

TG1

## Long COVID in kidney transplant recipients: a systematic review

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TUESDAY - Moderated Poster Session, HALL Q, March 10, 2026, 16:00 - 17:00

### Introduction

Kidney transplant recipients are at heightened risk of adverse outcomes from SARS-CoV-2 infection due to long-term immunosuppression and multiple comorbidities. While the acute impact of COVID-19 has been widely documented, the burden of persistent sequelae—commonly described as long COVID, post-acute sequelae of SARS-CoV-2, or post-COVID condition—remains less well characterised in this population. Clarifying prevalence, symptom patterns, risk factors, and graft-related outcomes is essential for shaping clinical pathways and long-term transplant care.

### Methods

We undertook a systematic review following PRISMA guidelines. Searches were conducted in PubMed, Google Scholar and the Cochrane Library up to June 2025 and registered in PROSPERO. Eligible studies were published in English, with adult kidney transplant recipients (>18 years), and reported persistent symptoms after acute COVID-19. Definitions of long COVID varied across studies and were accepted provided symptoms persisted beyond the acute phase. Two reviewers (PJ and WA) independently screened records, extracted data, and appraised study quality. Discrepancies were adjudicated with RC. Findings were narratively synthesised, with subgroup analysis of transplant-specific versus broader immunosuppressed cohorts.

### Results

Of 734 records screened, 28 studies met inclusion criteria, including 10 focused specifically on kidney transplant recipients (n≈2,600). Reported prevalence of long COVID ranged from 21% to 70%, reflecting differences in study design and definitions. Commonly described symptoms included fatigue (34–65%), dyspnoea (18–39%), cognitive impairment or “brain fog” (12–28%), musculoskeletal pain (15–41%), and post-exertional malaise. Several risk factors were recurrently associated with long COVID: female sex, obesity, diabetes, older age, reduced baseline graft function, and prior severe or hospitalised acute infection. Higher cumulative corticosteroid exposure, impaired vaccine response, and greater immunosuppression burden emerged as additional signals in smaller series, while frailty was highlighted in select cohorts.

Evidence for graft outcomes varied. Kodiyapalakkal et al. (JASN 2022) reported 12% developing graft dysfunction and 1.6% graft loss, alongside 18% with persistent symptoms. Manfro et al. (Exp Clin Transplant 2025), in a prospective 24-month study, demonstrated that severe acute COVID was associated with significant decline in estimated glomerular filtration rate (~9 mL/min/1.73 m<sup>2</sup>), increased rejection, graft loss, and mortality, whereas mild disease was not associated with major long-term complications. Other cohorts generally reported preserved graft function but noted higher risks when immunosuppression was substantially reduced during acute illness.

Studies in broader solid organ transplant populations showed comparable prevalence and symptom clusters, though estimates were heterogeneous due to differing definitions and methods.

#### Discussion

Long COVID is a frequent and clinically meaningful problem in kidney transplant recipients, with prevalence estimates higher than in the general population and risk factors amplified by immunosuppression and comorbidity. The symptom profile mirrors that of non-transplant cohorts but carries additional implications for graft outcomes and immunosuppression management. While most recipients maintain graft function, severe acute COVID appears to drive long-term functional decline and rejection risk. These findings underscore the importance of structured follow-up, multidisciplinary care, and harmonised research definitions to enable robust cross-cohort comparison. Establishing prospective registries and standardised reporting frameworks will be critical to inform practice and optimise outcomes in this vulnerable population.

TG2

## Real-world clinical outcomes and practical prescribing implications for the use of tirzepatide in patients living with kidney disease: an evaluation of service delivery at a tertiary centre

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The use of tirzepatide in people with chronic kidney disease (CKD) has attracted much interest because of the association of T2DM and obesity with kidney disease progression. Post-hoc analyses of the SURPASS trials have shown improved eGFR slope loss and albuminuria in those with diabetic kidney disease treated with tirzepatide versus standard diabetes therapies. Despite these findings, it is unknown whether clinical outcomes in a real-world setting are similar to published studies in patients with advanced CKD. For patients with a kidney transplant, the outcomes for tirzepatide-use are even less clear, particularly its potential impact on immunosuppression levels. Further, the implications for developing a service that effectively supports prescribing of tirzepatide to kidney patient cohorts have not been explored.

We developed guidelines for the introduction of tirzepatide to a) patients with diabetes and CKD3a-5A2-3 and b) patients with diabetes and a kidney transplant. These guidelines included information on initiation of tirzepatide, monitoring of response to the drug and important contraindications. For the transplant patients this was supported by a Renal-Diabetes MDT monthly meeting.

For patients with CKD3a-5A2-3, we asked whether tirzepatide promoted improvements in metabolic outcomes (weight loss, Hba1c) in line with published trials without deleterious impact on kidney outcomes (eGFR, albuminuria) and collected information on adverse events. Fifty patients prescribed tirzepatide in the CKD cohort were followed prospectively over 16 weeks. Baseline data revealed mean age 61.1 yr, weight 103.2 kg, Hba1c 64.5 mmol/mol, eGFR 36 ml/min/1.73m<sup>2</sup>, and uACR 205.3 mg/mmol. At 16 weeks, there was mean weight loss of 7.3% from baseline, with improvements in mean Hba1c (6.8%) and albuminuria (13.4%) and no impact on eGFR. Further, we observed major reductions in total daily insulin dose from 0-16 weeks. Whilst the drug was well tolerated with few adverse events, we responded to a drop in bicarbonate from week 0-4 (mean 23.2 to 20.1 mmol/L) by co-prescribing sodium bicarbonate.

For the kidney transplant cohort, in addition to the metabolic and kidney outcomes above, we reviewed impact on tacrolimus levels pre- and post-tirzepatide. Fifty transplant patients (30/50 males) were included with mean baseline data showing age of 56.6 yr, weight 107.7 kg, Hba1c 66.5 mmol/mol, eGFR 48 ml/min/1.73m<sup>2</sup>, uPCR 91.8 mmol/mg. Mean weight loss at 16 weeks was 6.9% from baseline whilst Hba1c decreased by 7.49%. An initial drop in eGFR from week 0-4 of 7.6% recovered to baseline at week 16 and the drug was generally well tolerated, although there was one episode of acute pancreatitis. There was evidence of

increased variability in tacrolimus levels following initiation of tirzepatide (coefficient of variation: pre- 18.32 vs. post- 22.83%,  $p < 0.05$  by t-test).

Tirzepatide induces weight loss and improved glycaemia comparable to published studies in both patients living with CKD3a-5A2-3 and those with a kidney transplant. In transplant patients, there was a significant decline in eGFR on initiation of the drug that recovered and there was a higher degree of tacrolimus level variability which may warrant close monitoring through specialist clinics. We demonstrate our experience of developing a service to support the prescribing of tirzepatide to patients with kidney disease.

TG3

## Systematic Review: Use of Granulocyte Colony-Stimulating Factor in Managing Neutropenia and Outcomes in Kidney Transplant Recipients

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TUESDAY - Moderated Poster Session, HALL Q, March 10, 2026, 16:00 - 17:00

### Introduction:

Kidney transplant recipients are vulnerable to developing neutropenia during the early post-transplant period. Neutropenia is often multifactorial with immunosuppressive medication, viral infection and antiviral or antibiotic therapy as leading causes. Neutropenia increases the risk of infection, hospitalisation, and mortality. Granulocyte Colony-Stimulating Factor (G-CSF) is widely used in oncology and haematology but is much less familiar to nephrologists. G-CSF may exert immune-modulatory effects that may increase the risk of graft rejection, particularly when immunosuppressive drugs are reduced or discontinued. Evidence on G-CSF use in kidney transplantation is limited, and its safety remains uncertain in this setting. This systematic review aims to evaluate the impact of G-CSF on clinical outcomes in KTRs, including graft rejection, graft failure, mortality, and other adverse effects.

### Methods:

A sensitive search strategy was applied to Medline, PubMed, Embase, and the Cochrane Library from inception to 30 June 2025, following PRISMA guidelines. Screening was performed independently by two reviewers, with full-text assessment conducted for potentially eligible studies. Data extraction captured study and population characteristics, neutropenia definitions, G-CSF dosing and administration protocols, and clinical outcomes. Quality assessment was performed using the Newcastle–Ottawa Scale. Due to marked heterogeneity in study design, neutropenia definitions, and outcome reporting, a meta-analysis was not feasible; a narrative synthesis was therefore conducted.

### Results:

Of the 1,487 citations screened, 12 retrospective cohort studies met the inclusion criteria (see table). There was substantial variability in neutropenia definitions and G-CSF regimens across studies. While several studies reported numerically higher rates of graft rejection among G-CSF-treated patients, these findings did not reach statistical significance. Similarly, outcomes such as graft failure and mortality varied, but showed no consistent or robust association with G-CSF use. Notably, some studies highlighted potentially concerning findings, including the development of donor-specific antibodies. Conversely, evidence suggested reduced hospitalisation rates in patients receiving G-CSF. Overall, the evidence base did not establish a significant link between G-CSF use and adverse transplant outcomes, although signals of possible immunological effects were observed.

### Discussion and Conclusion:

This review offers important insights into the use of G-CSF in kidney transplantation. While there were signals of increased DSA formation and numerically higher rates of rejection with G-CSF, the available evidence does not demonstrate a statistically significant association between G-CSF administration and adverse outcomes, including graft rejection, graft failure, or mortality.

Interpretation of these findings is limited by several factors: all included studies were retrospective in nature; there was wide heterogeneity in neutropenia definitions and G-CSF dosing protocols; and important confounders, such as concurrent treatment modifications, were often unaccounted for.

Our evidence is somewhat reassuring regarding the safety of G-CSF, but prospective, controlled studies are required to confirm our findings. Such trials are essential to delineate the safety profile of G-CSF in kidney transplant recipients and to identify patients at higher risk of adverse transplant outcomes such as rejection or DSA formation. Overall, the use of G-CSF after kidney transplantation requires further scrutiny, and nephrologists should work closely with haematologists to identify safer approaches for managing post-transplant neutropenia.

TG4

## Pre-transplant TTV titres: distribution, clinical correlates, and early trajectories of undetectable cases

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TUESDAY - Moderated Poster Session, HALL Q, March 10, 2026, 16:00 - 17:00

### Introduction

Torque Teno Virus (TTV) load is emerging as an important predictor of immunosuppression burden post-transplant. Pre-transplant titres may capture baseline set-points and influence early viral load kinetics. We describe the distribution and subgroup analyses of pre-transplant TTV viral load and characterise early post-transplant trajectories among patients with undetectable baseline titres.

### Methods

Serum TTV DNA (log copies/mL) was measured pre-transplant in 258 kidney recipients from the “TTV: A biomarker for Immunosuppression” study. Summary statistics were generated for the overall distribution. Kruskal–Wallis tests compared categorical subgroups (ethnicity, sex, diabetes, pre-emptive transplant, cause of ESKD, BMI group); Spearman correlation assessed age. Among patients with undetectable baseline TTV, longitudinal post-transplant values were summarised with a non-parametric smoother and 95% CIs for visualisation. All statistical analyses were done on R.

### Results

Pre-transplant TTV was widely distributed (mean 3.17, SD 1.46; median 3.37; range 0–9.01 log). Twenty-three patients (8.9%) were undetectable at baseline.

Subgroup analyses showed strong heterogeneity: ethnicity ( $p=1.05\times 10^{-7}$ ) with highest titres in Afro-Caribbean patients (median 4.03) and lowest in White patients (median 2.53); higher titres in males vs females ( $p=0.0023$ ; medians 3.53 vs 3.02); higher with diabetes ( $p=0.0153$ ; medians 3.74 vs 3.19); and lower in pre-emptive transplants vs those on dialysis ( $p=0.0273$ ; medians 3.02 vs 3.45). Cause of ESKD was associated with titre ( $p=0.0379$ ), with lowest values in cystic disease and highest in infection/malignancy. Age (Spearman  $p=0.49$ ) and BMI group ( $p=0.30$ ) showed no association.

All patients with undetectable baseline TTV developed detectable viraemia post-transplant. Their trajectories rose steeply during the first 10–12 weeks to approximately mid-6 log, then plateaued/slightly declined thereafter, aligning with expected kinetics under induction and early maintenance immunosuppression.

### Discussion

Pre-transplant TTV exhibits broad inter-individual dispersion and clinically relevant differences by ethnicity, sex, diabetes status, dialysis exposure, and ESKD aetiology, while no association was detected with age or BMI. An undetectable baseline viral load does not imply absence of subsequent viraemia; titres rise predictably after transplantation.

Limitations include single-centre, serum-only measurements and unequal subgroup sizes. Furthermore, subgroup comparisons are exploratory and unadjusted for multiplicity. These baseline data show TTV viral load reflects host and exposure factors; this needs to be replicated in multi-centre and multi-platform analyses.

#### Conclusion

Pre-transplant TTV titres are heterogeneous, with ~9% undetectable, and show clear demographic and clinical correlates. Patients with undetectable baseline titres demonstrate rapid early post-transplant increases. Baseline TTV may aid risk stratification and planning of early monitoring.

TG5

## Outcomes of renal transplantation in primary glomerulonephritis (GN) mapped to post-transplant proteinuria in the UK RaDaR registry

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**Background:** Renal transplant outcomes vary by primary renal diagnosis and are influenced by the development of proteinuria post transplantation. This study quantifies these relationships using the transplant outcome data from seven forms of GN and autosomal dominant polycystic kidney disease (ADPKD) in UK RaDaR.

**Methods:** RaDaR cohorts were identified with primary diagnoses of idiopathic nephrotic syndrome (INS), IgA nephropathy antineutrophil cytoplasmic antibody (ANCA) nephritis, membranoproliferative GN (MPGN)/C3 glomerulopathy, Alport syndrome, anti-GBM disease and membranous GN (PMN), with ADPKD as a non-GN comparator. The number of patients identified who had received a first renal transplant since 2005 was 6172, and 34% of these had proteinuria data available sufficient to quantify urine protein-to-creatinine ratio (UPCR) at 1 year post transplant. Table 1 shows key characteristics and demography at the time of transplantation and summary UPCR data at 1 year post transplant by diagnosis.

**Results:** Figure 1 shows outcomes of transplantation (survival without kidney failure or estimated glomerular filtration rate <10 mL/min/1.73 m<sup>2</sup>) for the eight groups; these vary by diagnosis with the highest rates of graft loss in the INS, MPGN and PMN groups. Figure 2 shows transplant survival for the total GN and ADPKD groups stratified by UPCR > or <0.5 g/g at 1 year post transplantation.

**Conclusion:** Transplant outcomes in GN vary by precise diagnosis and are generally worse than in ADPKD. Proteinuria at 1 year post transplantation is strongly correlated with long-term outcomes; values above 0.5 g/g are more frequent in certain GN and are associated with over 60% graft failure within 10 years. The mechanisms underlying these findings are being assessed in ongoing work.

TG6

## Risk factors for bacterial infections requiring hospital admission in the early and intermediate period after kidney transplantation.

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Infections in the early and intermediate period post-renal transplantation are well-recognised complications, often linked to surgical factors and intensive immunosuppression. While previous studies have examined general risk factors for post-transplant infections or hospital admissions, few have specifically addressed predictors for bacterial infection-related admissions and their outcomes. Our aim was to identify potential risk factors for severe infections requiring hospital admission in the first six months post-transplantation in order to better detect patients at increased risk. With this information, we hoped to be able to intervene at an earlier stage in their infection.

We audited all the patients who underwent a kidney transplant in a large, inner-city hospital, and then had their post-transplant follow-up locally. We identified potential infection risk factors for each patient, including age, obesity, diabetes status, and pre-transplant immunosuppression. Infection-related hospital admissions within 180 days post-transplant were documented, along with outcomes such as critical care admission, graft loss, or death. The association between each risk factor and infection occurrence was assessed using the appropriate statistical methods.

A total of 129 patients were included in the study. Their mean age was 52.7 (13.1) years and average BMI 27.2 (4.7) kg/m<sup>2</sup>. The most common cause of end-stage renal failure was IgA nephropathy (12.3%), followed by diabetic nephropathy (11.6%) and hypertension (11.6%). Thirty-nine (30%) patients developed an infection in the first 180 days post-transplant, with 57 individual episodes of infection. Diabetes was associated with a significantly higher risk of infection ( $p=0.0084$ ). A higher proportion of patients on pre-transplant haemodialysis developed an infection. There was no significant difference between the rate of infection occurring in patients receiving the high-risk immunosuppression protocol (27.6%) versus low-risk protocol (31%). Elevated BMI was also noted to be a risk factor for infection, but this did not meet statistical significance. Raised CRP at day 7 post-transplant ( $p=0.0046$ ) and on discharge ( $p=0.0183$ ) were both linked to a higher rate of infection leading to hospitalisation in the first six months following transplantation. The mean time to ureteric stent removal was 33 (16.7) days in the infection group, compared to 34.3 (14.2) days in the no infection group.

Of the infections, urinary tract infections were most common, followed by community-acquired pneumonias and infected collections. The median time between transplantation and diagnosis of infection was 24.5 (40.3) days. The median length of admission was 8 (7.6) days. Three episodes of infection led to a critical care admission with no episodes leading to graft loss or death.

The study showed diabetes, pre-transplant haemodialysis and higher BMI as risk factors for infection within 6 months post-kidney transplantation. Raised CRP at day 7 and on the date of discharge flagged patients at risk for subsequent infection. Time to ureteric stent removal was slightly longer than expected but this was not found to be a significant risk factor in our cohort. Despite infections requiring hospital admission in 30% of cases, there were no associated cases of graft loss or death.

TG7

## Single centre audit of vaccination status and respiratory viral infections in kidney transplant recipients

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### Introduction:

Kidney transplant recipients (KTRs) remain vulnerable to vaccine-preventable infections (VPIs), particularly respiratory viruses. The aim of this audit was to assess vaccination status and respiratory viral infection (SARS-CoV-2 and influenza A/B viruses) outcomes in a KTR cohort.

### Methods:

We have undertaken single centre, retrospective observational audit including adult (>18 years) kidney, simultaneous pancreas–kidney (SPK), and combined liver–kidney (CLK) transplant recipients between 1 January 2022 and 31 December 2023. Vaccination history was extracted from electronic patient records.

Outcomes were documentation of complete SARS-CoV-2 primary vaccination ( $\geq 2$  doses) and influenza vaccination. Respiratory viral infections (SARS-CoV-2 and influenza) were identified through virology department records and clinical notes, with hospitalisation, ICU admission, and mortality documented.

### Results:

Vaccination status of 1,633 transplant recipients was analysed: median age 54 years (IQR 43–64); 627 (38.4%) females; 1,088 (66.6%) recipients of deceased-donor transplant and 545 (33.4%) recipients of live-donor transplant. Ethnicity was 42.6% White, 18.4% Black, 19.8% other Asian, 1.6% Chinese, and 17.6% other ethnic group. Median time since transplant was 2,543 days (IQR 1,106–4,619).

A total of 1,472 (90.1%) had completed the primary schedule, 22 (1.4%) had an incomplete schedule, and 139 (8.5%) were unvaccinated. Median age was 55 years (IQR 44–64) in the fully vaccinated group and 48 years in both incompletely vaccinated and unvaccinated groups. Females comprised 38.2%, 31.8%, and 41.7% of each group, respectively.

During 2023, 430/1,633 patients (26.3%) underwent respiratory virus testing; 44 patients (11.4%) had confirmed infection: 38 (8.8%) SARS-CoV-2 and 11 (2.6%) influenza, including one dual infection. Hospitalisation occurred in 39/44 (88.6%) participants with a complete primary vaccination schedule and 5/5 (100.0%) unvaccinated participants. No ICU admissions or mortality among hospitalised patients were observed during the audit period.

### Discussion:

Kidney transplant recipients should be encouraged to remain up to date with recommended vaccinations to reduce infection risk and protect graft function. Audit results highlight the

need to develop a standardised tool to track and record vaccination history for transplant patients as part of routine follow-up proformas.

A collaborative approach between transplant and infectious diseases team should be considered, including whether transplant candidates might benefit from pre-transplant ID consultation. Stratification of vaccine responses could help identify patients at risk of suboptimal immunity.

TG8

## Incidence of major adverse cardiovascular events and cardiovascular mortality in kidney transplant recipients: A systematic review and meta-analysis

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### Background:

Cardiovascular (CV) disease remains the leading cause of death in patients with chronic kidney disease and continues to drive morbidity and mortality even after successful kidney transplantation. The incidence of major adverse cardiovascular events (MACE) and CV mortality in kidney transplant recipients (KTRs) has been reported inconsistently across studies.

### Methodology:

We systematically searched PubMed, Embase, Scopus, and Web of Science for observational cohorts and clinical trials reporting MACE and/or CV mortality in adult KTRs. Sixteen studies including approximately 22,387 patients were eligible. The primary endpoint was pooled incidence of MACE, defined as myocardial infarction, stroke, heart failure, or CV death. The secondary endpoint was CV mortality. Random-effects meta-analyses with Freeman-Tukey transformation were performed. Study quality was assessed using the Newcastle-Ottawa Scale.

### Results:

The pooled incidence of MACE was 13% (95% CI: 10–17%) across 16 studies, with individual study estimates ranging from 2% to 32% ( $I^2 = 98.5\%$ ). Subgroup analysis of five studies (5,791 patients) reporting CV mortality showed a pooled incidence of 8% (95% CI: 4–12%), with substantial heterogeneity ( $I^2 = 93.1\%$ ). Most included studies were of high methodological quality. See figure 1.

### Conclusions:

KTRs face a substantial burden of CV morbidity and mortality, with more than one in ten experiencing MACE and nearly one in twelve dying from CV causes post-transplant. These findings underscore the urgent need for systematic CV risk stratification, targeted preventive strategies, and tailored management protocols in the transplant population.

TG9

## TTV viral load trajectories after kidney transplantation

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### Introduction

Torque Teno Virus (TTV) load is an emerging pharmacodynamic biomarker after kidney transplantation. We characterised early post-transplant kinetics from zero to six months in the TTV: A biomarker for Immunosuppression study and explored whether patterns differ across clinical subgroups.

### Methods

Pre-transplant serum and serial post-transplant plasma TTV DNA were quantified (log copies/mL) in 268 kidney recipients at protocol weeks 0, 2, 4, 6, 8,12,18 and 24. The distributions were summarised by interval; changes between successive timepoints were tested with Wilcoxon rank-sum tests (not all samples were paired). Subgroup differences (sex, ethnicity, diabetes, pre-emptive transplant, ESKD aetiology, BMI, induction type) used Kruskal–Wallis tests at each timepoint. Smoothed cohort trajectories were displayed with a non-parametric smoother and 95% CIs for visualisation only.

### Results

Before transplant, median TTV was 3.37 log (IQR 1.76; n=258). After transplant, medians increased steadily to 6.42 log at week 12 (IQR 2.07), then did not differ at weeks 18 and 24 (p=0.85 and 0.68), indicating a plateau. The largest step occurred between weeks 8 to12 ( $\Delta$  median +1.10 log; p<0.001). All measured samples were detectable by week 12.

A density plot showed the highest observation density around weeks 4–8 at approximately 4.5–5.5 log, with progressive dispersion thereafter.

Subgroup analyses revealed earlier ( $\leq 8$  weeks) differences by sex, ethnicity, diabetes, pre-emptive status, ESKD aetiology and induction type, which attenuated by week 12. The induction effect persisted: recipients given cytotoxic induction had higher titres at week 18 (p=0.0229). BMI groups showed no consistent separation.

### Discussion

TTV rises predictably after transplant, peaking around week 12 then flattening, with early heterogeneity that largely converges by three months—except for a sustained induction signal. The steady increase and plateauing of TTV viral load post-transplant, and the difference between induction agents are consistent with earlier studies. Limitations: single-centre, serum-only baseline and plasma follow-up, incomplete pairing across visits, and multiple exploratory subgroup tests.

### Conclusion

Post-transplant TTV shows a rapid early rise to a week-12 plateau, universal detectability by week 12, and transient subgroup differences with a persistent elevation after cytotoxic

induction. These kinetics provide practical guidance for timing and interpretation of early TTV monitoring.

TG10

## A systematic review and meta-analysis of major adverse cardiovascular events and mortality following renal transplantation

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**Introduction:** Renal transplantation is the optimal method of renal replacement therapy in end-stage renal disease. These patients are believed to be at high risk of major adverse cardiovascular events (MACE) following transplantation, including stroke, myocardial infarction, or acute heart failure, but the reported rates are variable. The aim of this systematic review and meta-analysis is to evaluate the incidence of perioperative MACE and mortality following renal transplantation.

**Methods:** Cochrane, Google Scholar, ISRCTN, US Clinical Trials, and PubMed databases were searched until April 2024. Observational or interventional studies reporting the incidence of MACE and death of any cause after kidney transplantation were selected. Incidence data were pooled using the random-effects model. Heterogeneity was measured using an  $I^2$  statistic and was further explored using subgroup analysis. Publication bias was evaluated using funnel plots.

**Results:** Of the initially identified 216 studies, six studies met the inclusion criteria and followed up patients for 30 days. At 30-day follow-up, the pooled incidence of MACE was 3% (95% CI 1%-9%,  $p < 0.01$ ), and the pooled incidence of death was 1% (95% CI 0.2%-4%,  $p < 0.01$ ). For both analyses, the studies were shown to be highly heterogenous with  $I^2$  being  $> 95\%$ . When stratifying studies by data source, single-centre studies looking at MACE had an incidence of 8% (95% CI 1%-35%,  $p = 0.15$ ) with an  $I^2$  statistic of 52%, compared to database studies showing a pooled incidence of 3% (95% CI 0.3%-18%,  $p < 0.01$ ) with an  $I^2$  statistic of 99.8%. Database studies looking at mortality had a pooled incidence of 1% (95% CI 0.09%-11%,  $p < 0.01$ ), with an  $I^2$  statistic of 98%. There was only one single-centre study assessing mortality at 30 days. Leave-one-out analysis did not result in significant improvements to  $I^2$ , and pooled proportions remained similar.

**Discussion:** This analysis demonstrates that MACE and death outcomes are generally low in kidney transplant surgery. However, the results are highly heterogenous, likely due to a discrepancy in study design and pre-transplant screening evaluation, as well as a lack of clear definition of MACE. This highlights the need for more consistent pre-transplant workup and reporting of MACE outcomes of transplant patients.

TG11

## Human Metapneumovirus (HMPV) infection in Kidney Transplant Recipients: A Systematic Review

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TUESDAY - Moderated Poster Session, HALL Q, March 10, 2026, 16:00 - 17:00

### Introduction:

Human Metapneumovirus (HMPV) infection poses significant risks to kidney transplant recipients on immunosuppression.

While infection is typically self-limiting in healthy individuals, immunosuppression may increase its severity. The effect of HMPV within context of Kidney Transplantation is an intriguing topic & whilst there is gradual recognition of the infection within our transplant cohort there remains limited guidance detailing clinical features, effectiveness of current management strategies and how immunosuppression influences disease course in kidney transplant. This systematic review aims to address this knowledge gap by summarising the impact on outcomes and treatment strategies of HMPV infection in kidney transplant recipients

### Methods:

This study is registered with PROSPERO (CRD42025651994) A comprehensive literature search was conducted (Jan 2005-Jan 2025) in PubMed, EMBASE, Cochrane Library, and Medline databases using keywords such as "Human Metapneumovirus," "HMPV," "Kidney transplant," "Renal transplant," and "Immunosuppression." Eligibility criteria included studies reporting on HMPV prevalence, clinical features and outcomes in kidney transplant recipients. Duplicates were removed, and studies underwent two-phase screening by independent reviewers F.M and O.S. Data were extracted on study design, demographics, clinical features, treatments, and outcomes. PRISMA guidelines were followed (Figure-1).

### Results:

Of 13,556 studies screened, 7 full text publications were included after the exclusion of duplicates and based on relevance in the final review (Table 1)[3-18], Human metapneumovirus (HMPV) infection in renal transplant recipients shows highly variable severity, ranging from mild self-limiting illness to respiratory failure. Across studies, ICU admission rates ranged from 3–26%, with mortality up to 35% at one year. Risk factors included higher corticosteroid doses (>10 mg/day) and in severe cases reduction in immunosuppression was employed. Management varied, including ribavirin, intravenous immunoglobulin, adjustment of immunosuppression, and supportive care; however, no

antiviral has proven efficacy. Graft rejection occurred in some cases, but graft loss was uncommon. No licensed vaccine exists, though candidates are in development.

Discussion:

HMPV is a significant but under-recognised risk to kidney transplant recipients with emergence of cases at a global scale since late 2024 and outcomes influenced by immunosuppressive burden, co-morbidities and early recognition.

While most cases recover, a subset develop severe disease, ICU admission, and allograft complications. Current management is largely supportive, with variable use of ribavirin and immunosuppression adjustment, but robust evidence for antiviral benefit is lacking. There is currently no vaccine but development is underway and future research is likely to evaluate targeted anti-virals and vaccines to reduce morbidity and protect allograft function in this vulnerable group.

TG12

## Are we using SGLT2i and GLP1A in post transplant diabetes management?

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TUESDAY - Moderated Poster Session, HALL Q, March 10, 2026, 16:00 - 17:00

### Introduction

The high cardiovascular risk associated with kidney disease and transplantation is well established. The benefits of sodium-glucose co-transporter-2 (SGLT2) inhibitors and glucagon-like peptide-1 (GLP-1) receptor agonist have been reported and although no randomised control trials exist in transplantation the benefit of these agents are also emphasised in reducing cardiovascular risk and are being increasingly used in the transplant community. This study aimed to determine the proportion of kidney transplant recipients (KTR) with diabetes who are on sodium-glucose co-transporter-2 inhibitors (SGLT2i) and glucagon-like peptide-1 receptor agonists (GLP-1A). While also identifying patient or clinical factors associated with persistent use of insulin.

### Methods

A retrospective cohort study of all adult KTR with diabetes in May 2025 were identified using renal cv5 database. Demographic, clinical, biochemical and immunosuppression data were collected. Those with Type 1 Diabetes Mellitus (T1DM) were excluded. Patients in their acute hyperglycaemia phase < 3months post-transplant were also excluded. Out of this cohort it was then assessed whether patients were on a SGLT2i or GLP-1 inhibitor if another agent was needed. Glycaemic control was measured by glycated haemoglobin (HbA1c) at baseline and post-transplant follow-up.

### Results

151 KTR with diabetes (45 T2DM, 106 PTDM) were included. Those on SGLT2i T2DM KTR 16% (7/45) compared to 26% (28/106). Males on SGLT2i in T2DM KTR and PTDM KTR were 4% and 17% respectively illustrating a preponderance of females being prescribed these agents more. Median age of T2DM KTR on SGLT2i 73 years (62-75) vs PTDM KTR median 59 years (51-69). White and Asian PTDM KTR were had a higher proportion of patients on SGLT2i however there were no ethnic differences in SGLT2i in T2DM KTR. PTDM KTR had significantly better HbA1C control (p=0.04). There was no significant difference in eGFR Table 1.

GLP1A were given to 2% (1/45) T2DM KTR compared to 3% (3/106). No males were on GLP1A in T2DM KTR with a single male in PTDM KTR group again illustrating a preponderance of females being prescribed these agents more. Median age of T2DM KTR on SGLT2i 50 years vs PTDM KTR median 62 years (59-68). There was no ethnic differences

in GLP1A medications across either T2DM or PTDM KTR groups. PTDM KTR had significantly better HbA1C control ( $p < 0.01$ ). There was no significant difference in eGFR Table 2.

#### Discussion

Uptake of SGLT2i and GLP1A with established cardiovascular and renal benefits was low however PTDM KTR were prescribed these agents more frequently than those with T2DM. This emphasises the need for a more systematic approach of reviewing T2DM KTR medications who often have blurred lines of responsibility of who makes changes to their diabetic medications post-transplant exist. PTDM KTR are newly diagnosed and are more consistently commenced on these agents albeit overall the number of KTR on these agents continues to be low. A prescribing pharmacist review may help increase the number of KTR on these cardiorenal metabolic protective agents to optimise medications.