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Case Report

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RECURRENT POST-TRANSPLANT COMPLICATIONS CULMINATING IN MYCOPHENOLATE MOFETIL-INDUCED COLOPATHY IN A YOUNG RENAL ALLOGRAFT RECIPIENT: A CASE REPORT

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ABSTRACT

Gastrointestinal complications are common after renal transplantation and pose a diagnostic challenge because infective, drug-induced, and rejection-related causes overlap in presentation. We report a 26-year-old female who underwent live-related renal transplantation (mother donor) in February 2023 for hypertensive chronic glomerulonephritis with bilateral small kidneys, following 18 months of maintenance hemodialysis. The post-transplant course was marked by mycophenolate mofetil (MMF)-associated diarrhea, invasive pulmonary aspergillosis requiring liposomal amphotericin B and voriconazole with temporary MMF withdrawal, and recurrent episodes of acute kidney injury associated with diarrheal illness and graft dysfunction. Despite extensive infective work-up (stool, CMV, EBV, and other serologies) remaining largely negative, the patient continued to have persistent loose stools with fluctuating renal function. Colonoscopic biopsy ultimately demonstrated apoptotic crypt abscesses and focal basement membrane thickening without granulomas, viral inclusions, or parasites — histological features consistent with MMF-induced colopathy with an early collagenous colitis pattern. Dose reduction of MMF and tacrolimus, along with supportive care, resulted in clinical improvement and stabilization of graft function. This case highlights the importance of considering MMF-induced colopathy as a distinct entity in the differential diagnosis of persistent post-transplant diarrhea, particularly when infective causes have been reasonably excluded, and underscores the value of histopathological confirmation in guiding immunosuppression adjustment without compromising graft survival.

KEYWORDS: Renal transplantation, mycophenolate mofetil, MMF-induced colopathy, collagenous colitis, invasive pulmonary aspergillosis, acute kidney injury, graft dysfunction.

INTRODUCTION

Renal transplantation remains the treatment of choice for end-stage renal disease, offering superior survival and quality of life compared with maintenance dialysis. However, long-term graft and patient outcomes are frequently threatened by complications of chronic immunosuppression, including opportunistic infections, drug toxicity, and rejection. Gastrointestinal symptoms, particularly diarrhea, are reported in a substantial proportion of renal transplant recipients and carry a broad differential encompassing infective enteritis, cytomegalovirus (CMV) colitis, and drug-induced mucosal injury.

Mycophenolate mofetil (MMF), a cornerstone of maintenance immunosuppression, is well recognized for gastrointestinal toxicity, which may range from mild dyspepsia to severe diarrhea with histological mimics of inflammatory bowel disease or graft-versus-host disease. MMF-induced colopathy is likely under-recognized

because its endoscopic and histopathological features can overlap with infective and immune-mediated colitides, making it a diagnosis of exclusion. We present a case in which recurrent diarrheal illness and graft dysfunction over nearly three years were eventually attributed to MMF-induced colopathy following exhaustive exclusion of infective etiologies and confirmatory colonic biopsy.

CASE REPORT

A 26-year-old female, resident of Ibrahimpatnam, with chronic kidney disease presumed secondary to chronic interstitial nephritis, underwent live-related renal transplantation (LRRT) on 15th February 2023 with her mother as donor (blood group O positive). She had presented in September 2021 with blurring of vision and was found to have grade 3–4 hypertensive retinopathy with blood pressure of 250/140 mmHg, serum creatinine of 8.9 mg/dL, and bilaterally small kidneys, consistent with hypertensive chronic glomerulonephritis. She was maintained on thrice-weekly hemodialysis for 18 months prior to transplantation.

Pre-transplant work-up in both recipient and donor was unremarkable for transmissible infection (HIV, HBsAg, and anti-HCV negative; CMV IgG positive, IgM negative). Baseline investigations showed hemoglobin 8.7 g/dL, urea 86 mg/dL, creatinine 7.23 mg/dL, and HbA1c 5.2%. Echocardiography revealed left ventricular global hypokinesia with an ejection fraction of 45%, mild right ventricular dysfunction, and moderate tricuspid regurgitation.

Induction immunosuppression comprised methylprednisolone (1 g × 2 doses and 750 mg × 1 dose, followed by 1 g × 3 doses), with tacrolimus 1 mg administered the day prior to surgery. The donor renal vein was anastomosed end-to-side to the external iliac vein and the donor renal artery end-to-side to the internal iliac artery; neoureterocystostomy was performed using the modified Lich–Gregoir technique over a DJ stent. The intraoperative course was uneventful, with good on-table diuresis.

Postoperatively, the patient achieved excellent immediate graft function with high urine output and a progressive fall in serum creatinine from 2.4 mg/dL to 0.8 mg/dL at discharge. Maintenance immunosuppression consisted of tacrolimus, MMF, and prednisolone. Doppler ultrasonography of the graft remained normal throughout the admission. The course was complicated by graft-site and ascitic drain output with ascitic fluid analysis suggestive of a transudate; associated hypoalbuminemia was managed with intravenous human albumin. Blood pressure remained elevated and required antihypertensive therapy. Drains and the Foley catheter were removed prior to discharge, and the patient was discharged in stable condition at a weight of 47 kg on tacrolimus, MMF, prednisolone, and supportive medications.

The patient remained asymptomatic on regular follow-up from February to June 2023. In June 2023, she presented with loose stools; stool and blood investigations were unrevealing, and she was treated empirically with intravenous antibiotics and antimotility agents without significant improvement. MMF-induced diarrhea was suspected, and the MMF dose was reduced from 500 mg thrice daily to a staggered 500–250–250 mg regimen, with subsequent resolution of symptoms.

In September 2023, she developed chest pain and breathlessness. Electrocardiography and echocardiography were normal, but chest radiography showed a left upper zone homogeneous opacity. High-resolution computed tomography (HRCT) demonstrated subpleural consolidations with air bronchograms and cavitation, associated ground-glass opacities and septal thickening, and multifocal nodular opacities with cavitation in the right upper and middle lobes and left lower lobe — findings suggestive of cavitating pneumonia. Bronchoalveolar lavage (BAL) and left upper lobe bronchoscopic biopsy were initially inconclusive for infection, and MMF was reduced further to 500–250 mg. In October 2023, she developed pleuritic chest pain; sputum analysis was negative, but bronchoscopy confirmed fungal infection, and blood culture grew Pseudomonas species (sensitive to meropenem and levofloxacin). BAL galactomannan for Aspergillus was positive, with acid-fast bacilli and tuberculosis cultures negative. Repeat HRCT findings of cavitating pneumonia in the left upper lobe with nodular opacities in the right upper and lower lobes were consistent with invasive pulmonary aspergillosis.

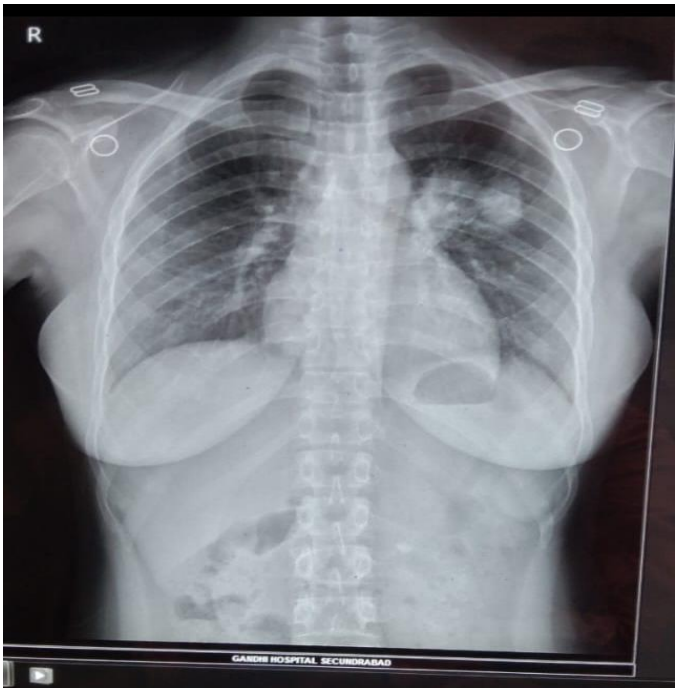

Figure 1: Chest radiograph (PA view), Gandhi Hospital, Secunderabad, October 2023, obtained during the workup for pleuritic chest pain, at the time bronchoscopy and BAL galactomannan confirmed invasive pulmonary aspergillosis. Patient identifiers have been redacted.

She was treated with liposomal amphotericin B (3 mg/kg) for three weeks, followed by oral voriconazole 200 mg twice daily for twelve weeks. MMF was temporarily discontinued and replaced with azathioprine 50 mg once daily during antifungal therapy (creatinine 2.5 mg/dL at that time), with MMF reintroduced after approximately three months. By October 2025, serum creatinine was 2.8 mg/dL with a trough tacrolimus level of 5.7 ng/mL; Doppler of the transplant renal artery was normal.

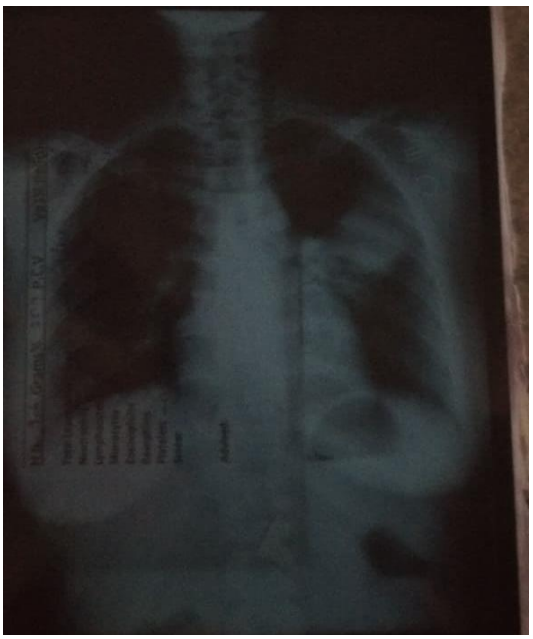

Figure 2: Follow-up chest radiograph, Gandhi Hospital, Secunderabad, November 2023, obtained during the course of liposomal amphotericin B/voriconazole therapy for invasive pulmonary aspergillosis.

In November 2025, she presented with acute gastroenteritis and acute kidney injury, with serum creatinine rising to 4.3 mg/dL. She was managed with antibiotics and intravenous hydration, with resolution of the acute kidney injury and discharge at a creatinine of 3.0 mg/dL. Upper gastrointestinal endoscopy showed non-erosive gastritis, for which appropriate treatment was initiated; stool microscopy for ova, cysts, and

culture, as well as CMV, EBV, Widal, dengue, malaria, and leptospira serologies, were all negative. The MMF dose was reduced to 250 mg twice daily, with improvement in creatinine to 3.1 mg/dL. Trough tacrolimus level was noted to be elevated at 25 ng/mL, prompting reduction of the evening tacrolimus dose to 1.5 mg (tacrolimus 2 mg morning, 1.5 mg evening).

In January 2026, she was readmitted with persistent loose stools, serum creatinine of 6.4 mg/dL, and severe metabolic acidosis. She was managed with intravenous hydration, antibiotics, antimotility agents, and bicarbonate correction, with improvement in creatinine to 4.0 mg/dL; a gastroenterology opinion was obtained. Stool studies for ova, cysts, and culture were again negative. CMV IgM was borderline positive with positive IgG, but CMV viral load was negative; EBV serology was negative.

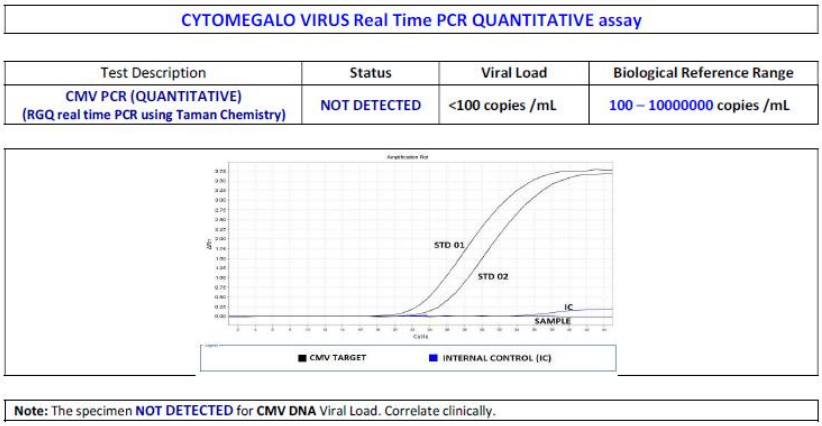

Figure 3: Cytomegalovirus (CMV) quantitative real-time PCR assay performed during the January 2026 admission, showing CMV DNA not detected (<100 copies/mL; reference range 100–10,000,000 copies/mL), supporting exclusion of CMV colitis as a cause of the patient's persistent diarrhea.

Colonoscopy with multiple colonic and rectal biopsies was performed. Histopathology showed superficial fragments of colonic mucosa without significant inflammation of the surface epithelium or lamina propria; sections from the left colon and rectum showed an occasional crypt with an apoptotic abscess and focal thickening of the subepithelial basement membrane entrapping capillary vessels, without granulomas, viral inclusions, or parasites. These features were reported as consistent with MMF-induced colopathy with an early collagenous colitis pattern.

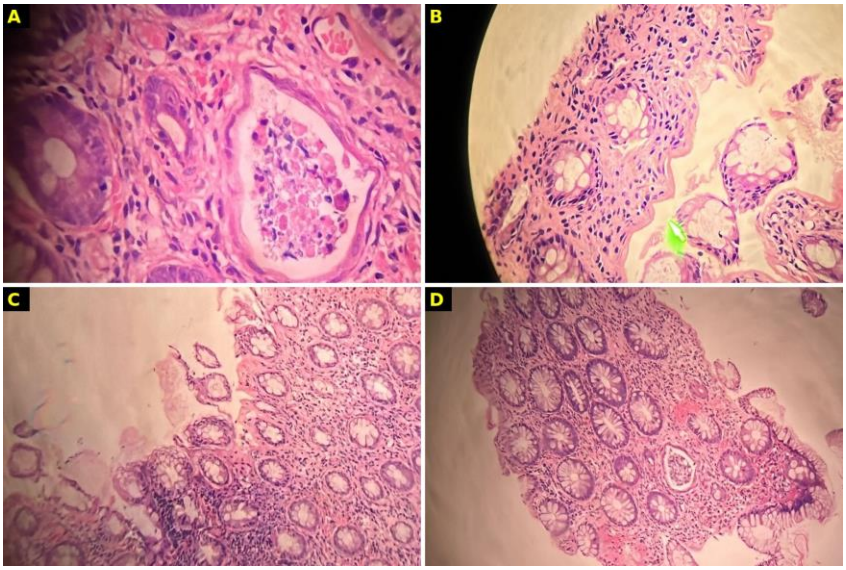

Figure 4: Photomicrographs of colonic and rectal biopsies (haematoxylin and eosin stain) obtained in January 2026. (A) Crypt with an apoptotic abscess and thickening of the subepithelial basement membrane entrapping capillary vessels. (B) Crypts showing increased crypt epithelial apoptosis without granulomas, viral inclusions, or parasites (arrow). (C, D) Colonic mucosa with preserved overall crypt architecture and mild lamina propria changes. These features were reported as consistent with MMF-induced colopathy with an early collagenous colitis pattern.

She was discharged with a serum creatinine of 3.1 mg/dL and a urine output of 2.5 L/day on further-reduced immunosuppression, with plans for close outpatient follow-up of renal function and gastrointestinal symptoms.

Table 1: Summary of Clinical Course.

Timepoint	Event	Key Findings / Management
Sept 2021	Initial presentation	Hypertensive retinopathy grade 3–4, BP 250/140 mmHg, creatinine 8.9 mg/dL, bilateral small kidneys; started on MHD
Feb 2023	Live-related renal transplant	Mother donor; induction methylprednisolone; discharge creatinine 0.8 mg/dL
Jun 2023	Loose stools	Infective work-up negative; suspected MMF-induced diarrhea; MMF dose reduced
Sep–Oct 2023	Pulmonary illness	Invasive pulmonary aspergillosis (BAL galactomannan positive); Pseudomonas bacteremia; treated with liposomal amphotericin B and voriconazole; MMF held, azathioprine substituted
Oct 2025	Follow-up	Creatinine 2.8 mg/dL, tacrolimus 5.7 ng/mL; graft Doppler normal
Nov 2025	Acute gastroenteritis with AKI	Creatinine rose to 4.3 mg/dL, resolved to 3.0 mg/dL; non-erosive gastritis on UGIE; MMF reduced to 250 mg BD
Jan 2026	Persistent diarrhea, AKI, metabolic acidosis	Creatinine 6.4 mg/dL; colonic biopsy showed apoptotic crypt abscesses and basement membrane thickening — MMF-induced colopathy; discharged at creatinine 3.1 mg/dL

DISCUSSION

This case illustrates the diagnostic complexity of persistent gastrointestinal symptoms in renal transplant recipients and the importance of considering drug toxicity once infective and immune-mediated causes have been reasonably excluded. Diarrhea affects a substantial proportion of solid organ transplant recipients on MMF-based regimens, and its pathogenesis is thought to relate to disruption of intestinal epithelial turnover through inhibition of inosine monophosphate dehydrogenase, leading to enterocyte apoptosis and mucosal injury.[1,2] Histologically, MMF-induced colopathy may mimic graft-versus-host disease or inflammatory bowel disease, with apoptotic crypt changes being a characteristic but nonspecific finding; the focal basement membrane thickening seen in this patient has also been described in early collagenous colitis, suggesting a possible overlapping or evolving histological picture.[3]

The patient's course was further complicated by invasive pulmonary aspergillosis, a recognized opportunistic infection in transplant recipients receiving intensive immunosuppression, particularly in the setting of augmented therapy or intercurrent neutropenia-inducing agents.[4] The temporal association between MMF reduction or withdrawal and clinical improvement in gastrointestinal symptoms — first during the diarrheal episode of June 2023 and again after biopsy-confirmed colopathy in January 2026 — supports a causal role for MMF, though recurrent acute kidney injury during diarrheal episodes likely reflected a combination of prerenal insult and calcineurin inhibitor nephrotoxicity, compounded by fluctuating tacrolimus trough levels observed over the course of follow-up.[5]

Extensive negative infective work-up, including stool microscopy and culture, CMV DNA quantification, and EBV serology, was essential in this case to exclude the more common infective and CMV-related causes of post-transplant colitis before attributing symptoms to MMF. This underscores a broader principle in transplant medicine: colonic biopsy with histopathological correlation remains central to distinguishing drug-induced colopathy from infective or rejection-related colitis, and dose adjustment of MMF — rather than discontinuation — was sufficient to achieve clinical improvement while preserving graft function in this patient.[6]

CONCLUSION

MMF-induced colopathy should be considered in renal transplant recipients with recurrent or persistent diarrhea once infective and CMV-related causes have been excluded, particularly when episodes correlate temporally with MMF dosing and improve with dose reduction. Colonic biopsy is valuable in confirming the diagnosis and guiding safe modification of immunosuppression. A multidisciplinary approach involving nephrology, gastroenterology, and histopathology is essential to balance control of drug toxicity against the risk of under-immunosuppression and graft rejection.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

CONSENT

Informed consent was obtained from the patient for publication of this case report and accompanying clinical details.

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