

Competing Death and Absolute Benefit in Dexmedetomidine-Exposed Patients with Dialysis-Requiring Acute Kidney Injury [Letter]

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Dear editor,

Hung et al¹ should be commended for addressing a neglected question in dialysis-requiring acute kidney injury (AKI-D): whether dexmedetomidine exposure after AKI is associated with durable survival and renal benefit. Still, two analytic issues deserve discussion before the results are translated into sedation practice.

The renal endpoints require closer handling of death as a competing event. In Table 2, dexmedetomidine was associated with lower 2-year mortality, 634 of 6354 patients (10.0%) versus 925 of 6354 (14.6%), HR 0.64. The same table reports fewer ESRD events, 895 (14.1%) versus 1029 (16.2%), HR 0.80, and fewer eGFR declines below 30 mL/min/1.73 m², 1231 (19.4%) versus 1384 (21.8%), HR 0.83. Figure 3 also shows early separation of mortality and ESRD curves after the landmark. Yet death precludes later observation of ESRD or laboratory-defined eGFR decline. If deaths were treated as simple censoring in Cox models, the estimated cause-specific hazard may not answer the bedside-relevant question: what is the actual cumulative probability of ESRD before death? Dexmedetomidine survivors have more time at risk for ESRD, whereas control patients may disappear from the renal-risk set through death. Recent AKI-D work reinforces this link: in a 6703-patient cohort, post-AKD kidney function was associated with mortality, major adverse cardiac events, ESKD, and readmission.² The SALTO follow-up also found substantial late kidney morbidity among severe-AKI survivors, including worsening renal function in 46 of 175 patients with available long-term kidney data and chronic dialysis in 5% of day-90 survivors.³ A Fine-Gray subdistribution model, cumulative incidence functions for ESRD with death as a competing event, or a sensitivity analysis using composite MAKE-type endpoints would make the renal inference more transparent.⁴

Besides, the magnitude of benefit also needs absolute framing. The mortality hazard ratio of 0.64 appears striking, but the absolute 2-year mortality difference was 4.6%, giving an approximate number needed to treat of 22 if causality holds. For ESRD, the absolute difference was 2.1%, approximate NNT 48; for eGFR decline, 2.4%, approximate NNT 42. These are potentially meaningful, but not self-interpreting. Reporting risk differences with confidence intervals, NNTs, and safety outcomes such as bradycardia, hypotension, and vasopressor escalation would help clinicians judge net benefit.

This study opens an important door. Competing-risk renal analyses, absolute-effect summaries, and dose/timing safety data would strengthen the case for a randomized trial and protect readers from over-reading a powerful association. We thank Hung et al for advancing this timely AKI-D discussion.

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Disclosure

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