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RELAPSE OF NEPHROTIC SYNDROME IN TIP-VARIANT FOCAL SEGMENTAL GLOMERULOSCLEROSIS AGAINST THE BACKGROUND OF ACTIVE PULMONARY TUBERCULOSIS: A CLINICAL CASE

Abstract. Focal segmental glomerulosclerosis is a heterogeneous morphological pattern of glomerular injury that may correspond to a primary, genetic, secondary, or undetermined process. The tip variant is frequently associated with nephrotic syndrome and a relatively better response to glucocorticosteroids; however, active infection substantially complicates both aetiological interpretation and the choice of pathogenetic therapy. The clinical significance of this issue is further reinforced by the high global burden of tuberculosis: according to the World Health Organization, approximately 10.7 million people developed tuberculosis worldwide in 2024. In such clinical situations, achieving a balance between the need to control the glomerular process and the risk of progression of the infectious disease during immunosuppression becomes especially important. The aim of this paper was to present a clinical case of relapse of nephrotic syndrome in a young woman with morphologically confirmed tip-variant focal segmental glomerulosclerosis against the background of active disseminated pulmonary tuberculosis and to analyse the diagnostic and therapeutic challenges.

In this 28-year-old patient, tip-variant focal segmental glomerulosclerosis was verified following kidney biopsy in 2024. In 2025, a relapse of nephrotic syndrome developed, with daily proteinuria of 5.25-6.3 g/day, marked hypoproteinaemia, hypoalbuminaemia, dyslipidaemia, oedema, ascites, and hydrothorax, while renal function remained preserved. At the same time, active disseminated pulmonary tuberculosis was diagnosed, which required a combination of anti-tuberculosis therapy with carefully balanced use of glucocorticosteroids, cyclophosphamide, nephroprotective treatment, and antithrombotic support. In this case, clinical severity was determined not by a reduction in glomerular filtration rate, but by the activity of the nephrotic syndrome and the hypercoagulable profile. This clinical case highlights the need for multidisciplinary management, individualisation of decisions regarding immunosuppression in the presence of active infection, and a focus on the dynamics of proteinuria, albumin levels, oedematous syndrome, and thrombotic risk rather than solely on creatinine values or glomerular filtration rate.

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Keywords: focal segmental glomerulosclerosis; tip variant; nephrotic syndrome; tuberculosis; immunosuppression; proteinuria; thrombotic risk; clinical case.

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РЕЦИДИВ НЕФРОТИЧНОГО СИНДРОМУ ПРИ ФОКАЛЬНО-СЕГМЕНТАРНОМУ ГЛОМЕРУЛОСКЛЕРОЗІ TIP-ВАРІАНТУ НА ТЛІ АКТИВНОГО ТУБЕРКУЛЬОЗУ ЛЕГЕНЬ: КЛІНІЧНИЙ ВИПАДОК

Анотація. Фокально-сегментарний гломерулосклероз є гетерогенним морфологічним патерном ушкодження клубочків, який може відповідати первинному, генетичному, вторинному або невизначеному процесу. Tip-варіант часто асоціюється з нефротичним синдромом і відносно кращою відповіддю на глюкокортикостероїди, однак активна інфекція істотно ускладнює як етіологічну інтерпретацію, так і вибір патогенетичної терапії. Додаткову клінічну вагу проблемі надає високе глобальне поширення туберкульозу: за даними Всесвітньої організації охорони здоров'я, у 2024 році у світі захворіли на туберкульоз близько 10,7 млн осіб. У таких клінічних ситуаціях особливого значення набуває баланс між необхідністю контролю гломерулярного процесу та ризиком прогресування інфекційного захворювання на тлі імуносупресії. Мета роботи – представити клінічний випадок рецидиву нефротичного синдрому у молодої жінки з морфологічно підтвердженим фокально-сегментарним гломерулосклерозом tip-варіанту на тлі активного дисемінованого туберкульозу легень і проаналізувати діагностичні та терапевтичні виклики. У 28-річної пацієнтки після нефробіопсії у 2024 році було верифіковано фокально-сегментарний гломерулосклероз tip-варіанту. У 2025 році розвинувся рецидив нефротичного синдрому з добовою протеїнурією 5,25-6,3 г/добу, вираженою гіпопротеїнемією, гіпоальбумінемією, дисліпідемією, набряками, асцитом і гідротораксом при збереженій функції нирок. Паралельно було встановлено діагноз активного дисемінованого туберкульозу легень, що потребувало поєднання протитуберкульозної терапії з ретельно зваженим застосуванням глюкокортикостероїдів, циклофосфаміду, нефропротективного та антитромботичного супроводу.

Клінічна тяжкість у цьому випадку визначалася не зниженням швидкості клубочкової фільтрації, а активністю нефротичного синдрому і гіперкоагуляційним профілем. Представлений клінічний випадок підкреслює необхідність мультидисциплінарного ведення, індивідуалізації рішень щодо імуносупресії при активній інфекції та орієнтації на динаміку протеїнурії, альбуміну, набрякового синдрому і тромботичного ризику, а не лише на показники креатиніну або швидкості клубочкової фільтрації.

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Ключові слова. фокально-сегментарний гломерулосклероз; tip-варіант; нефротичний синдром; туберкульоз; імуносупресія; протеїнурія; тромботичний ризик; клінічний випадок.

Introduction. Focal segmental glomerulosclerosis is regarded not as a distinct nosological entity, but as a morphological pattern of glomerular injury arising from various mechanisms of podocyte damage. The modern approach requires an aetiological distinction between primary, genetic, secondary, and undetermined focal segmental glomerulosclerosis, since it is the clinical and pathogenetic context that determines treatment strategy, the appropriateness of immunosuppression, and prognosis [1, 2, 3].

Within the Columbia classification, the tip variant has traditionally been associated with nephrotic syndrome and a comparatively higher likelihood of proteinuria remission; however, it should not be interpreted as an unconditionally benign histological variant.

Cohort studies indicate that prognostic significance depends not only on the morphological subtype, but primarily on the achievement of partial or complete remission of proteinuria [4, 7–9].

The coexistence of glomerular injury with active tuberculosis represents a separate clinical challenge. According to the findings of a recent review, a broad spectrum of glomerular lesions has been described in the setting of *Mycobacterium tuberculosis* infection, while the choice of immunosuppressive strategy under such circumstances remains difficult and requires an individualised approach [5]. At the same time, nephrotic syndrome itself is a state of high thrombotic risk, particularly when accompanied by massive proteinuria, severe hypoalbuminaemia, and laboratory signs of hypercoagulability [2, 6].

The clinical value of the present case lies in the fact that morphologically verified tip-variant focal segmental glomerulosclerosis with relapse of nephrotic syndrome occurred against the background of active disseminated pulmonary tuberculosis, creating a pronounced therapeutic dilemma between the need to control the glomerular process and the necessity to minimise the risks of immunosuppression in the setting of active infection.

Aim of the study. To present a clinical case of relapse of nephrotic syndrome in a patient with morphologically confirmed tip-variant focal segmental glomerulosclerosis against the background of active pulmonary tuberculosis and to discuss the diagnostic, therapeutic, and prognostic aspects of managing such a patient.

Clinical case. A 28-year-old woman was admitted to the nephrology department in December 2025 with complaints of oedema of the face, feet, and lower legs, general weakness, and fatigue. According to the discharge summary provided, the onset of the glomerular disease dated back to November 2023. In January 2024, following kidney biopsy, tip-variant focal segmental glomerulosclerosis was verified, after which glucocorticosteroid therapy was initiated.

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A worsening of the disease course was recorded from July 2025, when peripheral oedema recurred and proteinuria increased progressively. In August 2025, during a subsequent inpatient evaluation, disseminated pulmonary tuberculosis was diagnosed in addition to relapse of nephrotic syndrome. The patient underwent the intensive phase of specific anti-tuberculosis therapy followed by transition to the continuation phase. Because of persistence of nephrotic syndrome, methylprednisolone 32 mg/day was prescribed from 25 September 2025, and cyclophosphamide 50 mg twice daily was added from 28 October 2025.

During hospitalisation in December 2025, the patient's general condition was assessed as moderately severe. Her body weight was 45 kg and her height 165 cm, corresponding to a body mass index of approximately 16.5 kg/m². Facial puffiness, oedema of the feet and lower legs, and abdominal distension due to ascites were noted. Blood pressure was 115/70 mmHg, and heart rate was 74 beats per minute. On physical examination, vesicular breath sounds were present without wheeze or crackles, and the liver was palpable at the costal margin. Electrocardiography performed on 2 December 2025 showed sinus rhythm at a rate of 95 beats per minute and moderate myocardial changes. Imaging findings confirmed the systemic severity of nephrotic syndrome and the concomitant infectious process. According to ultrasonography of the abdominal organs and kidneys performed on 14 October 2025, free fluid was detected in the pelvis, together with left-sided hydrothorax and diffuse changes in the renal parenchyma. Computed tomography of the chest performed on 29 October 2025 without intravenous contrast demonstrated focal tuberculous lesions in both lungs and bilateral hydrothorax.

The laboratory profile was consistent with active nephrotic syndrome with preserved renal function. Daily urinary protein loss ranged from 5.25 to 6.3 g/day. Total serum protein decreased from 39.7 to 35.5 g/L. Marked hypoalbuminaemia was documented by the laboratory. Total cholesterol decreased from 19.9 to 10.9 mmol/L, while low-density lipoprotein cholesterol fell from 12.6 to 7.39 mmol/L, remaining substantially elevated. Serum creatinine fluctuated between 56.2 and 72.9 µmol/L, corresponding to chronic kidney disease stage G1 with preserved glomerular filtration rate. The coagulation profile demonstrated elevated fibrinogen and D-dimer levels, which, together with massive proteinuria and severe hypoproteinaemia, indicated a high risk of thrombotic complications.

During this hospitalisation, the patient received pulse therapy with methylprednisolone followed by oral methylprednisolone, cyclophosphamide, intravenous albumin in combination with a loop diuretic, nephroprotective therapy, correction of dyslipidaemia, calcium and vitamin D supplementation, as well as antithrombotic prophylaxis during pulse therapy. Anti-tuberculosis therapy was continued in accordance with the phthisiatrician's recommendations. After stabilisation of her condition, the patient was discharged for outpatient follow-up under the care of her general practitioner, nephrologist, and phthisiatrician, with recommendations for laboratory monitoring, assessment of daily proteinuria, and renal ultrasonography with Doppler examination.

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Table 1

Key stages of the disease course

Period	Event / clinical significance
November 2023	First inpatient hospitalisation due to glomerular kidney injury.
January 2024	Kidney biopsy: tip-variant focal segmental glomerulosclerosis; initiation of glucocorticosteroid therapy.
July 2025	Clinical deterioration with recurrence of oedematous syndrome and increasing proteinuria.
August 2025	Disseminated pulmonary tuberculosis diagnosed; specific anti-tuberculosis therapy initiated.
September–October 2025	Due to relapse of nephrotic syndrome, methylprednisolone 32 mg/day was prescribed, followed by addition of cyclophosphamide 50 mg twice daily.
1–12 December 2025	Hospitalisation with severe nephrotic syndrome, ascites, and hydrothorax despite preserved glomerular filtration rate; pulse therapy with methylprednisolone and adjustment of maintenance treatment were undertaken.

Table 2

Dynamics of key clinical and laboratory parameters during hospitalisation

Parameter	01 December 2025	05 December 2025	10 December 2025
Total serum protein, g/L	39.7	38.7	35.5
Serum creatinine, μmol/L	62.7	72.9	56.2
Total cholesterol, mmol/L	19.9	11.4	10.9
Low-density lipoprotein cholesterol, mmol/L	12.6	8.40	7.39
Daily urinary protein loss, g/day	5.25	–	6.3
D-dimer, laboratory value	–	4.23	–
D-dimer, laboratory value	–	–	3.0

Discussion. The present clinical case is noteworthy primarily because the clinical severity of the disease was determined not by a reduction in glomerular filtration rate, but by the activity of the nephrotic syndrome. In this patient with chronic kidney disease stage G1, massive proteinuria, progressive hypoproteinaemia, marked oedematous syndrome, ascites, hydrothorax, and significant dyslipidaemia persisted. This is consistent with the current understanding of focal segmental glomerulosclerosis as a clinicopathological syndrome in which treatment intensity should be determined not only by creatinine levels, but above all by the degree of podocyte injury, the level of proteinuria, and the presence of complications [2–4].

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The second important aspect concerns the differential diagnostic assessment. Although various patterns of glomerular injury have been described in *Mycobacterium tuberculosis* infection, in this case the kidney biopsy confirming tip-variant focal segmental glomerulosclerosis had been performed long before tuberculosis was detected. Therefore, interpreting the renal lesion as exclusively tuberculosis-associated would be an oversimplification. A far more justified conclusion is that active tuberculosis became a clinically significant modifying factor that increased the complexity of the course and narrowed the safe therapeutic corridor [5].

The third aspect is the therapeutic dilemma. The tip variant of focal segmental glomerulosclerosis is associated with comparatively better steroid responsiveness; however, the decision to continue glucocorticosteroids and add cyclophosphamide in the setting of active infection requires particularly careful balancing of expected benefit against infectious risks. In this case, the arguments in favour of a more active nephrological strategy included morphological verification of the process, a clinically significant relapse of nephrotic syndrome with fluid accumulation in body cavities, and preserved renal function, under which achieving remission of proteinuria remained a realistic clinical goal [2, 7, 8, 9].

The fourth aspect is thrombotic risk. In this patient, almost all adverse predictors were present simultaneously: massive proteinuria, marked hypoalbuminaemia, dyslipidaemia, elevated fibrinogen, increased D-dimer, and substantial limitation of physical activity due to the oedematous syndrome. For this reason, antithrombotic support in this clinical scenario appears pathogenetically justified and consistent with current views on thrombosis prevention in nephrotic syndrome [2, 6].

The practical value of this clinical case also lies in its multidisciplinary dimension. The nephrologist determines the strategy for controlling the glomerular process and adjusting immunosuppression; the phthisiatrician assesses the effectiveness and safety of anti-tuberculosis therapy and infectious risks; and the general practitioner/family doctor coordinates long-term outpatient follow-up, monitoring of laboratory parameters, treatment adherence, and timely detection of complications. For patients of this kind, such a model of coordination appears to be the most rational [9, 10].

A limitation of this clinical case is the absence in the discharge summary of a full morphological description of the kidney biopsy, detailed specification of the anti-tuberculosis regimen, and prolonged follow-up after discharge. In view of this, the article does not allow a definitive assessment of the long-term durability of the therapeutic response; however, it clearly illustrates the key clinical challenge, namely decision-making in the setting of conflict between active infection and the need to control immune-mediated podocyte injury.

Conclusions

1. Relapse of nephrotic syndrome in tip-variant focal segmental glomerulosclerosis may have a severe clinical course even in the presence of preserved glomerular filtration rate.

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2. Active pulmonary tuberculosis substantially complicates both the aetiological interpretation of glomerular injury and the choice of a safe immunosuppressive strategy; in such situations, medical history, kidney biopsy findings, and multidisciplinary management are of fundamental importance.

3. Management of such patients should be guided by the dynamics of proteinuria, albumin levels, oedematous syndrome, and thrombotic risk, rather than solely by creatinine values or glomerular filtration rate.

4. Long-term control of disease course and treatment safety requires coordinated work of the nephrologist, phthisiatrician, and general practitioner/family doctor.

References

1. World Health Organization. (2025). *Global tuberculosis report 2025*. Geneva: World Health Organization.

2. Kidney Disease: Improving Global Outcomes Glomerular Diseases Work Group. (2021). KDIGO 2021 clinical practice guideline for the management of glomerular diseases. *Kidney International, 100*(4 Suppl.), S1-S276. doi:10.1016/j.kint.2021.05.021

3. Bonilla, M., Efe, O., Selvaskandan, H., & Lerma, E.V. (2024). A review of focal segmental glomerulosclerosis classification with a focus on genetic associations. *Kidney Medicine, 6*, 100826. doi:10.1016/j.xkme.2024.100826

4. D'Agati, V.D., Kaskel, F.J., & Falk, R.J. (2011). Focal segmental glomerulosclerosis. *New England Journal of Medicine, 365*(25), 2398-2411. doi:10.1056/NEJMra1106556

5. Forster, A., Sabur, N.F., Iqbal, A., Vaughan, S., & Thomson, B. (2024). Glomerulonephritis during *Mycobacterium tuberculosis* infection: Scoping review. *BMC Nephrology, 25*, 285. doi:10.1186/s12882-024-03716-6

6. Lin, R., McDonald, G., Jolly, T., Batten, A., & Chacko, B. (2020). A systematic review of prophylactic anticoagulation in nephrotic syndrome. *Kidney International Reports, 5*(4), 435-447. doi:10.1016/j.ekir.2019.12.001

7. D'Agati, V.D., Alster, J.M., Jennette, J.C., Thomas, D.B., Pullman, J., Savino, D.A., et al. (2013). Association of histologic variants in focal segmental glomerulosclerosis clinical trial with presenting features and outcomes. *Clinical Journal of the American Society of Nephrology, 8*(3), 399-406. doi:10.2215/CJN.06100612

8. Kawaguchi, T., Imasawa, T., Kadomura, M., Kitamura, H., Maruyama, S., Ozeki, T., et al. (2022). Focal segmental glomerulosclerosis histologic variants and renal outcomes based on nephrotic syndrome, immunosuppression and proteinuria remission. *Nephrology Dialysis Transplantation, 37*(9), 1679-1690. doi:10.1093/ndt/gfab267

9. Trivedi, M., Pasari, A., Chowdhury, A.R., Abraham, A., & Pandey, R. (2015). Tip variant of focal segmental glomerulosclerosis: Is it truly a benign variant? *Renal Failure, 37*(5), 763-768. doi:10.3109/0886022X.2015.1014755

10. Vysochyna, I.L., Kramarchuk, V.V., & Yashkina, T.O. (2023). Impostors among family doctors. *Clinical and Preventive Medicine, 4*(26), 75-80. doi:10.31612/2616-4868.4(26).2023.11

Література

1. World Health Organization. (2025). *Global tuberculosis report 2025*. Geneva: World Health Organization.

2. Kidney Disease: Improving Global Outcomes Glomerular Diseases Work Group. (2021). KDIGO 2021 clinical practice guideline for the management of glomerular diseases. *Kidney International, 100*(4 Suppl.), S1-S276. doi:10.1016/j.kint.2021.05.021

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3. Bonilla, M., Efe, O., Selvaskandan, H., & Lerma, E.V. (2024). A review of focal segmental glomerulosclerosis classification with a focus on genetic associations. *Kidney Medicine, 6*, 100826. doi:10.1016/j.xkme.2024.100826

4. D'Agati, V.D., Kaskel, F.J., & Falk, R.J. (2011). Focal segmental glomerulosclerosis. *New England Journal of Medicine, 365*(25), 2398-2411. doi:10.1056/NEJMra1106556

5. Forster, A., Sabur, N.F., Iqbal, A., Vaughan, S., & Thomson, B. (2024). Glomerulonephritis during *Mycobacterium tuberculosis* infection: Scoping review. *BMC Nephrology, 25*, 285. doi:10.1186/s12882-024-03716-6

6. Lin, R., McDonald, G., Jolly, T., Batten, A., & Chacko, B. (2020). A systematic review of prophylactic anticoagulation in nephrotic syndrome. *Kidney International Reports*, *5*(4), 435-447. doi:10.1016/j.ekir.2019.12.001

7. D'Agati, V.D., Alster, J.M., Jennette, J.C., Thomas, D.B., Pullman, J., Savino, D.A., et al. (2013). Association of histologic variants in focal segmental glomerulosclerosis clinical trial with presenting features and outcomes. *Clinical Journal of the American Society of Nephrology, 8*(3), 399-406. doi:10.2215/CJN.06100612

8. Kawaguchi, T., Imasawa, T., Kadomura, M., Kitamura, H., Maruyama, S., Ozeki, T., et al. (2022). Focal segmental glomerulosclerosis histologic variants and renal outcomes based on nephrotic syndrome, immunosuppression and proteinuria remission. *Nephrology Dialysis Transplantation, 37*(9), 1679-1690. doi:10.1093/ndt/gfab267

9. Trivedi, M., Pasari, A., Chowdhury, A.R., Abraham, A., & Pandey, R. (2015). Tip variant of focal segmental glomerulosclerosis: Is it truly a benign variant? *Renal Failure*, *37*(5), 763-768. doi:10.3109/0886022X.2015.1014755

10. Vysochyna, I.L., Kramarchuk, V.V., & Yashkina, T.O. (2023). Impostors among family doctors. *Clinical and Preventive Medicine, 4*(26), 75-80. doi:10.31612/2616-4868.4(26).2023.11

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