WJPMR, 2026, 12(9), 166-170

Case Report

Impact Factor: 7.533

WORLD JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.wjpmr.com

ISSN (O): 2455-3301
ISSN (P): 3051-2557
Coden USA: WJPMBB

A JOURNEY THROUGH RENAL TRANSPLANTATION – FROM STABLE GRAFT FUNCTION TO AN UNEXPECTED DUODENAL NEUROENDOCRINE TUMOR

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DOI: https://doi.org/10.5281/zenodo.22160127

How to cite this Article: Saraswathi Yashaswini[1*], Manjusha Yadla[2]. (2026). A Journey Through Renal Transplantation – From Stable Graft Function To An Unexpected Duodenal Neuroendocrine Tumor. World Journal of Pharmaceutical and Medical Research, 12(9), 166–170.

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Article Received on 01/08/2026 **Article Revised on 21/08/2026** **Article Published on 01/09/2026**

ABSTRACT

Renal transplant recipients require long-term surveillance for graft dysfunction, infections, drug toxicity and malignancy. We report a 44-year-old man with presumed chronic tubulointerstitial nephritis who underwent ABO-compatible living-related renal transplantation from his mother in July 2023. Initial graft function was excellent, with serum creatinine 1.3 mg/dL at discharge. Over the subsequent two years, creatinine gradually increased, followed in 2025 by recurrent diarrhea, vomiting and gastrointestinal bleeding. Evaluation demonstrated apoptotic enteropathy and later a well-differentiated grade 1 neuroendocrine tumor on duodenal biopsy, with synaptophysin positivity and Ki-67 1-3%. Ga-68 DOTATATE PET-CT showed subtle somatostatin-receptor-expressing lesions in the D3/D4 duodenum without regional or distant disease. Concurrently, progressive allograft dysfunction prompted graft biopsy, which was negative for rejection but showed IgA nephropathy with 40% interstitial fibrosis and tubular atrophy. MMF was withdrawn because of suspected drug-related diarrhea and replaced with azathioprine. Tacrolimus exposure was subsequently reduced after an elevated trough level. The patient was referred for multidisciplinary oncology management and commenced long-acting octreotide therapy. This case highlights the diagnostic complexity of progressive graft dysfunction in transplant recipients when gastrointestinal symptoms, medication toxicity, infection, chronic allograft injury and an unexpected neoplasm coexist.

KEYWORDS: Kidney transplantation; renal allograft dysfunction; IgA nephropathy; neuroendocrine tumor; duodenal tumor; tacrolimus; mycophenolate mofetil; octreotide.

INTRODUCTION

Kidney transplantation provides the preferred form of renal replacement therapy for suitable patients with kidney failure, but long-term graft survival depends on careful assessment of immunological, infectious, metabolic and medication-related complications. Recurrent or de novo glomerular disease, chronic interstitial fibrosis and tubular atrophy, calcineurin-inhibitor toxicity, infections and malignancy may all contribute to deterioration in graft function. IgA nephropathy may recur after transplantation and is clinically important because recurrence can be associated with subsequent graft dysfunction and loss.[1-3] Malignancies are also an important long-term complication of immunosuppression. Neuroendocrine tumors of the gastrointestinal tract are uncommon and may be incidentally detected during evaluation of persistent gastrointestinal symptoms.

CASE REPORT

A 44-year-old man from Ranga Reddy was a known case of CKD stage 5D, with the native kidney disease presumed to be chronic interstitial nephritis. He initially presented in June 2023 with pedal edema, decreased urine output, loss of appetite and shortness of breath for approximately two weeks. Serum creatinine was 24

mg/dL and ultrasonography demonstrated bilaterally contracted kidneys. He underwent maintenance hemodialysis for approximately two months before receiving a living-related renal transplant from his mother on 01 July 2023.

The donor and recipient were both blood group B positive and ABO compatible. HLA matching was 3/6, donor-specific antibodies were negative and CDC crossmatch was negative. Warm ischemia time was 2 minutes 30 seconds and cold ischemia time was 1 hour 30 minutes. Induction was performed with methylprednisolone. Postoperatively, graft function improved rapidly, with serum creatinine reaching near-normal levels by postoperative day 3. Serial graft Doppler examinations demonstrated good perfusion and the patient maintained satisfactory urine output. He was discharged on postoperative day 9 with serum creatinine 1.3 mg/dL on triple immunosuppression comprising prednisolone, tacrolimus and mycophenolate mofetil (MMF).

Three months after transplantation, he developed post-transplant diabetes mellitus and was started on metformin 500 mg twice daily. His serum creatinine gradually increased from the post-transplant baseline of 1.3 mg/dL to approximately 1.8 mg/dL over the following two years.

Gastrointestinal Symptoms and Graft Dysfunction

In February 2025, the patient developed watery diarrhea, four to five episodes daily, without blood or foul smell. Serum creatinine increased to 2.3 mg/dL and mild metabolic acidosis was noted. Stool examination for ova, cysts and occult blood was negative, and CMV and EBV PCR were not detected. Graft ultrasonography and Doppler were normal. After intravenous antibiotics and symptomatic treatment, creatinine improved to 1.8 mg/dL. Subsequent creatinine values were 1.8 mg/dL on 02 March 2025 and 1.9 mg/dL on 12 April 2025.

In May 2025, he presented with vomiting and bleeding per rectum. Upper gastrointestinal endoscopy (UGIE) on 18 May showed erosive antral gastritis (Figure 1). Creatinine increased to 2.9 mg/dL. Colonoscopy demonstrated grade III external hemorrhoids and grade II hemorrhoids, while mucosal biopsy showed edema and congestion with apoptotic enteropathy, terminal ileitis and left-sided colitis. There were no features suggestive of inflammatory bowel disease and CMV immunohistochemistry was negative. Renal biopsy was advised but the patient was initially unwilling. He was discharged with creatinine 2.4 mg/dL.

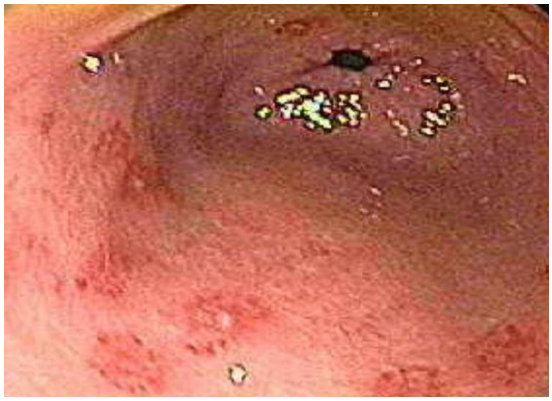

Figure 1: Upper gastrointestinal endoscopy (May 2025) showing erosive antral gastritis with mucosal erythema and scattered erosions.

In July 2025, persistent vomiting and loose stools prompted repeat UGIE, which showed non-erosive corporeal gastritis, erosive antral gastritis and non-erosive duodenitis. Rapid urease testing was positive for H. pylori and eradication therapy with an H. pylori kit and PPI was advised. Because of the possibility of MMF-associated diarrhea, MMF was discontinued and azathioprine 75 mg/day was initiated.

Progressive Allograft Dysfunction and Biopsy Findings

Serum creatinine increased to 2.77 mg/dL on 08 July 2025 and 4.06 mg/dL on 16 July 2025. During the same period, the patient developed herpes zoster and received oral acyclovir for one week. A renal allograft biopsy performed on 18 July 2025 was negative for acute rejection and showed IgA nephropathy with approximately 40% interstitial fibrosis and tubular atrophy (IFTA).

Diagnosis of Duodenal Neuroendocrine Tumor

Review of the gastrointestinal biopsy at NIMS subsequently demonstrated a well-differentiated neuroendocrine tumor, Grade 1, with synaptophysin positivity and Ki-67 of 1-3% (Figures 2 and 3). Medical oncology consultation recommended somatostatin-receptor imaging. Ga-68 DOTATATE PET-CT performed on 29 July 2025 demonstrated subtle somatostatin-receptor-expressing lesions in the D3/D4 portion of the duodenum, possibly representing neuroendocrine tumor, with no other somatostatin-receptor-avid locoregional or distant disease (Figures 4 and 5).

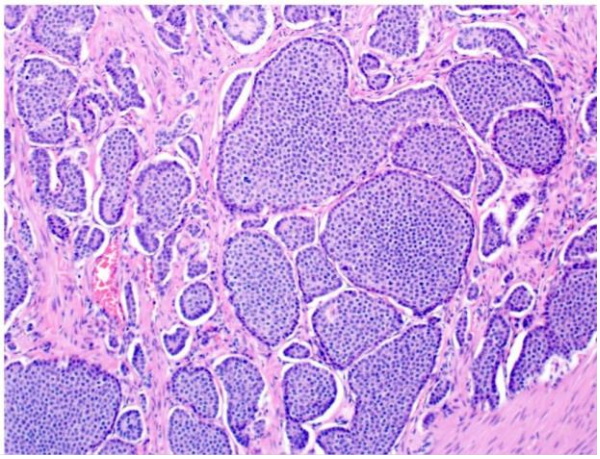

Figure 2: Histopathology (H&E) of the duodenal biopsy showing nests of monomorphic tumor cells with uniform round nuclei separated by delicate fibrovascular septa, consistent with a well-differentiated neuroendocrine tumor.

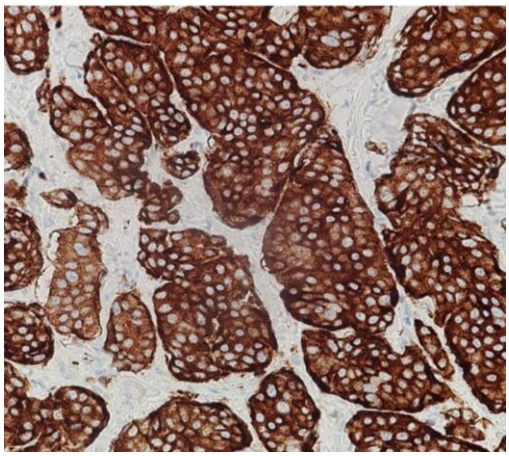

Figure 3: Immunohistochemistry showing diffuse, strong cytoplasmic positivity for synaptophysin in the tumor cells, confirming neuroendocrine differentiation.

On 05 August 2025, surgical oncology assessed the functional status of the tumor. Random blood sugar, C-peptide, serum gastrin and serum chromogranin A were evaluated. Short-acting octreotide 100 micrograms subcutaneously twice daily was advised for five days. After two doses, the patient developed loose stools and vomiting for two days; these symptoms resolved within approximately 24 hours of stopping octreotide.

Repeat UGIE on 07 August 2025 demonstrated a 5 x 5 mm antral nodule and five small nodules, each less than 2 mm, in D2/D3. Biopsy of the antral nodule showed mild chronic gastritis and was negative for intestinal metaplasia, H. pylori, chromogranin, synaptophysin, INSM1 and Ki-67. Subsequently, serum chromogranin A was positive, whereas serum gastrin and C-peptide were negative.

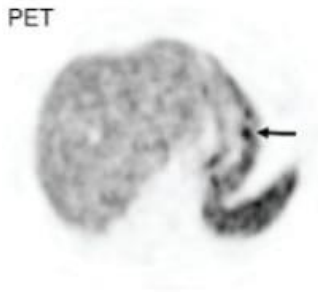

Figure 4: Ga-68 DOTATATE PET (29 July 2025) showing a subtle focus of somatostatin-receptor-avid tracer uptake (arrow) corresponding to the D3/D4 duodenal lesion, without evidence of loco-regional or distant disease

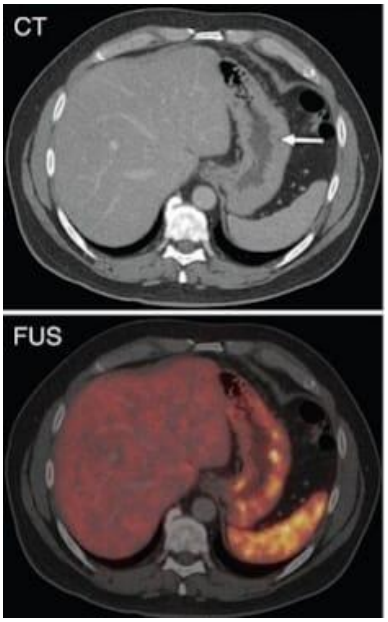

Figure 5: Corresponding axial CT (top) and fused Ga-68 DOTATATE PET-CT (bottom) images localizing the somatostatin-receptor-expressing lesion (arrow) to the D3/D4 segment of the duodenum.

Modification of Immunosuppression and Oncology Management

By August 2025, immunosuppression had been modified to prednisolone 10 mg once daily, tacrolimus 2 mg twice daily and azathioprine 50 mg in the morning and 25 mg in the evening. Loose stools subsided following withdrawal of MMF. Serum creatinine remained approximately 4.08 mg/dL on 26 August 2025.

In November 2025, the tacrolimus trough concentration was 17 ng/mL, following which the tacrolimus dose was reduced to 2 mg in the morning and 1.5 mg in the evening. The patient was subsequently evaluated at MNJ Hospital for the well-differentiated neuroendocrine tumor and treatment with long-acting octreotide (Octreotide LAR) was planned at four-weekly intervals for six doses.

Table 1: Chronological summary of major clinical events, graft function, gastrointestinal evaluation, immunosuppression changes and neuroendocrine tumor work-up.

Date	Clinical event	Key findings	Management/outcome
01 Jul 2023	Living-related renal transplant	Mother donor; 3/6 HLA match; DSA/CDC negative	Good graft function; discharge Cr 1.3 mg/dL
Feb 2025	Watery diarrhea	Cr 2.3 mg/dL; CMV/EBV negative; graft Doppler normal	Supportive treatment; Cr improved to 1.8
May 2025	Vomiting + PR bleeding	Apoptotic enteropathy; ileitis/colitis; CMV IHC negative	Renal biopsy advised; Cr 2.4 at discharge
Jul 2025	Persistent GI symptoms + graft dysfunction	Cr rose to 4.06; graft biopsy: IgA nephropathy, IFTA 40%	MMF stopped; azathioprine started
Jul 2025	Duodenal NET diagnosed	Well-differentiated Grade 1; Ki-67 1-3%; SSTR-positive lesions	DOTATATE PET-CT; oncology referral
Aug-Nov 2025	Immunosuppression/NET management	Loose stools improved; tacrolimus trough 17 ng/mL	Tacrolimus dose reduced; Octreotide LAR planned

DISCUSSION

This case illustrates the diagnostic challenge of progressive renal allograft dysfunction in a patient with concurrent gastrointestinal disease. The initial rise in creatinine occurred in association with diarrhea and vomiting, raising several possibilities including volume depletion, gastrointestinal infection, medication toxicity and calcineurin-inhibitor exposure. The negative CMV and EBV testing and normal graft Doppler helped narrow the differential, while subsequent biopsy demonstrated IgA nephropathy with substantial chronic tubulointerstitial injury.

Although the patient's native kidney disease was presumed to be chronic interstitial nephritis rather than biopsy-proven IgA nephropathy, IgA nephropathy identified in the allograft is clinically significant. In transplant recipients, IgA deposits may represent recurrent disease when the native disease is IgA nephropathy or de novo IgA nephropathy when the original kidney disease was different. Large multicenter data have demonstrated that IgA nephropathy can recur after transplantation and that recurrence is associated with increased risk of graft loss.[1,2] Therefore, in this patient, the biopsy finding should be interpreted in conjunction with the clinical history and original native-kidney diagnosis.

The gastrointestinal manifestations were initially attributed in part to possible MMF-related enteropathy, a recognized complication of mycophenolate therapy. Withdrawal of MMF was followed by improvement in diarrhea, supporting a possible drug-related component. However, the subsequent identification of a well-differentiated duodenal neuroendocrine tumor highlights the importance of continued evaluation when symptoms persist or recur despite modification of immunosuppression.

The tumor was classified as a well-differentiated Grade 1 neuroendocrine tumor with low proliferative activity (Ki-67 1-3%). Ga-68 DOTATATE PET-CT demonstrated SSTR-expressing duodenal lesions without evidence of distant disease, suggesting localized disease on the available imaging. The multidisciplinary approach involving nephrology, gastroenterology, medical oncology and surgical oncology was therefore important in balancing oncological management with preservation of renal allograft function and appropriate immunosuppression.

The case also demonstrates the importance of therapeutic drug monitoring in transplant recipients. The later tacrolimus trough concentration of 17 ng/mL provided an additional potential contributor to graft dysfunction and prompted dose reduction. In a transplant recipient with diarrhea, tacrolimus exposure can become unpredictable and may increase, further emphasizing the need for close monitoring.

CONCLUSION

Progressive renal allograft dysfunction in a transplant recipient should be approached systematically, particularly when accompanied by gastrointestinal symptoms. In this patient, multiple overlapping factors-including gastrointestinal illness, suspected MMF-associated enteropathy, elevated tacrolimus exposure, IgA nephropathy with 40% IFTA and an unexpected localized duodenal neuroendocrine tumor-contributed to a complex clinical course. Early allograft biopsy, careful review of gastrointestinal pathology, therapeutic drug monitoring and multidisciplinary management were essential in establishing the diagnosis and guiding treatment.

ACKNOWLEDGEMENT

The authors acknowledge the Departments of Nephrology, Gastroenterology, Pathology, Medical Oncology and Surgical Oncology involved in the evaluation and management of this patient.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

PATIENT CONSENT

Written informed consent for publication of the clinical information and relevant investigations was obtained from the patient.

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