondary, genetic, or undetermined origin. Secondary causes may be associated with systemic conditions such as diabetes mellitus, SLE, malignancy, medications, and infections (5). In this patient, blood glucose levels were within normal limits; autoimmune markers (ANA, C3, C4) were unremarkable; and viral serologies for hepatitis B, hepatitis C, and HIV were negative. Furthermore, there was no clinical evidence of malignancy, and the patient had no history of exposure to medications known to induce FSGS. A genetic form of FSGS was considered unlikely due to the adult onset of disease, absence of family history, and good response to immunosuppressive therapy. These findings reasonably exclude both secondary and genetic causes, thereby supporting a diagnosis of primary FSGS. This form of FSGS is often mediated by circulating permeability factors that break down the glomerular filtration barrier, resulting in proteinuria and a condition known as nephrotic syndrome (1,3,8,9). Among adults, FSGS accounts for around 40% of nephrotic syndrome cases. Estimating the true burden of FSGS is difficult because of variations in renal biopsy availability across regions. Reported incidence ranges from 0.2 to 1.8 per 100,000 population. Approximately half of individuals with persistent nephrotic-range proteinuria develop end-stage renal disease within three to eight years, and the illness is more common in men (2,3).

In adults, primary nephrotic syndrome due to glomerular diseases such as minimal change disease (MCD), membranous nephropathy (MN), or FSGS is often associated with anemia. Unfortunately, data on the true prevalence of anemia in nephrotic syndrome remain limited. Feinstein reported that nearly 59% of patients developed anemia, most often in those with steroid-resistant nephrotic syndrome, whereas Chenn observed a lower prevalence of 22.61% in primary nephrotic syndrome (10,11). Anemia in nephrotic syndrome with good renal function is complex and poorly understood. This anemia frequently occurs as a consequence of kidney fibroblasts producing less erythropoietin, which plays a central role in red blood cell formation. Erythropoietin is essential for stimulating red blood cell production in the bone marrow, regulating iron use, and supporting hemoglobin synthesis and reticulocyte release (12). The presence of severe anemia with low serum iron, decreased TIBC, and markedly elevated ferritin suggests anemia of chronic disease.

In our patient, the presence of a positive direct Coombs test suggested concern for possible immune-mediated hemolysis. However, it is well recognized that a positive result is not always associated with hemolysis, making careful clinical and biochemical evaluation essential. In this case, there was no splenomegaly, indirect bilirubin remained within normal limits, and LDH was only slightly elevated, suggesting that hemolysis was not clinically significant. Previous studies have shown that a positive direct Coombs test without hemolysis is not uncommon, particularly in patients with recent transfusion exposure. The reported frequency ranges from 1% to 15% among hospitalized patients without overt hemolysis or having a clear etiology. The significance of a positive direct Coombs test can only be understood in the full clinical context, taking into account the patient's history, medications, recent transfusions or transplants, and supporting laboratory data. In transfused patients, a positive direct Coombs test may represent the earliest signal of alloantibody formation, which can occur within two to three days after transfusion as part of a secondary or anamnestic immune response. In the present case, given the history of multiple transfusions and the absence of laboratory or clinical evidence of hemolysis, the positive result is most likely attributable to alloantibody development rather than ongoing hemolysis (13–15). Importantly, beyond immune-mediated phenomena, nephrotic syndrome itself predisposes patients to other hematologic complications, most notably a hypercoagulable state and an increased risk of thrombosis (16).

Although Doppler ultrasound of the left upper extremity excluded the presence of DVT, the patient's edema gradually improved after initiation of LMWH. This apparent paradox can be understood by considering several pathophysiological and clinical factors. Ultrasound imaging of the upper extremity may yield false-negative results, particularly in early or small thrombi or in veins that are anatomically difficult to visualize, such as the subclavian or axillary veins. Additionally, severe edema can hinder the visualization of veins due to thickening of subcutaneous tissue and altered blood flow, potentially reducing the sensitivity of Doppler studies (17–19). Therefore, a subclinical or developing thrombus cannot be entirely excluded. In nephrotic syndrome, patients frequently develop a pronounced hypercoagulable state, resulting from multiple contributing factors. These include a genetic predisposition, increased platelet number and activity, and localized activation of the coagulation system within the kidneys. However, the primary driver is generally considered to be the urinary loss of anticoagulant and profibrinolytic proteins, combined with compensatory hepatic overproduction of procoagulant factors such as fibrinogen and clotting factors V and VIII. Although significant urine loss of antithrombin III has been considered as a major contribution to the imbalance of pro- and anti-coagulant factors. Among these factors, serum albumin stands out as the most important independent predictor of thrombotic risk, especially when levels drop below 2 g/dL. Low albumin not only reduces oncotic pressure and promotes fluid retention and edema, but also concentrates coagulation factors (20,21).

Managing edema in nephrotic syndrome needs to be individualized, as uncontrolled swelling can quickly lead to serious problems such as difficulty moving, higher infection risk, and skin injury. In this context, pressure ulcers are an often-overlooked complication, especially when severe edema and prolonged immobility are present. These ulcers develop when constant pressure damages the skin and deeper tissues over bony areas. Recognizing them early and providing timely care is crucial to prevent progression into severe or even life-threatening conditions (22,23).

4. CONCLUSION

This case highlights the complexity of managing FSGS presenting with nephrotic syndrome in a young adult. Beyond heavy proteinuria and hypoalbuminemia, the patient developed significant complications, including severe anemia, hypercoagulability, and pressure ulcers, all of which contributed to her overall morbidity. Careful diagnostic workup, renal biopsy, and therapy with corticosteroids and cyclosporine led to remission and stabilization of her condition. The case underscores the importance of early recognition, comprehensive management, and close monitoring of both renal and systemic complications in order to improve outcomes for patients with FSGS.

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