



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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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.

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