



## Diabetes and Diabetic Nephropathy Co-Occurring after Kidney Transplants Report on a Case

Surasak Kantachuvesiri\*

Department of Medicine, Division of Nephrology, Ramathibodi Hospital, Thailand

### Abstract

Membranous nephropathy (MN) is a glomerular complaint generally set up in transplanted feathers. The natural history of MN post-transplant is changeable and robotic absolution is uncommon. Diabetic nephropathy (DN) is also generally seen in cases with dragged new onset diabetes mellitus after transplant (NODAT). Still, there have been no former reports ofco-existing MN and DN in transplanted feathers. We herein report a case of 53- time-old manly with earlypost-transplant proteinuria and bitsy hematuria due to MN with posterior clinical robotic absolution. Due to the early onset of complaint after transplant and presence of serumanti-phospholipase A2 receptor (anti-PLA2R) antibody, the substantiation suggests primary intermittent MN in this case. He was also diagnosed with NODAT, with fair glycaemic control with oral hypoglycaemic agents. Sixteen times after absolution, he developed intermittent proteinuria and progressive impairment of renal function. The allograft vivisection revealed both MN and DN. Both conditions may have contributed to the development of glomerular pathology in this case.

**Keywords:** Diabetic nephropathy; Natural coumarins; Oxidative stress; Inflammation; Advanced glycation end products

### Introduction

Membranous nephropathy( MN) is a common cause of nephrotic pattern in grown-ups and is one of the most frequent glomerular conditions set up in thepost-transplant period leading to poor allograft survival( 2). New onset diabetes mellitus after transplant (NODAT) has been reported to do in 4 – 25 of renal transplant donors and may lead to diabetic nephropathy (DN), which results in lower allograft and case survival. Still, there have been no former reports ofco-existing of MN and DN in transplanted feathers. Then, we report a case ofco-existing post-transplant MN with DN as a complication of NODAT [1].

### Material and Methods

A 53- time-old Thai joker with end- stage renal complaint (ESRD) due to hypertensive nephropathy entered an HLA-identical allograft renal transplant from his family in February 1997. He'd no previous history of diabetes mellitus (DM) but his mama was a diabetic. Induction treatment with methylprednisolone 1000 mg, 500 mg, and 250 mg was given on days 0, 1 and 2, independently. The conservation immunosuppressive authority comported of cyclosporine A 350 mg/ day and prednisolone 20 mg/ day. His postoperative period was uneventful and allograft function was excellent [2].

Within four months after the transplant, his serum creatinine position rose from1.2 mg/ dL to3.3 mg/ dL. Allograft vivisection was performed and showed no signs of graft rejection. Cyclosporin-convincing nephrotoxicity was diagnosed since the loftiest cyclosporin trough position reported was 414 ng/ ml. After the lozenge of cyclosporin was lowered, the serum creatinine position dropped to1.6 mg/ dL.

In 1998, he developed DM, which was controlled with glipizide 5 mg/ day and sitagliptin 100 mg/ day performing in fair glycaemic control with HbA1c situations between6.3 and8.1 (Fig. 1C). His systolic blood pressure varied between 130 and 160 mmHg and diastolic blood pressure varied between 80 and 110 mmHg [3].

In 1999, urinalysis during follow up revealed proteinuria with a urine protein of2.8 g/ day, RBC 5 – 10/ HPF with no cellular casts. Allograft vivisection revealed thickening of the glomerular capillary

walls in all glomeruli. An opinion of MN and hyaline arteriosclerosis was made. Enalapril was given to control proteinuria with poor response. He'd robotic clinical absolution after 4 times of treatment.

In 2014, he presented with bending edema in both legs, and positive dipstick proteinuria. The UPCR was1.85 g/ g, serum albumin of3.29 g/ dL, cholesterol of 185 mg/ dL andanti-phospholipase A2 receptor (anti-PLA2R) was detected in the serum [4]. Renal ultrasonography revealed a normal appearance of the renal allograft. HBsAg, anti-HBs, Anti-HCV andante-HIV were negative. Allograft vivisection was performed and showed thickened capillary wall and expanded meningeal matrix in the glomeruli weak direct IgG deposit on immunofluorescence and thickened basement membrane (510 – 614 nm.) on electron microscopy, compatible with diabetic nephropathy, class 2. In addition, grainy deposits of IgG along the capillary wall corresponded with harpoons on light microscopy and foci of electron lucent and thick deposits in sub epithelium and intramembranous all features of membranous nephropathy. C4d was negative in the per tubular capillaries but positive in glomerular capillaries.

### Discussion

We present this case Ofcom-existing MN and DN in a transplant case. One time after transplantation he developed NODAT, the most common complication in renal transplant cases and has been reported to do in 4 – 25 of donors. The cause of NODAT in this case was allowed to be due to the side goods of the immunosuppressive agents including cyclosporine A and prednisolone combined with a family history of DM. In former studies, the domestic history of DM along with race,

\*Corresponding author: Surasak Kantachuvesiri, Department of Medicine, Division of Nephrology, Ramathibodi Hospital, Thailand, E-mail: Kantachuvesiri\_sk@ks.co.th

**Received:** 01-Feb-2023, Manuscript No: TROA-23-89955, **Editor assigned:** 03-Feb-2023, PreQC No: TROA-23-89955 (PQ), **Reviewed:** 16-Feb-2023, QC No: TROA-23-89955, **Revised:** 21-Feb-2023, Manuscript No: TROA-23-89955, **Published:** 28-Feb-2023, DOI: 10.4172/troa.1000160

**Citation:** Kantachuvesiri S (2023) Diabetes and Diabetic Nephropathy Co-Occurring after Kidney Transplants Report on a Case. *Transplant Rep* 8: 160.

**Copyright:** © 2023 Kantachuvesiri S. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

age, inheritable background, former glucose dogmatism, rotundity, hepatitis C contagion, and cytomegalovirus infection were threat factors of NODAT. The case's NODAT was fairly controlled with oral hypoglycaemic agents [5].

Two times after transplantation, he developed proteinuria and bitsy haematuria. Allograft vivisection revealed thickening of the glomerular capillary walls in all glomeruli, harmonious with the histopathological features of MN. From former reports, intermittent MN passed earlier than de novo MN ( $15.58 \pm 19.13$  vs.  $49.27 \pm 32.71$  months), our case was thus considered as intermittent MN, which has been reported to do in 2.5 of transplanted cases. Such a significant difference suggests that different mechanisms are involved in the physiopathology of these conditions. For de novo MN, HCV and patron-specific antibody feel to be the important etiologic factors. Since no given etiologist of MN including viral infection, malice, autoimmune complaint or cryoglobulinemia have been linked to date, we believe he has idiopathic MN. In addition, the allograft vivisection also revealed hyaline arteriosclerosis, which can be set up in hypertensive nephropathy, calcineurin asset nephrotoxicity, or DN. Enalapril was given to control proteinuria but without response. He'd robotic clinical absolution after 4 times of treatment. Robotic absolution of idiopathic MN has been observed among transplant cases and is a well-known point [6, 7].

Seventeen times after transplant, he presented with leg edema and intermittent proteinuria. The discrimination judgments were intermittent MN, DN, and transplant glomerulopathy. Allograft vivisection revealed histopathological features of both MN and DN. The hepatitis and HIV viral studies were negative except for anti-phospholipase A2 receptor (PLA2R) antibody which was detected in the serum. Anti-PLA2R antibody is a biomarker that can be used to separate between idiopathic and secondary MN. Thus, it was more likely that the case had idiopathic intermittent MN. In this case, DN was linked 16 times after the opinion of NODAT. In former studies, NODAT has been reported to beget DN roughly times after transplantation [8].

Indeed though MN and DN are common in the post-transplant periodic-existence of MN and DN has yet to be reported. Post-transplant robotic absolution of MN can be observed, but MN may lead to poor allograft survival. Long standing DM after order-transplant causes significant pathological injury to the allograft, performing in lowered allograft and case survival. The United States Renal Data System (USRDS) has easily stated the relationship between NODAT and a 63 increase in graft failure and an 85 increase in mortality. Collectively, MN and DN have a negative impact on allograft survival. We believe that having both MN and DN may contribute to an indeed worse outgrowth than having either complaint alone.

In native feathers, all cases with MN should admit stylish probative care, including treatment with ACEI/ ARB, lipid lowering agents, and acceptable control of blood pressure. The use of immunosuppressive remedy should be considered on an individual base. In this case, probative care and immunosuppressive remedy were formerly given. It's well known that immunosuppresses are demanded to avoid graft rejection, but their salutary goods on post-transplant MN haven't yet been validated [9, 10].

### Acknowledgment

None

### Conflict of Interest

None

### References

1. Balzano G, Maffi P, Nano R, Mercalli A, Melzi R, et al. (2016) Autologous Islet Transplantation in Patients Requiring Pancreatectomy: A Broader Spectrum of Indications Beyond Chronic Pancreatitis. *Am J Transplant* 16:1812-1826.
2. Chaouch MA, Leon P, Cassese G, Aguilhon C, Khayat S, et al. (2022) Total pancreatectomy with intraportal islet autotransplantation for pancreatic malignancies: a literature overview. *Expert Opin Biol Ther* 22: 491-497.
3. Siegel M, Barlowe T, Smith KD, Chaidarun SS, LaBarre N, et al. (2020) Islet autotransplantation improves glycemic control in patients undergoing elective distal pancreatectomy for benign inflammatory disease. *Clin Transplant* 34: 13891.
4. Tanhehco YC, Weisberg S, Schwartz J (2016) Pancreatic islet autotransplantation for nonmalignant and malignant indications. *Transfusion* 56: 761-770.
5. Zureikat AH, Nguyen T, Boone BA, Wijkstrom M, Hogg ME, et al. (2015) Robotic total pancreatectomy with or without autologous islet cell transplantation: replication of an open technique through a minimal access approach. *Surg Endosc* 29: 176-83.
6. Bhayani NH, Enomoto LM, Miller JL, Ortenzi G, Kaifi JT, et al. (2014) Morbidity of total pancreatectomy with islet cell auto-transplantation compared to total pancreatectomy alone. *HPB (Oxford)* 16: 522-527.
7. Morgan KA, Nishimura M, Uflacker R, Adams DB (2011) Percutaneous transhepatic islet cell autotransplantation after pancreatectomy for chronic pancreatitis: a novel approach. *HPB (Oxford)* 13: 511-516.
8. Jin SM, Oh SH, Kim SK, Jung HS, Choi SH, et al. (2013) Diabetes-free survival in patients who underwent islet autotransplantation after 50% to 60% distal partial pancreatectomy for benign pancreatic tumors. *Transplantation* 95: 1396-403.
9. Muratore S, Zeng X, Korc M, McElyea S, Wilhelm J, et al. (2016) Metastatic Pancreatic Adenocarcinoma After Total Pancreatectomy Islet Autotransplantation for Chronic Pancreatitis. *Am J Transplant* 16: 2747-2752.
10. Bolzano G, Maffi P, Nano R, Zerbi A, Venturini M, et al. (2013) Extending indications for islet autotransplantation in pancreatic surgery. *Ann Surg* 258: 210-218.