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Case Report

Recurrent Iga Nephropathy in A Kidney Transplant Patient: A Case Report

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Abstract

There are no universally accepted guidelines for the treatment of recurrent IgA nephropathy. The clinical course of this condition is variable due to the fact that it can be diagnosed in asymptomatic patients on a protocol biopsy, in patients with mild hematuria or proteinuria, or in patients with a rapidly deteriorating kidney function. Our aim was to present the case of a kidney transplant patient with biopsy proven IgA nephropathy. Our findings show that glomerular filtration pressure can be successfully decreased with combined antihypertensive treatment and SGLT2 inhibitor treatment. Refill with personalized immunosuppressive regimen helped us achieve partial remission.

Keywords: Kidney transplantation; Recurrent IgA nephropathy; SGLT2-inhibitor

Introduction

It is common in patients receiving kidney transplantation for kidney failure due to IgA nephropathy that IgA deposits recur in the transplanted kidney. The clinical course of recurrent IgA nephropathy is variable as the condition may be diagnosed in asymptomatic patients on a protocol biopsy,, in patients with mild hematuria or proteinuria, or in patients with a rapidly deteriorating kidney function. As a result, reported rates of recurrence vary significantly between 9% and 61%, mainly due to diverse biopsy protocols and differences in follow-up [1]. Recent studies have shown that recurrence of IgA nephropathy usually manifests a couple of years after transplantation, and longer follow-up studies showed lower survival rates after 5-10 years [2]. A number of risk factors for IgA nephropathy recurrence have been described, including younger age at transplant, transplant without an induction agent, higher HLA-mismatch, and early steroid withdrawal immunosuppressive regimens [4-13]. Biopsies often reveal crescents and fibrinoid necrosis that are also predictors for poor graft outcome. Data from Eurotransplant Registry with 1200 IgA nephropathy subjects found strong association with HLA-B8 and HLA-DR3 [14-17].

Case History

We report the case of a 67-year-old man with biopsy proven IgA nephropathy. According to his history, he had had hypertension since the age of 27, 6 months of hemodialysis, and received a transplant at the age of 43. He had stable kidney graft function, and his main immunosuppression treatment included cyclosporin A and mycophenolate mofetil. He had no preformed or de novo donor specific antibodies at all. He had diabetes, for which metformin was started. In 2020 the patient was diagnosed with microscopic hematuria, accompanied by proteinuria with a range of 2 g-1500 mg /24 hours. We recommended graft biopsy and checked his immune laboratory parameters. Serum electrophoresis did not show monoclonality, and cryoglobulin was also negative (Table 1). Histology showed IgA nephropathy in graft kidney, as shown in Figure 1. We increased immunosuppression in order to achieve a higher cyclosporin level, and increased mycophenolate mofetil for 3 months. An SGLT2 inhibitor (dapagliflozin) was also administered in addition to ARB's. The patient was followed up by our team. There were no significant reductions in eGFR before or after dapagliflozin administration, and no dehydration or urogenital infections were observed during the treatment course; proteinuria (ACR: albumin creatinine ratio) decreased. Table 1 summarises our patients' clinical data.

Gender	**Male**	
Age (years)	67	
		after treatment
Primary renal disease	IgA nephropathy	
Period after transplantation (years)	23	
Waist circumference (cm)	93	87
Immunosuppressant	CyA, MPA	
Concomitant drugs	dapagliflozin, metformin, telmisartan, ezetimibe, fluvastatin	dapagliflozin, metformin, telmisartan, ezetimibe, fluvastatin
Complications	Fatty liver dyslipidemia, hypothyreosis	
Family history	DM (-) Renal disease (-)	
Past history	HT	
Laboratory		
IgA	elevated	
ANCA	negative	
cryoglobulin	negative	
DSA (MFI)	negative	
ACR (mg/mmol)	**>70**	**15>**
creatinine (µmol/L)	124	134
eGFR (ml/min/1,73 m^2)	51	46
HgbA1 C (%)	**6.7**	**6.1**
hgb (g/L)	126	118
LDL-C (mmol/L)	4.93	3.1
HDL-C (mmol/L)	1.01	1.03

Abbreviations: ACR: Albumin Creatinine Ratio; Cya: Cyclosporin A; DM: Diabetes Mellitus; HT: Hypertension; Igan: Iga Nephropathy; EGFR: Estimated Glomerular Filtration Rate; MMF: Mycophenolate Mofetil

Table 1: Clinical profile of the patient.

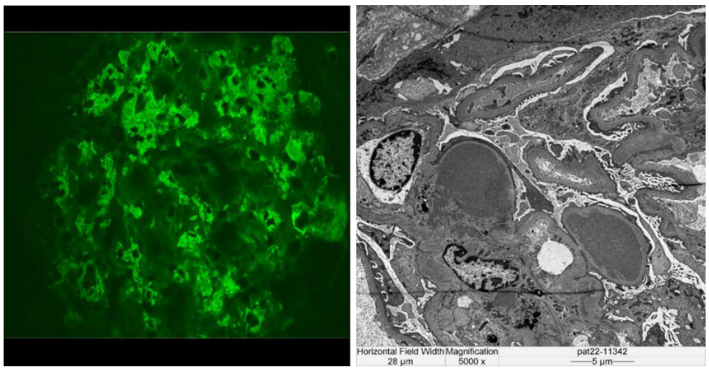

Figure 1: 1a. immunostaining confirms mesangial IgA deposits and IgA positivity in the glomerular mesangium with typical tree branch pattern **1b**. electronmicroscopy: mesangial deposits near the paramesangial glomerular basal membrane, electron-dense deposition in mesangium, podocyte fusion in 30-40%.

Discussion

There are no universally accepted guidelines for the treatment of recurrent IgA nephropathy. Management of recurrent IgA deposits focused mainly on starting or increasing ACEi or ARB treatment. Additional therapies, such as pulse steroids or intravenous cyclophosphamide, lack strong evidence in literature. Nonetheless, in 25% of patients, one of these treatments was used [18,19]. As the DAPA-CKD trial included more patients with immunoglobulin A nephropathy (IgAN) than any of the previous IgAN trials, dual renin-angiotensin/SGLT2 inhibition may become the new standard. In differential diagnosis we need to take into consideration postinfectious glomerulonephritis, shunt nephritis, endocarditis related glomerulonephritis, and IgA dominant infection related GN [20-23].

Conclusion

To conclude, we presented a recurrent IgA np case after kidney transplantation. There were no significant reductions in eGFR before or after dapagliflozin administration, and no dehydration or urogenital infections were observed during the treatment course. Glomerular filtration pressure can be successfully decreased with combined antihypertensive and SGLT2 inhibitor treatment. After refill with individualized immunosuppressive regimen and dual RAAS blockade with SGLT2 inhibitors we could achieve partial remission. This case report has several limitations. First, this report is a single-center study including only one case; but as there are a few reports worldwide, we do hope that this presentation will trigger further research.

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