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Elevated mycophenolic acid levels after kidney transplantation: avoiding unnecessary dose reduction through team-based TDM interpretation

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Abstract: Mycophenolate mofetil (MMF) is a cornerstone immunosuppressive agent used in combination with calcineurin inhibitors and corticosteroids to prevent acute allograft rejection in solid organ transplant recipients. Long-term transplant outcomes depend not only on the pharmacological efficacy of immunosuppressive regimens but also on individualized dosing strategies and sustained medication adherence. Owing to the complex and highly variable pharmacokinetics of MMF's active metabolite, mycophenolic acid (MPA), therapeutic drug monitoring (TDM) has become an important tool for optimizing treatment safety and efficacy.

We report a case of a kidney transplant recipient with unexpectedly elevated MPA exposure in the absence of clinical toxicity. A detailed pharmacokinetic assessment combined with a comprehensive clinical pharmacist-led medication review indicated that the increased MPA concentrations and elevated $AUC_{0\text{-}12h}$ were most likely related to impaired renal elimination of mycophenolic acid glucuronide (MPAG). Reduced MPAG clearance may enhance enterohepatic recirculation (EHR) of MPA, resulting in increased systemic exposure. This case highlights the importance of interpreting elevated MPA concentrations within a broader clinical context and underscores the critical role of clinical pharmacists, as members of an interdisciplinary team, in integrating TDM with comprehensive pharmacokinetic assessment to ensure safe and effective long-term immunosuppressive therapy.

Keywords: kidney transplant recipient, clinical pharmacist, enterohepatic recirculation, therapeutic drug monitoring, limited sampling strategy.

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Introduction

Mycophenolate mofetil (MMF) is an ester prodrug and a cornerstone immunosuppressive agent administered in combination with calcineurin inhibitors, most commonly tacrolimus, in solid organ transplant recipients. [1] Following oral administration, MMF is rapidly absorbed and undergoes extensive presystemic hydrolysis to its active metabolite, mycophenolic acid (MPA). [2, 3] MPA exerts its immunosuppressive effect through potent, reversible, and non-competitive inhibition of inosine-5′-monophosphate dehydrogenase, the rate-limiting enzyme in de novo guanosine nucleotide synthesis. Because activated T and B lymphocytes rely predominantly on this pathway for proliferation, MPA selectively suppresses lymphocyte expansion while sparing other cell types capable of utilizing salvage pathways. Further, MPA undergoes hepatic glucuronidation to the inactive metabolite mycophenolic acid glucuronide (MPAG). MPAG is primarily eliminated renally; therefore, reduced graft function may decrease MPAG clearance and lead to MPAG accumulation. Increased MPAG availability can enhance enterohepatic recirculation (EHR) through biliary excretion of MPAG, intestinal deconjugation, and subsequent reabsorption of MPA, thereby increasing systemic MPA exposure despite unchanged MMF dosing. [2, 4, 5]

Despite its well-established efficacy, the clinical management of MMF-based immunosuppression in kidney transplant recipients remains challenging. MMF exhibits a relatively narrow therapeutic window: insufficient exposure is associated with an increased risk of acute and late allograft rejection, whereas excessive exposure predisposes patients to toxicity. [6, 7] Gastrointestinal adverse effects are common and frequently lead to dose modification or treatment discontinuation. Hematological complications, particularly leukopenia and anemia, require close laboratory monitoring and may necessitate dose reduction or temporary discontinuation; notably, up to 50% of patients require MMF dose adjustment or discontinuation within the first year after transplantation. [8] In addition, MMF therapy increases susceptibility to infectious complications, including opportunistic viral and fungal infections, further complicating post-transplant care. [2]

MMF pharmacokinetics is characterized by substantial inter- and intra-patient variability and is influenced by multiple clinical and pharmacological factors. Clinically relevant drug-drug interactions play a key role, particularly with calcineurin inhibitors. Cyclosporine interferes with EHR of MPA and may significantly reduce systemic exposure, in contrast to tacrolimus-based regimens. [9] Additional sources of variability include non-linear absorption, high and variable plasma protein binding (approximately 97% albumin-bound), genetic polymorphisms, changes in graft function, and variability in EHR. [4, 5, 10, 11, 12, 13, 14, 15, 16] Importantly, routine clinical markers such as serum creatinine, creatinine clearance, or liver transaminase levels do not consistently correlate with MPA exposure in stable transplant recipients, limiting their utility as predictors of individual pharmacokinetic exposure. [5, 17, 18, 19]

Although MMF was initially prescribed using fixed dosing regimens, it is now well recognized that MPA exposure, expressed as the area under the concentration-time curve (AUC), may vary more than tenfold between patients, ranging from less than 10 mg·h/L to over 100 mg·h/L. [4, 10, 11, 12] Single-point concentration measurements, particularly trough concentration (C_0), correlate poorly with overall exposure and clinical outcomes. [4, 20, 21] Consequently, AUC has emerged as the preferred pharmacokinetic parameter for assessing MPA exposure and guiding dose individualization.

For kidney transplant recipients, a therapeutic target range for MPA $AUC_{0\text{-}12h}$ of 30–60 mg·h/L is generally accepted. [4, 11, 22] However, full 12-hour pharmacokinetic profiling is logistically

demanding and impractical in routine clinical practice. To address this limitation, limited sampling strategy (LSS) has been developed and validated to estimate AUC using a small number of strategically timed blood samples, including abbreviated equations based on concentrations collected within the first 6 hours post-dose ($AUC_{0\text{-}6h}$) to predict the full $AUC_{0–12h}$. [20, 22, 24] The transition from fixed-dose MMF administration to exposure-guided dosing based on therapeutic drug monitoring (TDM) represents a critical step toward personalized immunosuppressive therapy, integrating pharmacokinetic principles with individualized clinical decision-making.

Case description

A 47-year-old man with end-stage renal disease underwent kidney transplantation in 2020. He had been maintained on stable maintenance immunosuppression for approximately three years post-transplant, consisting of MMF 750 mg twice daily, prolonged-release tacrolimus 7 mg once daily, and prednisone 5 mg once daily. The patient was admitted due to biopsy-confirmed recurrence of membranous nephropathy in the transplanted kidney. On the day of admission, laboratory tests revealed a serum creatinine level of 181 μmol/L, an estimated glomerular filtration rate (eGFR) of 37 mL/min/1.73 m^2 calculated using the MDRD equation, and a urine protein-to-creatinine ratio of 79.1 mg/mmol. Urinalysis demonstrated hematuria.

The patient's medical history was significant for arterial hypertension, gout, a past episode of tuberculosis, and atrial fibrillation. Concomitant medications included amlodipine, ramipril, bisoprolol, allopurinol, apixaban, doxazosin, magnesium supplementation, and vitamin D. The patient had also been diagnosed with moderate intellectual disability, necessitating an individualized and closely supervised approach to long-term pharmacotherapy.

On the day of MPA AUC measurement, the patient reported that he had not taken the morning dose of MMF prior to blood sampling and denied any recent changes in medication or supplementation. No clinical signs or symptoms suggestive of MMF toxicity were observed, including gastrointestinal complaints (diarrhea or abdominal pain), infectious symptoms, dysuria, or haematological adverse effects. Laboratory evaluation revealed stable renal function and no clinically relevant abnormalities in liver enzymes, serum albumin, haemoglobin, or white blood cell count.

MPA exposure was assessed using an LSS, and plasma MPA concentrations were determined by an enzymatic immunoassay method. The estimated $AUC_{0\text{-}12h}$ was calculated using a validated LSS equation based on C_{1h}, C_{4h}, and C_{6h} concentrations ($AUC_{0\text{-}12h} = 14.9 + 1.3 \cdot C_{1h} + 3 \cdot C_{4h} + 3.7 \cdot C_{6h}$), [24] yielding an apparently supratherapeutic value of 86 mg·h/L, exceeding the recommended therapeutic range of 30–60 mg·h/L.

A comprehensive medication reconciliation and pharmacotherapy review were conducted by a clinical pharmacist. Potential causes of increased MPA exposure — including hepatic dysfunction, hypoalbuminemia, gastrointestinal adverse effects, drug-drug interactions, and infectious complications — were systematically assessed and excluded. In the context of recurrent membranous nephropathy and documented graft pathology, altered elimination of MPAG was considered a potential explanation for the increased MPA exposure. Subtle alterations in MPAG elimination and EHR related to graft pathology were therefore considered a plausible contributing mechanism. In the absence of clinical toxicity or modifiable pharmacokinetic factors, the MMF dose was maintained, and MPA exposure was reassessed after three weeks.

At follow-up, the patient remained clinically stable with preserved graft function and no signs of MMF-related toxicity. Serum creatinine was 178 μmol/L, with an eGFR (MDRD) of

38 mL/min/1.73 m². A full pharmacokinetic profile was obtained at steady state, with nine blood samples collected over a 12-hour dosing interval (C_0, C_{40min}, C_{1h}, C_{2h}, C_{3h}, C_{4h}, C_{6h}, C_{8h}, and C_{10h}). Individual pharmacokinetic parameters were calculated using non-compartmental analysis (Phoenix WinNonlin v. 8.4, Certara, USA). The $AUC_{0\text{-}12h}$ was 45.25 mg·h/L (Fig. 1), within the therapeutic range, and the remaining pharmacokinetic parameters were consistent with published data for stable kidney transplant recipients.

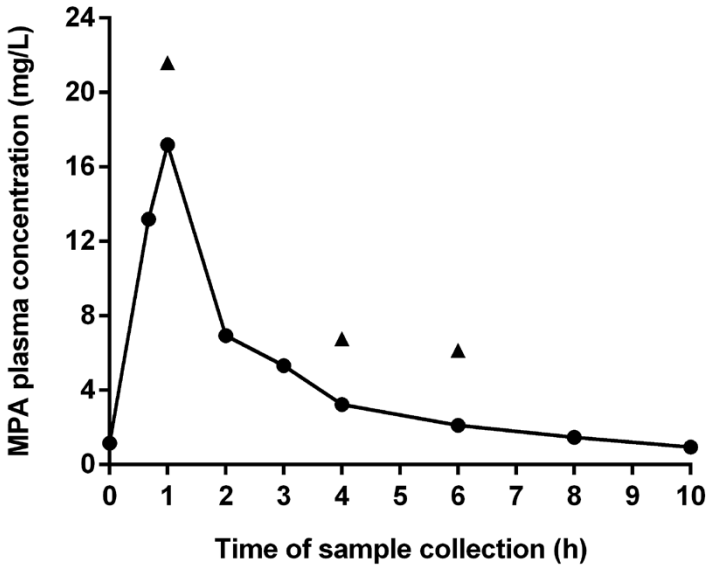

Fig. 1. Comparison of the full 0–10 h steady-state plasma MPA concentration-time profile (nine sampling points) with the selected time points (C_{1h}, C_{4h}, C_{6h}) applied for LSS-derived $AUC_{0\text{-}12h}$ estimation in a kidney transplant recipient. Black circles represent the full concentration-time profile; triangles indicate sampling points used for LSS-based AUC estimation.

Discussion

This case illustrates the clinical complexity of interpreting elevated MPA exposure in the kidney transplant recipient and underscores the importance of integrating TDM with comprehensive pharmacokinetic, pharmaceutical and clinical assessments. A single supratherapeutic AUC value, particularly when derived from LSS, should not automatically prompt dose reduction in the absence of clinical toxicity. LSS models relying predominantly on early post-dose concentrations (0–4 h) may be especially sensitive to shifts in T_{max}, delayed absorption, and coexisting variability in EHR, potentially leading to biased AUC estimates.

In the present case, the initially elevated LSS-derived $AUC_{0\text{-}12h}$ estimate was most plausibly explained by impaired renal elimination of MPAG. Although MPAG concentrations were not measured in this case, reduced MPAG clearance in the setting of graft dysfunction may enhance

EHR and increase systemic MPA exposure; this interpretation remains indirect and is based on the clinical context and available literature. [5] This mechanism has been previously described in kidney transplant recipients and represents an important contributor to interindividual variability in MPA pharmacokinetics. Accordingly, elevated MPA exposure may not necessarily reflect inappropriate dosing but may instead be related to altered metabolite handling and enhanced EHR associated with graft dysfunction.

This approach allows comparison of LSS-derived $AUC_{0\text{-}12h}$ estimates with the reference full $AUC_{0\text{-}12h}$, enabling assessment of bias, precision, and predictive performance of simplified monitoring strategies. [25] This methodology is particularly relevant for MPA, for which full $AUC_{0\text{-}12h}$ is considered the most reliable marker of systemic exposure in solid organ transplant recipients. [26, 27]

Intensive pharmacokinetic assessment is also valuable in cases of unexplained variability in drug concentrations despite stable dosing. Substantial intraindividual fluctuations in MPA concentrations may occur without dose modification and may reflect alterations in absorption kinetics, impaired renal elimination of MPAG, reduced hepatic glucuronidation capacity, variability in EHR, clinically relevant drug-drug interactions, and interindividual genetic differences affecting metabolizing enzymes UGT (e.g. UGT1A9, UGT2B7) and transporters (e.g. MRP2). [4, 26, 28] In such situations, characterization of the full concentration-time profile and calculation of $AUC_{0\text{-}12h}$ enable a more accurate evaluation of systemic exposure and the underlying pharmacokinetic mechanisms. Concurrent comparison with LSS-derived $AUC_{0\text{-}12h}$ provides insight into the applicability and limitations of abbreviated sampling strategies under conditions of increased pharmacokinetic variability.

In the present case, the initially estimated LSS-derived $AUC_{0\text{-}12h}$ was markedly elevated, whereas subsequent full pharmacokinetic profiling demonstrated an $AUC_{0\text{-}12h}$ within the therapeutic range. This discrepancy suggests that transient pharmacokinetic alterations, rather than true sustained overexposure, influenced the abbreviated estimate. It is unlikely that this difference is attributable solely to analytical variability and is more plausibly explained by transient pharmacokinetic perturbations. The absence of MMF-related adverse effects further supports the interpretation that the initial supratherapeutic estimate did not reflect clinically meaningful overexposure.

These findings highlight both the strengths and limitations of LSS in routine practice. Although LSS-based monitoring is validated and cost-effective, its accuracy may be reduced in the setting of altered pharmacokinetics, particularly enhanced EHR. Unexpected TDM results should therefore prompt careful evaluation rather than immediate therapeutic modification.

A recent study [29] has highlighted limitations of conventional LSS models in kidney transplant recipients receiving tacrolimus, particularly under conditions of variable EHR. Model performance appears strongly influenced by this variability, and approaches incorporating later post-dose sampling time points demonstrate improved predictive accuracy. In this context, inclusion of a 6-hour post-dose concentration likely contributed to a more reliable estimation of exposure compared with models based exclusively on early sampling. The marked discrepancy between the initially elevated LSS-derived $AUC_{0\text{-}12h}$ estimate based on abbreviated sampling points and the subsequent full $AUC_{0\text{-}12h}$ within the therapeutic range further suggests that such pharmacokinetic alterations may influence abbreviated sampling-based estimates. Collectively, these observations indicate that LSS-derived AUC values should be interpreted cautiously in patients with altered pharmacokinetics. When clinically indicated, confirmation by full pharmacokinetic profiling may prevent unnecessary dose modification.

More recently, novel LSS models have been proposed to specifically address the limitations imposed by variable EHR. In the study by Mohamed *et al.* [24] involving kidney transplant recipients, LSS models were developed to simultaneously predict $AUC_{0\text{-}12h}$ of MPA, its metabolites (MPAG and acyl mycophenolic acid glucuronide), as well as the extent of MPA EHR. Importantly, the models were developed using a balanced representation of patients with high and low EHR and included late post-dose concentration time points. The best-performing model accurately and reproducibly predicted the $AUC_{0\text{-}12h}$ of MPA and its metabolites, while a separate model enabled reliable estimation of EHR extent. Taken together, these findings indicate that LSS specifically designed to account for EHR and incorporating at least one late post-dose sampling time point (≥5 hours after drug administration) can substantially improve the accuracy of MPA exposure assessment.

In light of these data, selecting an LSS model tailored to the predominant pharmacokinetic mechanisms in a given patient, rather than routinely applying schemes based solely on early time points, is crucial. This case indicates that in patients with significant variability in EHR and altered MPAG metabolism, early MPA concentrations may not reflect true exposure over the 0–12-hour interval, potentially leading to systematic errors in AUC estimation.

An important practical implication is that discrepancies between LSS results and full pharmacokinetic profiling may themselves serve as a clinical signal suggesting the presence of enhanced EHR or altered MPA distribution and albumin binding. In such situations, the LSS result should not automatically be attributed to analytical error but rather considered a cue for more comprehensive pharmacokinetic assessment, including later sampling time points and metabolite analysis. Furthermore, this case highlights the potential role of MPAG measurements as a supportive component of TDM interpretation, particularly when observed AUC values are unexpected in the context of the administered dose and clinical presentation. Finally, these findings suggest that future MPA TDM strategies should move toward adaptive models that account for late sampling points and such variability rather than relying on universal LSS equations. Such an approach may reduce the risk of unjustified MMF dose modification and improve therapeutic safety in patients with atypical pharmacokinetics.

The interpretation of these findings relied on close collaboration within an interdisciplinary transplant team. Clinical pharmacists, physicians, and nurses each contributed essential expertise: pharmacists applied pharmacokinetic principles to contextualize TDM results; physicians integrated these insights with clinical assessment and graft function monitoring; and nurses ensured accurate sample collection and vigilant patient observation. This coordinated approach was crucial in preventing unnecessary modifications to immunosuppressive therapy, optimizing patient safety, and preserving long-term graft function. This report reinforces the importance of interdisciplinary teamwork in managing complex pharmacotherapy in kidney transplant recipients.

Conclusions

This case highlights the complexity of interpreting elevated MPA exposure in kidney transplant recipients and demonstrates that increased MPA concentrations and AUC values may result from impaired renal elimination of MPAG and enhanced EHR, particularly in patients with unstable graft function.

Importantly, elevated MPA exposure should not automatically prompt dose reduction, especially when based on a single abbreviated pharmacokinetic assessment and in the absence of

clinical toxicity, as inappropriate dose modification may increase the risk of under-immunosuppression and graft rejection. Although LSS models are clinically feasible and cost-effective, this case underscores their limitations in the presence of altered pharmacokinetic processes and highlights the need for careful interpretation within the broader clinical and pharmacokinetic context.

A personalized, multidisciplinary approach, combining TDM with comprehensive clinical and pharmacokinetic assessments, remains pivotal for optimizing immunosuppressive therapy and improving long-term transplant outcomes. Clinical pharmacists, as part of the interdisciplinary care team, play an essential role in embedding advanced monitoring strategies, including TDM of MMF and LSS, into routine clinical practice.

Author contribution

A.Ś. — conceptualization, pharmaceutical patient care, pharmacokinetic analysis, writing, editing, discussion; A.C. — conceptualization, pharmacokinetic analysis, writing, discussion, corresponding author; Ł.H. — pharmaceutical patient care, discussion; A.S., A.G. — pharmaceutical patient care, discussion; K.K., K. S. — medical patient care, review, consulting; A.W. — conceptualization, supervision, writing.

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Conflict of interest

None declared.

Abbreviations

AUC	— area under the concentration-time curve
$AUC_{0\text{-}12h}$	— area under the curve concentration-time from 0–12 h after drug administration
$AUC_{0\text{-}6h}$	— area under the concentration-time curve from 0–6 h after drug administration
C_0	— concentration before drug administration
C_{40min}–C_{10h}	— concentration from 40 min up to 10 h after drug administration
DDIs	— drug-drug interactions
eGFR	— estimated glomerular filtration rate
EHR	— enterohepatic recirculation
LSS	— limited sampling strategy
MMF	— mycophenolate mofetil
MPA	— mycophenolic acid
MPAG	— mycophenolic acid glucuronide
PK	— pharmacokinetic
TDM	— therapeutic drug monitoring
Tmax	— time to maximum concentration

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