Mid-Hemodialysis Levetiracetam as Rescue Therapy in Patients Having Seizures During Hemodialysis Session

Dear Editor,

Hemodialysis (HD)-associated seizures are a frequent complication.[1] Individuals receiving HD exhibit a higher propensity for seizures than those on peritoneal dialysis.[2] We observed two patients aged 72 and 66 years, with type 2 diabetes mellitus, hypertension, hypothyroidism and seizure disorder, undergoing maintenance hemodialysis. Both were on a regimen of levetiracetam 500 mg twice daily. An additional dose of levetiracetam 250 mg was also administered as per the recommendation. Despite this, both experienced seizure episodes during HD, leading to multiple admissions to the intensive care unit and inadequate dialysis sessions. HD is known to lower serum levetiracetam levels, potentially resulting in subtherapeutic concentrations.[3] To address this issue, we administered an additional 250 mg dose of levetiracetam to both patients one hour after initiating the HD session. This adjustment effectively prevented further seizures. It has been suggested that rapid changes in the osmotic and chemical compositions of extracellular fluid during HD may contribute to the pathogenesis of HD-associated seizures.[4] For patients on dialyzable antiepileptic medications having a preference for HD, administering an additional dose (in this case, levetiracetam) midway through the HD session appears to be a viable strategy.

Conflicts of interest: There are no conflicts of interest.

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How to cite this article: Azad P, Rizvi YS. Mid-Hemodialysis Levetiracetam as Rescue Therapy in Patients Having Seizures During Hemodialysis Session. Indian J Nephrol. 2025;35:446. doi: 10.25259/IJN_649_2024

Received: 27-10-2024; **Accepted:** 17-11-2024; **Online First:** 31-01-2025; **Published:** 10-04-2025

DOI: 10.25259/IJN_649_2024

Mycophenolate-Induced Dysphagia in a Kidney Transplant Recipient

Dear Editor,

Upper gastrointestinal (GI) symptoms such as dysphagia and odynophagia in immunocompromised hosts are generally caused by viral or fungal infections.[1] We present a kidney transplant recipient who developed progressive dysphagia and odynophagia caused by mycophenolate mofetil (MMF) that improved after discontinuation of MMF and serial endoscopic esophageal dilatation.

A 49-year-old male [cytomegalovirus immunoglobulin G negative (CMV IgG −ve), Epstein-Barr virus immunoglobulin G positive (EBV IgG +ve)] underwent living donor kidney transplantation from his sister (CMV IgG +ve, EBV IgG +ve) using Thymoglobulin induction and tacrolimus/MMF maintenance along with infection prophylaxis using valgancyclovir, trimethoprim-sulfamethoxazole, and nystatin. Discharge serum creatinine was 1.5 mg/dL. One month later, he developed progressive dysphagia, odynophagia, reduced appetite, and weight loss. MMF was replaced with azathioprine. The upper endoscopy showed a benign intrinsic severe distal esophageal stricture that was dilated to 7 mm and biopsy showed focal acute inflammation [Figure 1]. Specimen stained negative for fungal elements, CMV and herpes simplex virus (HSV). He required nine more stricture dilatations in over three months to a final diameter of 18 mm. Dysphagia gradually resolved with better dietary intake and weight gain over the next few months.

MMF selectively acts on B and T lymphocytes. Gastrointestinal (GI) toxicity is reported in 40–85% of patients taking MMF and more commonly involves the lower GI tract.[2] Upper GI toxicity usually results in ulcerative esophagitis, reactive gastropathy, and duodenal ulcers. MMF-related esophageal stricture is extremely rare. The mechanism of MMF-related GI toxicity is not clear but may involve MMF-induced

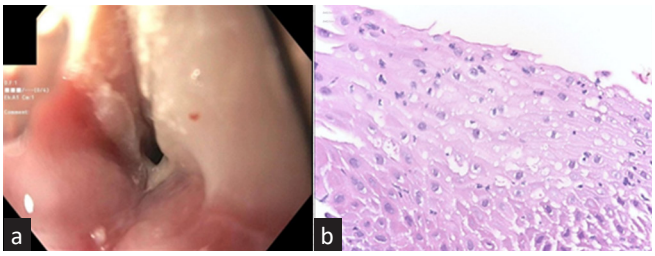

Figure 1: (a) Upper endoscopy showing benign intrinsic severe distal esophageal stricture, (b) Stricture biopsy showing focal acute inflammation. Hematoxylin and Eosin stain x400.

blockade of guanosine nucleotide synthesis upon which the rapidly replicating enterocytes partially depend on, thus disrupting the GI epithelial barrier.[3] MMF metabolites, including mycophenolic acid acyl-glucuronide, can elicit hypersensitivity and autoimmune reactions.[4]

MMF should be considered as a potential etiology for dysphagia from the esophageal stricture in a transplant recipient, especially when common etiologies such as reflux esophagitis and infectious esophagitis are excluded. A high index of suspicion is needed for early diagnosis and timely discontinuation of MMF, since a delay in the diagnosis can contribute to significant morbidity.

Conflicts of interest: There are no conflicts of interest.

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How to cite this article: Sureshkumar KK, Ryhal R, Nashar K, Daloul R. Mycophenolate-Induced Dysphagia in a Kidney Transplant Recipient. Indian J Nephrol. 2025;35:446-7. doi: 10.25259/IJN_680_2024

Received: 07-11-2024; **Accepted:** 22-11-2024; **Online First:** 26-12-2024; **Published:** 10-04-2025

DOI: 10.25259/IJN_680_2024

Evaluating the Impact of Therapeutic Anticoagulation on Acute Kidney Injury in COVID-19: Insights and Future Directions

Dear Editor,

The study by Bansal *et al.*[1] provides valuable insights into the incidence, risk factors, and outcomes of acute kidney injury (AKI) in high-risk COVID-19 patients from a tertiary care center in India. The findings highlight a significant burden of AKI, particularly among intensive care unit (ICU) patients, with 88% developing AKI. Many of these cases required kidney replacement therapy (KRT) and were associated with a high mortality rate of 74%. These results underscore the severe implications of AKI, especially in patients with comorbidities like diabetes and coronary heart disease, as well as those with elevated inflammatory markers such as C-reactive protein, ferritin, and lactate dehydrogenase.[2]

A critical limitation, however, is the absence of data on the number of patients receiving therapeutic dose anticoagulants, such as heparin. This gap limits our ability to assess whether therapeutic-dose anticoagulation could reduce AKI risk in COVID-19 patients. Given COVID-19's tendency to induce thrombotic and inflammatory complications, therapeutic-dose anticoagulants may protect the kidneys by reducing microvascular thrombosis and inflammation—key factors in AKI pathophysiology.[3] Including such data could provide valuable insights, revealing whether a significant association exists between therapeutic-dose anticoagulation and reduced AKI incidence or improved recovery outcomes. Notably, therapeutic-dose heparin has shown promise in other studies for potentially reducing AKI risk, which makes this an important area for further investigation.[4,5]

Understanding the role of therapeutic-dose anticoagulation could be transformative, potentially influencing treatment protocols to reduce AKI incidence and improve patient survival.[6] If protective effects are confirmed, therapeutic-dose anticoagulation may inform optimized care strategies, enhancing outcomes and quality of life for critically ill COVID-19 patients.

In conclusion, while this study sheds light on AKI's burden in COVID-19, addressing the missing data on therapeutic anticoagulation could unlock new avenues in AKI prevention and shape treatment strategies for critically ill patients.