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CO-EXISTENCE OF IGA NEPHROPATHY WITH FOCAL SEGMENTAL GLOMERULONEPHRITIS IN RENAL ALLOGRAFT- A RARE CASE.

Nephrology

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KEYWORDS

INTRODUCTION

FSGS is one of the leading glomerular causes of kidney failure in adults[4]. When no secondary cause (genetic, viral, drug-associated, or adaptive[5] can be identified and the patient presents with a clinical history of nephrotic syndrome, FSGS is termed primary or idiopathic. FSGS can progress to chronic kidney disease requiring renal replacement therapy.FSGS can also recur after transplantation in the graft. Reported rates of recurrence are wide, ranging from 17% to 55% [6] in smaller studies and from 9% to 15%[7] in registry-based studies. Recurrence of FSGS with IgA nephropathy post kidney transplantation as in our patient is rarely reported, warranting this case report.

Case Report

Informed consent obtained from the family members before reporting the case report to the journal.

A 20 years old female patient known case of Chronic Kidney Disease on Maintenance Hemodialysis thrice a week , presented to our OPD in December 2022 for Renal Transplantation workup with biological father as prospective donor.

Patient was diagnosed with Nephrotic syndrome at 3 years of age. She was treated with steroids and labelled as steroid resistant nephrotic syndrome(SRNS). Renal biopsy was done, histopathology revealed Focal segmental glomerulosclerosis (FSGS)- NOS type. She was treated with Calcineurin Inhibitors (Tacrolimus) and lost for follow up. September 2022, patient was admitted at Private hospital with breathlessness and was diagnosed with Chronic kidney disease and was initiated on hemodialysis, subsequently left Arterio-venous fistula was constructed.

Workup for renal transplantation was done after explaining the process and risk of recurrence of FSGS in renal graft post transplantation. Genetic analysis was done and was reported as, no pathogenic or likely pathogenic variants causative of FSGS phenotype were detected. Renal transplantation was done on 20th February 2024 and initiated on triple immune-suppressants (Tacrolimus, Mycophenolate sodium and Prednisolone).Delayed graft function was noted with discharge S. Creatinine as 1.5mg/dl and best S.Creatinine as 1.1mg/dl, which increased to 2.3mg/dl(Table 1). Lowest Urine Protein-creatinine ratio noted was 2.3, which increased to 9gms per day over a period of 2 months. Patient was subjected to graft biopsy after ruling out obstruction, infection and Tacrolimus toxicity, biopsy revealed IgA nephropathy with Focal mesangial proliferative glomerulonephritis with focal sclerosis and moderate interstitial fibrosis and tubular atrophy (IFTA) (Figure 1). Patient is currently on Plasmapheresis. With treatment her S.Creatinine has reduced to 1.5mg/dl.

Table 1: S. Creatinine and Urine PCR Values Measured Post Transplantation.

Month/Year	S.Creatinine	Urine PCR
February 2024	1.5mg/dl	20.2(Pre transplant)
March2024	1.1mg/dl	8.37
April 2024	1.3mg/dl	4.4
May 2024	1.3mg/dl	-
June 2024	1.3mg/dl	2.44
July 2024	1.6mg/dl	3.19
August 2024	1.5mg/dl	-
September 2024	1.5mg/dl	-
October2024	1.5mg/dl	-
November 2024	1.7mg/dl	-
December 2024	1.7mg/dl	8.15
January 2025	1.8mg/dl-2.0mg/dl	9.11
February 2025	2.3mg/dl	9.15

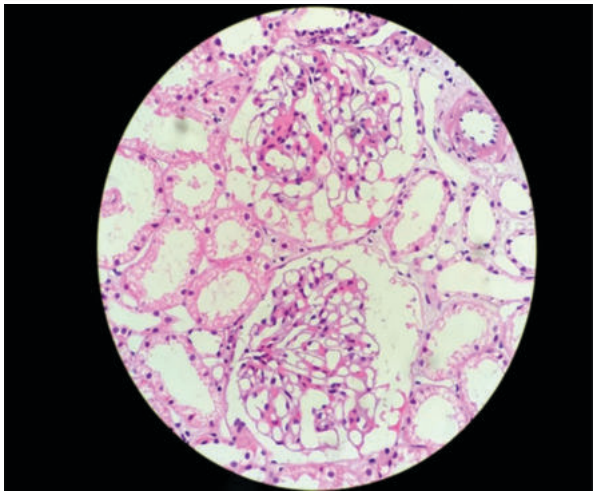

Figure 1:Light Microscopic Picture Showing Focal Mesangial Proliferative Glomerulonephritis with Focal Sclerosis and Interstitial Fibrosis and Tubular Atrophy (IFTA)

DISCUSSION

Glomerular disease is one of the leading causes of kidney failure, representing the third most common reason for kidney transplantation. After kidney transplantation, glomerular disease has been identified as

an important contributor to allograft failure in registry studies worldwide[8].

Among glomerular diseases,recurrence of primary FSGS after kidney transplant is immensely challenging. Recurrence rates after transplant vary from 30%–60% between studies[9].The pathogenesis of recurrent FSGS is still largely unknown, although the presence of a circulating factor toxic to podocytes is highly suggestive[10].Treatment of recurrence focuses in removing this factor from the circulation, and thus most centers use therapeutic plasmapheresis or immuno-adsorption, alone or in combination with rituximab[11]. Despite the use of multiple treatment approaches, resistant disease and graft loss remain common.

The incidence of recurrent IgA nephropathy increases with time after transplant. Recurrence rates vary from 10% to 30% in studies with for-cause biopsies, and 25%–53% in studies with protocol biopsies[12].IgA nephropathy with FSGS, there has been reported faster decline in GFR compared to IgA nephropathy alone[3].

Our patient who underwent Kidney transplantation, developed graft dysfunction with worsening S. Creatinine and Proteinuria. Patient was subjected for graft biopsy after ruling out obstruction, infection and tacrolimus toxicity which showed,combined recurrence of IgA nephropathy and FSGS.Incidently finding of IgA can also be possible. Our patient was subjected for Inj Methylprednisolone 500mg on three days and therapeutic plasmapheresis. With 4 sessions of plasmapheresis, her creatinine reduced from 2.3mg/dl to 1.5 mg/dl, urine PCR reduced to 1.3 and is still on follow up.

In conclusion, we describe a rare case of concurrent recurrence of FSGS and IgA nephropathy post kidney transplantation.

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