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Case Report

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A Case of Nephrotic Syndrome Presenting With Pulmonary Embolus in a Kidney Transplant Patient

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Abstract

Recurrence of glomerulonephritis (GN) is considered an important cause of allograft failure in kidney transplant recipients. It can present after transplantation with complications that are difficult to manage. In this case report, a 53-year-old woman was admitted to the hospital with abdominal pain and swelling in the right lower quadrant during the first month after kidney transplant. Following hospitalization, she complained of sudden-onset dyspnea and chest pain. Thorax computed tomography examination revealed pulmonary embolism (PE); thus, anticoagulation therapy was started. Her 24-h urine proteinuria was quantified as 16695 mg/day, and due to low albumin levels, she was diagnosed with nephrotic syndrome. However, kidney biopsy could not be performed due to the bleeding tendency of the patient. Due to a probable diagnosis of recurrent focal segmental glomerulosclerosis, plasmapheresis was performed. A total of eight plasmapheresis resulted in a complete recovery of the patient considering her proteinuria. In conclusion, recurrence of GN and development of PE due to nephrotic syndrome are well known, but difficult-to-manage. Thus, nephrology follow-up after kidney transplant is crucial.

Keywords: Nephrotic syndrome, pulmonary embolus, kidney transplant

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INTRODUCTION

Recurrence of glomerulonephritis (GN) following kidney transplant is a well-known complication and is considered an important cause of allograft failure in kidney transplant recipients (1). Occasionally, it presents with a devastating clinical picture, and the management can be difficult (2). Here, we present the case of a kidney transplant patient in whom pulmonary embolism (PE) developed due to severe nephrotic syndrome.

CASE PRESENTATION

A 53-year-old female patient who had undergone a living donor (her husband) kidney transplant 1 month ago (May 2017) was admitted to the outpatient clinic with abdominal pain and swelling in the right lower quadrant. She had a history of left radical nephrectomy that was performed in her childhood. The cause of nephrectomy was not documented. The patient has been followed-up by our nephrology outpatient clinic since 2010. Initially, she presented with proteinuria in nephrotic range, hypoalbuminemia, edema, and hyperlipidemia. A kidney biopsy was scheduled; however, it could not be performed because of the presence of cysts in her single kidney. The condition of her kidney gradually deteriorated until the initiation of hemodialysis in January 2017.

During an ultrasonographic examination, performed for the investigation of her abdominal pain, a 16×8 cm of

homogeneous fluid collection between the urinary bladder and the transplanted kidney was detected in the right lower quadrant. The patient was hospitalized for further investigation of the fluid. A sample obtained from the abdominal fluid through punction was evaluated as transudate.

Four days after admission to the hospital, she complained of sudden-onset chest pain and dyspnea. Due to a clinical suspicion of PE, a thorax computerized tomography (CT) was performed. The radiological findings were consentient with PE; therefore, anticoagulation therapy with enoxaparin 0.6 ml subcutaneously twice a day was started. On echocardiographic examination, ejection fraction was found to be 55%, and a thrombus was suspected in the right atrium. Transesophageal echocardiography did not show any vegetations or thrombi. A work-up for thrombophilia profile was performed. Protein S (81.5%), antiphospholipid IgG (1.26 U/mL), antiphospholipid IgM (0.26 U/mL), anticardiolipin IgG (1.05 GPL U/mL), and anticardiolipin IgM (0.69 MPL U/mL) levels were normal; however, protein C levels (159.6%) were high. Her laboratory findings were as follows: urea, 50 mg/dL; creatinine, 1.09 mg/dL; serum albumin, 4.03 g/dL; hematocrit, 35%; leucocytes, 6100/mm^3; and platelets, 181000/mm^3. Urinary sediment examination revealed four leucocytes and seven erythrocytes per high power field, and urinary protein excretion was positive. Collection of 24-h urine sample showed proteinuria at 16695 mg/day and microalbuminuria at 12709 mg/day. Activated partial thromboplastin time was 30.8 s, and international normalized ratio was 0.83. The patient's blood albumin levels had decreased to 3.43 mg/dL.

A diagnosed of nephrotic syndrome, with focal segmental glomerulosclerosis (FSGS) as its possible cause, was considered. However, because of the anticoagulation therapy and risk of bleeding, a biopsy could not be performed. Based on the clinical judgment and considering the high probability of FSGS, a total of eight plasmaphereses were performed during 4 weeks. Eventually, proteinuria regressed significantly, and the clinical picture of nephrotic syndrome remitted completely. The final laboratory findings were as follows: proteinuria decreased from 16695 mg/day to 348 mg/day and microalbuminuria from 12709mg/day to 187 mg/day. Written informed consent was obtained from the patient who participated in this study.

DISCUSSION

Thrombosis in patients with nephrotic syndrome can occur due to various causes such as enhanced platelet activation and aggregation, enhanced coagulation system activation via high-molecular-weight coagulation factor accumulation, decreased endogenous anticoagulant concentrations, and decreased fibrinolytic system activity. Intravascular volume depletion due to nephrotic syndrome, changes in glomerular hemostatic system, and exposure to corticosteroids are other exacerbating factors for a thromboembolic event (3, 4).

GN is a common cause of allograft failure in up to 20% of kidney transplant recipients. Nevertheless, the risk of recurrence depends significantly on the primary glomerular disease (1). Recently, the establishment of protocol biopsy programs and registry databases has provided more precise data regarding the incidence and impact of recurrent GN on allograft failure. Post-transplant proteinuria is a common finding that has a significant impact on allograft failure and patient survival (5, 6). In our patient, a kidney biopsy could not be performed because she had a single kidney prior to transplantation and was on anticoagulation treatment after transplantation. Therefore, the exact pathological diagnosis of the kidney disease could not be made. However, FSGS was considered based on the clinical evidence and rapid response to the treatment. Additionally, it is well known that FSGS reoccurs in 30%-50% of kidney recipients and is associated with two types of clinical presentation. More commonly, it occurs within hours or days of the transplantation and results in rapid allograft loss if left untreated. Neither post-transplant duration of hemodialysis nor immunosuppressive treatment choice changes the probability of graft survival or recurrence of FSGS (7, 8). It can also have an insidious onset and develops over months or years and presents with symptoms that are similarly to those of early reoccurrence (7, 8). An unidentified permeability factor is considered to play a role in the pathogenesis of recurrent GN after kidney transplant. Therefore, plasmapheresis is recommended for the treatment of recurrence. In a meta-analysis of 423 patients, 71% of the patients exhibited a complete or partial remission after undergoing appropriate plasmapheresis for the treatment of recurrence of FSGS after kidney transplant (9).

CONCLUSION

In the present report, we described the treatment of a patient with a probable FSGS diagnosis who presented with recurrent GN, nephrotic syndrome, and PE 1 month after kidney transplant. Plasmapheresis resulted in disease remission.

Informed Consent: Written informed consent was obtained from the patient who participated in this study.

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