Intensive Care Med
https://doi.org/10.1007/s00134-025-08028-3

EDITORIAL

Kidney replacement therapy in critically ill obese patients: an urgent call for precision and evidence-based practice!

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The global rise in obesity presents new and complex challenges in critical care [1, 2]. Obesity is a well-established risk factor for chronic kidney disease (CKD), end-stage renal disease [3], and increased mortality [4]. However, the association between obesity and mortality is not straightforward. Paradoxically, numerous studies have described an "obesity paradox," where overweight and moderately obese patients—especially with comorbidities—often show better outcomes than those with normal or low body mass index [5]. The obesity paradox refers to increased disease risk from obesity, yet paradoxically improved survival [5]. This paradox appears not only in CKD and end-stage renal disease but also in heart failure, coronary artery disease, pneumonia, and acute respiratory distress syndrome [5, 6].

CKD pathophysiology in obesity is multifactorial, involving glomerular hyperfiltration, increased sodium reabsorption, and elevated metabolic and oxygen demands, which contribute to glomerulomegaly and focal segmental glomerulosclerosis [5–7]. Intriguingly, the obesity paradox has also been described in acute kidney injury (AKI), with studies suggesting better survival in obese AKI patients [8].

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This comment refers to the article available online at https://doi.org/10.1007/s00134-025-07990-2.

What are the study's main findings and their interpretation in light of the current literature?

In a recent issue of *Intensive Care Medicine*, Dr. Monet and colleagues present compelling evidence from a 15-year retrospective cohort study evaluating kidney replacement therapy (KRT) in critically ill obese patients [1]. Among the key findings, the authors report an independent association between KRT and increased 90-day mortality in obese ICU patients, despite a significant decline in AKI incidence over time. This decline contrasts with some contemporary data [5] and may represent a second paradox. They state it may reflect improved care and AKI prevention over the past 15 years [1]. Although AKI incidence declined, the proportion of patients requiring KRT remained stable, suggesting that while milder AKI cases are better managed or prevented, the most severe forms of AKI—those requiring dialysis—have not followed the same trend. The study also addresses the ongoing debate on KRT dosing in obese patients. Some experts support for dosing based on actual body weight, citing increased urea distribution in obesity,while others caution against against it due to limited urea diffusion into adipose tissue[9]. A recent retrospective study of 1033 AKI patients found that indexing continuous KRT dose to actual or adjusted body weight was associated with increased mortality, whereas using ideal body weight was not. Moreover,. These findings support using ideal body weight as a potentially modifiable strategy to mitigate risk in obese patients [10]. Another important contribution of Dr. Monet's study is its focus on long-term renal outcomes in obese ICU survivors. About 10% of those who underwent KRT remained dialysis-dependent one year after ICU discharge [1]. This aligns with the broader literature, though recent studies

show mixed results on renal recovery. In one pilot study, obesity did not significantly affect 6-month renal recovery in patients with or without pre-existing CKD [11].The results are surprising and against the current knowledge [5]. However, whether this holds true in the post-KRT setting remains to unclear.

Study strengths and limitations

This study offers real-world insights into a critically underexplored population.. Its strengths include a large sample size, long observation period, and rigorous statistics. However, some limitations exist.Notably,the lack of a non-obese control group limits external comparisons. Key clinical variables—such as delivered dialysis dose (particularly relevant in obese patients), anticoagulation strategies, timing of KRT initiation, and modality-specific complications—were not consistently captured or analyzed. The single-center design, along with a high prevalence of cirrhosis, may also limit generalizability.

Future research directions

This study raises several questions that future research should address. Foremost among them (Fig. 1):

- What is the optimal KRT dose for obese patients: should we use ideal body weight, actual body weight or adjusted body weight?
- How does AKI incidence differ between obese and non-obese patients?

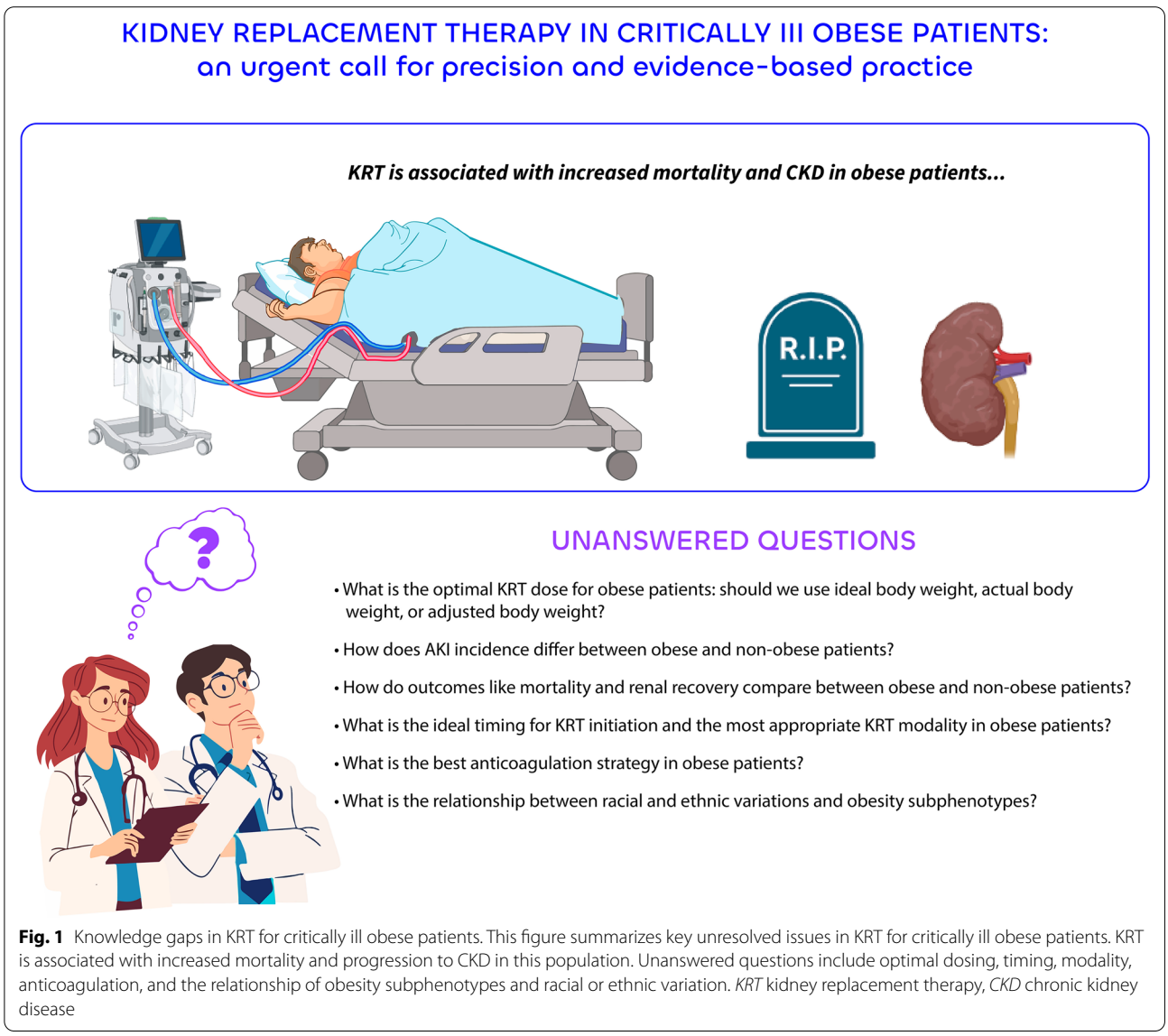

Fig. 1 Knowledge gaps in KRT for critically ill obese patients. This figure summarizes key unresolved issues in KRT for critically ill obese patients. KRT is associated with increased mortality and progression to CKD in this population. Unanswered questions include optimal dosing, timing, modality, anticoagulation, and the relationship of obesity subphenotypes and racial or ethnic variation. *KRT* kidney replacement therapy, *CKD* chronic kidney disease

- How do outcomes like mortality and renal recovery compare between obese and non-obese patients?
- What is the ideal timing for KRT initiation and the most appropriate KRT modality in obese patients?
- What is the best anticoagulation strategy in obese patients?
- What is the relationship between racial and ethnic variations and obesity subphenotypes?

In addition to randomized controlled trials mechanistic studies are needed to clarify the obesity paradox and the pathophysiological progression from AKI to CKD in this population. In a recent retrospective study, AKI risk was higher in obese patients but not in the most severely ill [12]. Another paradox was that AKI development was associated with higher mortality in normal-weight and overweight, but not in obese patients [12]. Another paradox was that AKI was linked to higher mortality in normal-weight and overweight patients, but not in obese patients [13]. The longstanding issue of indexing urinary output criteria for AKI remains a major barrier to reproducible research in obese patients.Racial and ethnic variations also warrant further study. For example, an earlier cohort study found Asian patients lacks a survival advantage at higher body mass index, with some evidence of increased mortality [14]. This underscores the need for diverse, multicentric studies that reflect population heterogeneity. Furthermore, growing evidence highlights the existence of distinct obesity phenotypes, and accounting for this heterogeneity remains a major challenge in large-scale studies [15].

Conclusions

Obesity, AKI, and KRT intersect in a complex and understudied clinical landscape. The work by Dr. Monet and colleagues serves as a crucial wake-up call. Their findings highlight not only the vulnerability of critically ill obese patients requiring KRT but also the many unknowns that continue to surround optimal care in this population. Now is the time to launch well-designed randomized controlled trials and mechanistic studies to address these knowledge gaps and ultimately improve outcomes for this growing high-risk population.

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Funding
None.

Declarations

Conflicts of interest
PMH, AJ and HYL report no conflicts of interest related with the content of this editorial.

Ethical approval
PMH, AJ and HYL declare that the writing of the Editorial was fair, balanced, and grounded in truth. We declare that we have done an accurate reporting and did respect ethical standards in the selection and presentation of information.

Publisher's Note

Received: 20 June 2025 Accepted: 1 July 2025
Published online: 14 July 2025

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