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PRIMARY RECURRENT FOCAL SEGMENTAL GLOMERULOSCLEROSIS AFTER KIDNEY TRANSPLANTATION. CASE STUDY.

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Summary

Introduction. Primary recurrent focal segmental glomerulosclerosis (FSGS) after kidney transplantation (KT) is a serious complication and a significant risk factor for graft loss. The incidence of FSGS worldwide varies from 1.4 to 21 cases per million population. FSGS develops due to damage to the visceral epithelial cells of the kidney glomerulus (podocytes) and is classified as a "podocytopathy."

Objective. This study aims to demonstrate an early recurrence of FSGS with the development of nephrotic syndrome based on a clinical case and to evaluate the short-term feasibility of plasmapheresis sessions.

Methods. The article describes a clinical case of early recurrence of FSGS with the development of nephrotic syndrome. The primary treatment for disease recurrence after kidney transplantation includes plasmapheresis sessions and the administration of rituximab.

Results. Analysis of the clinical case revealed that kidney graft thrombosis was a consequence of a drop in blood pressure and a slowdown in blood flow at the anastomosis site during a plasmapheresis session, ultimately leading to graft loss and the patient's return to dialysis therapy.

Conclusions. For subsequent planned kidney transplantation, the patient is at extremely high risk of disease recurrence according to the literature, making the procedure not feasible in the short term.

Keywords: *kidney transplantation, focal segmental glomerulosclerosis, nephrotic syndrome.*

Резюме

ПЕРВИЧНЫЙ РЕЦИДИВИРУЮЩИЙ ФОКАЛЬНО - СЕГМЕНТАРНЫЙ ГЛОМЕРУЛОСКЛЕРОЗ ПОСЛЕ ТРАНСПЛАНТАЦИИ ПОЧКИ. КЛИНИЧЕСКИЙ СЛУЧАЙ.

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Введение. Первичный рецидивирующий фокально-сегментарный гломерулосклероз (ФСГС) после трансплантации почки (ТП) является серьезным осложнением и значимым фактором риска потери трансплантированной почки. По разным оценкам в мире заболеваемость ФСГС варьируется от 1,4 до 21 случая на

миллион населения. ФСГС развивается вследствие повреждения висцеральных эпителиальных клеток клубочка почек (подоцитов) и относится к группе «подоцитопатий».

Целью данного исследования. На примере описанного клинического случая указать на необходимость оценки рисков возврата заболевания ФСГС после трансплантации почки, ввиду быстрого возврата заболевания с развитием нефротического синдрома с потерей функции пересаженного органа.

В статье описывается клинический случай проведения трансплантации почки от живого родственного донора, у пациента с хронической болезнью почек 5 стадии в исходе ФСГС, с ранним возвратом заболевания De novo, с развитием нефротического синдрома. Основным лечением в рецидиве заболевания после трансплантации почки является проведение сеансов плазмофереза и введение ритуксимаба.

Результаты исследования. В результате анализа клинического случая было выявлено, что тромбоз трансплантата почки явился следствием падения артериального давления и замедления скорости кровотока в зоне анастомоза на фоне проведенного сеанса плазмафереза, что в конечном итоге привело к потере графта и возврата пациента на заместительную почечную терапию.

Выводы. Для последующей предполагаемой трансплантации почки по литературным данным пациент имеет крайне высокие риски возврата заболевания, что делает проведение данной процедуры в краткосрочной перспективе не целесообразным.

Ключевые слова: *трансплантация почки, фокально-сегментарный гломерулосклероз, нефротический синдром.*

Түйіндеме

БҮЙРЕК ТРАНСПЛАНТАЦИЯСЫНАН КЕЙІНГІ БІРІНШІЛІК ҚАЙТАЛАНАТЫН ФОКАЛЬДІ -СЕГМЕНТАРЛЫ ГЛОМЕРУЛОСКЛЕРОЗДЫҢ КЛИНИКАЛЫҚ ЖАҒДАЙЫНЫҢ СИПАТТАМАСЫ. КЛИНИКАЛЫҚ ЖАҒДАЙ.

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Кіріспе. Бүйрек трансплантациясынан кейінгі бастапқы қайталанатын фокальді-сегменттік гломерулосклероз (ФСГС) ауыр асқыну және трансплантацияланған бүйректің жоғалуының маңызды қауіп факторы болып табылады. Дүние жүзіндегі әртүрлі бағалаулар бойынша ФСГС жиілігі миллионға шаққанда 1,4-тен 21 жағдайға дейін өзгереді. ФСГС бүйрек шумағының (подоциттер) висцеральды эпителий жасушаларының зақымдалуына байланысты дамиды және "подоцитопатиялар"тобына жатады.

Бұл зерттеудің мақсаты клиникалық жағдай негізінде нефротикалық синдромның дамуымен ФСГС ауруының ерте оралуын көрсету және қысқа мерзімді перспективада плазмаферез сеанстарының орындылығын бағалау болып табылады.

Әдістері: мақалада нефротикалық синдромының дамуымен ФСГС ауруының ерте оралуы болған клиникалық жағдай сипатталған. Бүйрек трансплантациясынан кейін аурудың қайталануының негізгі леч плазмоферез сеанстарын жүргізу және ритуксимабты енгізу болып табылады.

Зерттеу нәтижелері. Клиникалық жағдайды талдау нәтижесінде бүйрек трансплантациясының тромбозы қан қысымының төмендеуінен және плазмаферез сеансы аясында анастомоз аймағында қан ағымының баяулауынан туындағаны анықталды, бұл ақыр соңында графттың жоғалуына және пациенттің бүйрек алмастыру терапиясына оралуына әкелді.

Қорытындылар. Әдеби деректерге сәйкес, келесі болжамды бүйрек трансплантациясын өткізуі науқас үшін аурудың қайта қайтып келу қаупі өте жоғары, сондықтан қысқа мерзімде бұл процедураны жүргізуі орынды емес.

Түйінді сөздер: *бүйрек трансплантациясы, фокальді-сегменттік гломерулосклероз, нефроттік синдром.*

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Introduction

Primary recurrent focal segmental glomerulosclerosis (FSGS) after kidney transplantation (KT) is a serious complication and a significant risk factor for the loss of the transplanted kidney [1]. FSGS is a disease characterized by proteinuria, the development of nephrotic syndrome with a high rate of progression with a fairly rapid development of the terminal form of chronic renal failure, also known as end-stage renal disease (ESRD). Relapses of FSGS after kidney transplantation are common and are associated with poor survival of the kidney allograft. Independent risk factors for relapse of FSGS include the onset of the disease in childhood, rapid progression of primary FSGS to ESRD, collapsing variant of FSGS, as well as a history of relapse of FSGS in a previous allograft.

The prevalence of FSGS in Kazakhstan is difficult to estimate due to the lack of widespread native kidney biopsy. Global statistics also provide variable data due to significant geographic and racial differences. The incidence of FSGS is estimated to range from 1.4 to 21 cases per million population [9]. FSGS can occur at any age, affecting approximately 7–10% of children and 20–30% of adults with nephrotic syndrome. In adults, FSGS is more common in men, with an incidence 1.5–2 times higher than in women. The incidence of FSGS is approximately 5 times higher in black patients compared to white patients, with annual incidence in the United States of America (USA) being 24 and 5 cases per million population, respectively [11, 6].

FSGS develops as a result of damage to the visceral epithelial cells of the renal glomerulus (podocytes) and belongs to the group of "podocytopathies". According to the pathogenetic mechanism, FSGS can be divided into primary, secondary and genetically determined. In primary FSGS, widespread damage to podocytes is associated with "permeability factors" circulating in the blood. It is assumed that these factors cause structural and functional changes in podocytes, promote the spreading of their pedunculated processes, apoptosis and detachment from the basement membrane with disruption of the glomerular barrier and the development of proteinuria [5]. Soluble urokinase-type plasminogen activator receptors, cardiotrophin-like factor-1, antibodies to CD40 and others are considered as possible permeability factors, but their nature is currently not entirely clear. Among patients with nephrotic syndrome, the prevalence of FSGS ranges from 12 to 35% and has a high recurrence rate in the transplanted kidney [12].

Early histologic findings of recurrent FSGS include subtle glomerular changes on light microscopy [10, 4, 8] but significant podocyte effacement on electron microscopy; foot process prolapse with possible podocyte dropout leads to the development of segmental glomerular lesions [3, 7]. Experimental and clinical data suggest the existence of circulating permeability factors, such as soluble urokinase-type plasminogen activator receptor (suPAR), cytokine-like factor-1 (CLCF-1), CD40 axis, and apolipoprotein A-Ib (ApoA-Ib), in the pathogenesis of recurrent FSGS. These biomarkers, including circulating permeability factors, may facilitate earlier diagnosis of post-transplant FSGS and may guide the development of new therapies that may be more effective and improve long-term outcomes of kidney transplantation.

Objective: Using the example of the described clinical case, to indicate the need to assess the risks of recurrence of FSGS disease after kidney transplantation, due to the rapid recurrence of the disease with the development of nephrotic syndrome with the loss of function of the transplanted organ

The objectives of the study include conducting a brief literature review of the accumulated experience on post-transplant focal segmental glomerulosclerosis (FSGS) and factors influencing its development, a detailed description of the clinical case of a patient with primary recurrent FSGS after kidney transplantation, and the formulation of conclusions based on the analysis of this clinical case on the use of various procedures and therapeutic approaches.

Description of a clinical case.

Informed consent of the patient and the conclusion of the Local Ethical Commission of LLP "NROC" were obtained for the description of this clinical case. Patient M. (male), 18 years old, was admitted to LLP "NROC" on a planned basis at 12:45 with the diagnosis: Terminal chronic renal failure as a result of FSGS. Nephrobiosia from 01/16/2021. CKD stage 5. (GFR according to CKD EPI 5 ml/min). Uremia. Symptomatic arterial hypertension stage 3. Program hemodialysis since June 2022. 10/05/2023. The disease first debuted at the age of 15. The patient was concerned about the presence of edema in the lower extremities within the shins, loose stools. During examination, proteinuria up to 3.3 g / l, red blood cells in the urine up to 20 in the field of vision, elevated cholesterol levels up to 7.53 mmol / l, creatinine level 76.79 μmol / l, decreased albumin in the blood to 25 g / l, decreased total protein to 42 g / l. A biopsy of native kidneys was performed, morphological signs of focal segmental glomerulosclerosis were diagnosed. A diagnosis of chronic kidney disease stage 3A (CKD3A) against the background of nephrotic syndrome, steroid-resistant variant (GFR 85 - 74 ml / min) was made. The patient received treatment with

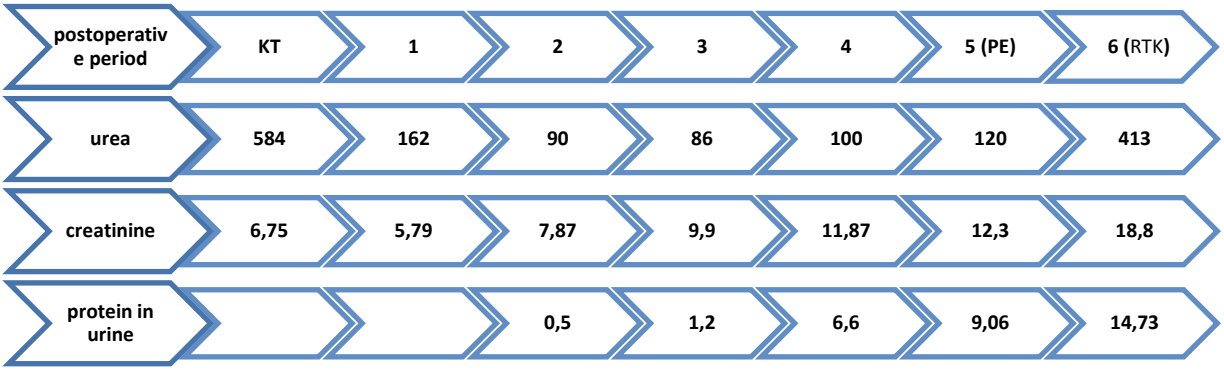
prednisolone 8 mg/every other day, cyclosporine 200 mg/day, during the treatment, partial clinical and laboratory remission was noted. Anemia and metabolic disorders were corrected. Given the presence of nephrotic syndrome, cyclosporine was discontinued, Endoxan 100 mg/day was started, however, during therapy on the 10th day of taking the drug, panic syndrome was noted, skin vesicular rash all over the body, endoxan was discontinued, mycophenolate mofetil (MMF) 1500 mg/day, angiotensin-converting enzyme (ACE) inhibitors 10 mg/day were prescribed under the control of blood pressure (BP) and glucocorticosteroids (GCS). Despite the treatment, renal failure progressed with the development of stage 5 CKD, at the age of 17 the patient was taken to program hemodialysis.

Anamnesis of life and disease. The patient grew and developed according to his age. Denies tuberculosis and venereal diseases. Of the past illnesses, he notes colds. Hemotransfusions were not performed. Heredity is not burdened. Contact with infectious patients is denied. The epidemiological environment is clean. Allergological anamnesis is not burdened.

One year later, the patient expressed a desire to undergo a kidney transplant. The recipient's sister, aged 25, acted as a donor; according to the examination results, the donor had no medical contraindications. The donor's GFR was 97 ml/min/1.73 m2. In terms of preoperative preparation, LP-003672 tacrolimus was started 3 days before the operation, 2.0 mg 2 times a day, manufactured by Nanopharma Development LLC, Republic of Tatarstan, Kazan. On the day of the operation, induction was performed with L04AC02 Basiliximabum 20 mg intravenously, manufactured by Novartis Pharma Stein AG, Switzerland, P N015709/01 Methylprednisolonum 1000 mg intravenously, manufactured by Pfizer, USA. There were no technical complications during the operation, the anastomosis of the renal artery with the internal iliac artery was applied end-to-end, taking into account the small diameter of the external iliac artery. There were no surgical complications in the early postoperative period. The course of the postoperative period was uneventful without complications. The function of the transplanted kidney remained normal, the creatinine level was 86 μmol / l. On the fourth day after the operation, the patient noted a gradual increase in creatinine to - 100 μmol / l, urea - 11.87 mmol / l, in the general urine analysis (GUA) proteinuria up to 6.6 g / l, according to the ultrasound Dopplerography of the renal vessels (USDG) there is no perfusion disorder of the transplanted kidney, in dynamics on the fifth day the creatinine level increased to 120 μmol / l, in the GUA proteinuria up to 9.06 g / l. Given the lack of possibility of morphological verification, biopsy of the transplanted kidney was not performed. Given the history of the disease and the characteristic clinical picture, this condition was considered as a relapse of FSGS de novo on the transplanted kidney. Immunosuppressive therapy was continued: L04AD02 prograf 3.0 mg 2 times a day, manufacturer Astellas Ireland Co. Ltd, Ireland, P N016017/01 myfortic 540 mg 2 times a day, Novartis Neva LLC, Russia, H02AB06 prednisolone 20 mg 1 time per day, Gedeon Richter, Russia. The patient was prescribed plasmapheresis sessions every other day, 3 procedures with replacement of fresh frozen plasma (FFP) and Albumin. On the fifth day, the patient underwent the first plasmapheresis session with albumin and FFP replacement in the evening. After the plasmapheresis session, 2 hours later at night, the patient noted weakness, felt abdominal pain in the umbilical region, dizziness, darkening in the eyes, pain in the fistula area, after which there was a single loose stool, the medical staff diagnosed a drop in blood pressure to 80/40 mm Hg, an infusion of saline solution was performed, blood pressure stabilized, an analgesic drug ketoprofen 800 mg intravenously 1 time, Hanmi Pharmaceutical Co., Ltd, South Korea was prescribed, the pain gradually regressed. In the morning, the fistula on the left forearm is painful to palpation, dense, there is no blood flow. Palpation of the kidney transplant is not enlarged, elastic consistency. Painless on palpation. There was no urination through a urethral catheter at the time of examination of diuresis. The patient underwent Doppler ultrasonography of the transplanted kidney; blood flow was not detected; CT was performed in angio mode; there was no blood flow in the transplanted kidney. According to laboratory data, urea is 18.18 mmol/l, creatinine is 413 μmol/l, and proteinuria with a protein level of 14.75 g/l was found in the general urine analysis.

Figure 1.

Dynamics of laboratory parameters from the moment of kidney transplantation until the moment of removal of the transplanted organ



KT – kidney transplantation;
PE – Plasma exchange;
RTK – Remove Transplanted Kidney.

The patient was urgently taken for surgery, which lasted 3 hours, revision of the transplanted kidney. **Objectively.** During the surgery, kidney transplant thrombosis was diagnosed. Intraoperative attempts to save the transplanted organ were unsuccessful. The kidney transplant was removed. The histological conclusion revealed morphological signs of FSGS with damage to podocytes. Haemorrhages and necrosis in the medulla of the kidney - transplant. Thrombosis, pyelitis, urethritis. In the tissues surrounding the transplant, areas of fibrosis and signs of inflammation were found. The incidence of this complication is from 0.1% to 0.2% of cases and the development of this complication with a high degree of probability leads to the loss of the transplanted organ [1, 2].

Discussion.

FSGS has a high risk of recurrence in the early stages after kidney transplantation. To date, there is no clear evidence of the effectiveness of pretransplant treatment with plasmapheresis or rituximab in the treatment of postoperative recurrence of FSGS after kidney transplantation. The main treatment for recurrence of the disease after kidney transplantation is plasmapheresis sessions and rituximab [2]. One study by Korean scientists indicates that pretransplant treatment with plasmapheresis is associated with a lower risk of immediate recurrence of FSGS after kidney transplantation. However, the isolated use of rituximab did not demonstrate a significant effect in preventing disease recurrence [13]. A 2023 study by Song et al. focused on the study of FSGS recurrence after kidney transplantation depending on pretransplant treatment. The authors noted that pretransplant treatment with plasmapheresis or rituximab does not always prevent early recurrence of FSGS and concluded that new therapeutic strategies are needed to improve treatment efficacy [14]. A systematic review of 77 cases of recurrent FSGS after kidney transplantation showed that about 71% of patients achieved remission after treatment with plasmapheresis. However, delay in treatment initiation and high proteinuria at relapse were associated with lower odds of remission. The use of rituximab also showed mixed results [15].

In this particular case, there was an early recurrence of FSGS with the development of nephrotic syndrome. Kidney graft thrombosis was a consequence of a drop in blood pressure and a slowdown in the blood flow velocity in the anastomosis area during a plasmapheresis session, which ultimately led to the loss of the graft and the patient's return to renal replacement therapy. According to the literature, the patient has an extremely high risk of disease recurrence for subsequent prospective kidney transplantation, which makes this procedure inappropriate in the short term.

Conclusions: This case highlights the importance of careful monitoring and an individual approach to the treatment of patients with FSGS, especially in the presence of factors that contribute to thrombosis. In the future, special attention should be paid to the development of new protocols for the treatment and prevention of FSGS recurrence, including a study of the effectiveness of various immunosuppressive regimens and additional therapeutic methods.

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