Reply to Reviewer 3:

1.In the revised article, *Staphylococcus aureus* and *coagulase-negative staphylococci (CoNS)* are presented separately in the footnotes to Table 2 in the Results section. Two of the seven Staphylococcus isolates were identified as S. aureus, and five as CoNS. We also added an explanation of their clinical significance. While S. aureus bacteriuria in patients with catheters is considered clinically significant, CoNS isolates were most often interpreted as possible contaminants, consistent with previous reports. This distinction increases the accuracy and interpretability of our microbiological data.

2. In the revised *Methods* section, we have specified the EUCAST version and year used for antimicrobial susceptibility testing (EUCAST v13.0, 2023). We have also listed the antibiotic panels tested for the main bacterial groups (*E. coli*, *Klebsiella spp.*, *Enterococcus spp.*, *Pseudomonas aeruginosa*, *Staphylococcus spp.*). Furthermore, we clarified that resistance percentages represent only the isolates tested for each antibiotic (n/N), and this has been explicitly indicated in the table footnotes. Regarding interpretive categories, we used the EUCAST S/I/R system and combined *susceptible (S)* and *increased exposure (I)* as "susceptible" in descriptive statistics, as recommended by EUCAST reporting guidelines. A statement clarifying this handling has been added to the *Methods* section for transparency.

3. To enhance clarity and ensure proper interpretation of resistance mechanisms, the main resistance table (Table 2) has been reformatted to present two visually distinct sections: Gram-negative (E. coli, Klebsiella spp., Pseudomonas aeruginosa, Acinetobacter spp.) and Gram-positive (Enterococcus spp., Staphylococcus spp.) bacteria. ESBL and KPC reporting is now limited to Gram-negative organisms, consistent with their underlying β-lactamase-mediated resistance mechanisms. Additionally, the Methods section has been updated to specify the confirmatory procedures used. Extended-spectrum β-lactamase (ESBL) production was confirmed phenotypically using combination disk tests with clavulanic acid, while carbapenemase activity (including KPC and OXA-48) was evaluated using inhibitor-based tests in accordance with EUCAST recommendations.

4. In the revised manuscript, we have clarified the actual resistance percentages for each compared group (e.g., first year vs. subsequent years, single vs. recurrent infections) within the Results section to enhance interpretability. Although odds ratios and 95% confidence intervals were not calculated due to limited subgroup sizes and unequal group distributions, we acknowledge this as a limitation and have explicitly stated it in the Limitations paragraph of the Discussion. The revised text now emphasizes that the reported p-values indicate statistical associations rather than precise effect magnitudes.

5. Data on prior antibiotic exposure (both for prophylaxis and treatment of previous infections) were not systematically recorded in our dataset; therefore, we were unable to analyze the correlation between prior antibiotic use and the emergence of resistance. We agree that such data would provide valuable insights into the dynamics of resistance development among kidney transplant recipients. This point has been acknowledged in the *Discussion* section as an additional limitation, and future studies including detailed antimicrobial exposure histories are warranted to better clarify these associations.

6. We fully agree that multiple episodes per patient introduce within-subject correlation and that mixed-effects or GEE models would be appropriate in larger datasets. However, given our modest sample (123 patients, 363 episodes) and small, unbalanced subgroups, fitting adjusted mixed/GEE models would likely yield unstable estimates and wide confidence intervals. For this reason, we reported group-level comparisons using chi-square/Fisher's exact and appropriate tests, and we explicitly acknowledge this as a limitation. To address the reviewer's concern transparently, we have expanded the Limitations to note that mixed-effects/GEE modeling and adjusted effect estimates (with 95% CIs) were not performed and that the ≈85% female predominance may act as a potential confounder. We agree that future multicenter studies with larger cohorts should employ mixed-

effects/GEE models adjusting for sex, age, diabetes, donor type, and device/prophylaxis status to provide adjusted effect sizes with confidence intervals.

7. In the revised manuscript, the term catheterization has been clarified to refer primarily to temporary urinary devices used in the early postoperative period, such as Foley catheters and ureteral stents, which were typically removed within 7–14 days after transplantation. A small number of patients required indwelling urinary catheters or nephrostomy tubes due to postoperative complications (e.g., urinary leak, ureteral obstruction). However, detailed data on the duration and number of these devices were not available; therefore, stratified analysis of resistance by device presence was not performed. This clarification has been added to the Methods section.

8. We thank the reviewer for this important linguistic and ethical correction. In the revised manuscript, the term *"cadaver donor"* has been replaced with *"deceased donor"* throughout, in accordance with current international transplant terminology and ethical guidelines. The phrase *"patients in clinical death"* has also been removed to avoid ambiguity. All references now clearly differentiate between *deceased* and *living donors*. Information on the type of deceased donation (DBD vs. DCD) was not available in our dataset and has therefore not been specified.

9. The Methods section has been revised to specify our center's perioperative and prophylactic antimicrobial protocols. Perioperative prophylaxis at our institution consists of a single preoperative dose of ceftiaxon (2 g IV) administered within 60 minutes before incision, with no postoperative continuation unless clinically indicated. All kidney transplant recipients routinely receive trimethoprim–sulfamethoxazole (TMP–SMX) prophylaxis (80/400 mg orally once daily) for at least six months post-transplantation to prevent Pneumocystis jirovecii pneumonia and recurrent UTIs.We have also noted in the Discussion that our lower ESBL rates may partly reflect the limited perioperative antibiotic exposure and systematic use of TMP–SMX prophylaxis, which is consistent with current European and KDIGO recommendations.