SYSTEMATIC REVIEW

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Temporal Relationship and Clinical Outcomes of Acute Kidney Injury Following Acute Respiratory Distress Syndrome: A Systematic Review and Meta-Analysis

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OBJECTIVES: Conduct a systematic review and meta-analysis to assess prevalence and timing of acute kidney injury (AKI) development after acute respiratory distress syndrome (ARDS) and its association with mortality.

DATA SOURCES: Ovid MEDLINE(R), Ovid Embase, Ovid Cochrane Central Register of Controlled Trials, Ovid Cochrane Database of Systematic Reviews, Ovid PsycINFO database, Scopus, and Web of Science thought April 2023.

STUDY SELECTION: Titles and abstracts were screened independently and in duplicate to identify eligible studies. Randomized controlled trials and prospective or retrospective cohort studies reporting the development of AKI following ARDS were included.

DATA EXTRACTION: Two reviewers independently extracted data using a pre piloted abstraction form. We used Review Manager 5.4 software (Cochrane Library, Oxford, United Kingdom) and Open Meta software (Brown University, Providence, RI) for statistical analyses.

DATA SYNTHESIS: Among the 3646 studies identified and screened, 17 studies comprising 9359 ARDS patients met the eligibility criteria and were included in the meta-analysis. AKI developed in 3287 patients (40%) after the diagnosis of ARDS. The incidence of AKI at least 48 hours after ARDS diagnosis was 20% (95% CI, 0.18–0.21%). The pooled risk ratio (RR) for the hospital (or 30-d) mortality among ARDS patients who developed AKI was 1.93 (95% CI, 1.71–2.18). AKI development after ARDS was identified as an independent risk factor for mortality in ARDS patients, with a pooled odds ratio from multivariable analysis of 3.69 (95% CI, 2.24–6.09). Furthermore, two studies comparing mortality between patients with late vs. early AKI initiation after ARDS revealed higher mortality in late AKI patients with RR of 1.46 (95% CI, 1.19–1.8). However, the certainty of evidence for most outcomes was low to very low.

CONCLUSIONS: While our findings highlight a significant association between ARDS and subsequent development of AKI, the low to very low certainty of evidence underscores the need for cautious interpretation. This systematic review identified a significant knowledge gap, necessitating further research to establish a more definitive understanding of this relationship and its clinical implications.

KEYWORDS: acute kidney injury; acute respiratory distress syndrome; kidney failure; meta-analysis; respiratory failure; systematic review

Acute kidney injury (AKI) and acute respiratory distress syndrome (ARDS) are frequently encountered in critically ill patients and are associated with significant morbidity and mortality rates (1–4). Combination of ARDS and AKI nearly doubles the mortality rate (5). Clinical

DOI: 10.1097/CCE.0000000000001054

KEY POINTS

Question: What is the incidence of acute kidney injury (AKI) after acute respiratory distress syndrome (ARDS)? Does ARDS increase the risk of AKI and is it associated with increased mortality?

Findings: AKI frequently occurs after ARDS, increasing mortality risk. ARDS independently raises AKI risk, and AKI post-ARDS independently raises mortality risk. A knowledge gap exists in current literature regarding the AKI-ARDS temporal relationship.

Meaning: ARDS patients should be closely monitored for AKI, as its development independently worsens outcomes. Further research is needed to elucidate the temporal dynamics of AKI and ARDS for improved patient care.

and experimental evidence has highlighted the significant interplay between the lungs and kidneys, indicating a complex lung-kidney "crosstalk" (4). However, despite accumulating evidence suggesting the interaction between ARDS and AKI, a significant knowledge gap remains regarding the temporal relationship between AKI and ARDS and their impact on clinical outcomes. Only few studies have investigated the timing of AKI in relation to the ARDS onset. Understanding the temporal relationship between AKI and ARDS might be important for comprehending their pathogenesis, identifying appropriate treatment options, and improving management for critically ill patients.

We conducted a systematic review and meta-analysis adhering to rigorous methodological standards to address: 1) the incidence of AKI following the diagnosis of ARDS and 2) the impact of the timing of AKI development after ARDS diagnosis on patient mortality.

MATERIALS AND METHODS

We conducted this systematic review according to a prespecified protocol registered and published at the PROSPERO (https://www.crd.york.ac.uk/PROSPERO/, identification number : CRD42023387744), and this review was reported per Preferred Reporting Items of Systematic Reviews and Meta-Analysis statement (6).

Information Sources and Search Strategies

We conducted a comprehensive search in various databases until April 7, 2023, for English-language papers. Databases included Ovid MEDLINE(R), Epub Ahead of Print, In-Process & Other Non-Indexed Citations, Daily, Embase, Cochrane Central Register of Controlled Trials, Cochrane Database of Systematic Reviews, PsycINFO database, Scopus, and Web of Science. An experienced librarian designed the search strategy with input from the principal investigator. Controlled vocabulary and keywords were used to search for studies on ARDS and AKI in adult patients (**Supplemental Material 1**, http://links.lww.com/CCX/B307). The search strategy was restricted to English language to ensure the accuracy of the data interpretation. We did not include gray literature in our search strategy. This decision was made to focus on peer-reviewed and published studies to ensure a high standard of scientific rigor in our analysis. The search strategy was not peer-reviewed by a second research librarian. However, it was meticulously developed and reviewed within our research team, ensuring adherence to systematic review standards and best practices.

Eligibility Criteria and Selection Process

Randomized control trials and observational studies were eligible for inclusion. Studies were deemed eligible if they reported on the incidence of AKI following the diagnosis of ARDS in critically ill adult patients (≥ 18 yr old). Furthermore, we examined patient mortality based on the timing of AKI development, specifically after the ARDS diagnosis, and categorized it as early vs. late AKI development post-ARDS. Outcomes of interest were: 1) incidence of AKI after the diagnosis of ARDS, 2) mortality of patients who developed AKI after ARDS, 3) timing of developing AKI after ARDS, and 4) mortality based on the timing of developing AKI defined as AKI before the diagnosis of ARDS vs. after diagnosis ARDS and early AKI vs. late AKI after ARDS. "Early AKI" was defined as AKI occurring within (specific time frame, e.g., 48 hr) after the diagnosis of ARDS. In contrast, "Late AKI" was considered as AKI developing after this initial period. The determination of the timing of AKI was based on the data available from the included studies. We chose a 48-hour cutoff for early vs. late AKI development as creatinine elevation, a key indicator of AKI, typically manifests within this time frame post-ARDS. This timeframe aids in

distinguishing AKI as either a consequence of ARDS or as part of a concurrent pathologic process. We extracted information regarding the time of AKI onset from each study and categorized them accordingly. We excluded studies that did not specify the temporal relationship of AKI with ARDS, studies that reported only development of AKI before ARDS or concomitant occurrence of AKI with ARDS. AKI was classified as concomitant if it was diagnosed on the same day as ARDS or within 24 hours of ARDS diagnosis. We included studies only if they defined AKI by (Kidney Disease Improivng Global Outcmes, Risk, Injury, Failure, Loss of kidney function, and End-stage kidney disease, or Acute Kidney Injury Network criteria (7–9). We excluded studies that did not clearly indicate the diagnosis of ARDS, either through explicit wording or by using one of the accepted definitions for ARDS (10, 11). Studies that only included patients with respiratory failure or patients on mechanical ventilation or extracorporeal membrane oxygenation with no clarification that those patients had ARDS were also excluded. We excluded case reports, reviews, thesis, books, editorials, author responses, letters, comments, conference abstracts, guidelines, letters, notes, book chapters, surveys, and protocols/registries (ongoing study) with no available results.

Data Collection and Analysis

Two reviewers (H.H.T., N.N.) independently reviewed and adjudicated the potential eligibility of each abstract generated by the search strategy. Full-text articles were obtained unless both reviewers unanimously determined an abstract to be ineligible. Subsequently, each full-text report underwent individual assessment for final study inclusion. Disagreements were resolved by consensus. We used κ statistics to measure agreements.

We extracted data using a standardized form. We abstracted study identifiers (authors, journal, country, year of publication, and characteristics [study design, setting, sample size, inclusion and exclusion criteria, and definitions of AKI and ARDS]), participant information (demographics, comorbidities, and severity of disease), and outcomes (mortality among those with ARDS with and without AKI, timing of AKI and ARDS development, AKI stage, and temporal relation of AKI with ARDS to divide the enrolled patients into three groups of AKI before ARDS, AKI after ARDS, and concomitant AKI and ARDS). Two reviewers (M.C., H.H.T.) assessed extracted data and resolved the disagreement with consensus. We contacted authors of the studies to obtain missing data. We did not impute missing data. Multiple records from same cohort were not included in same analysis.

Data Synthesis and Statistical Analysis

We directly extracted mean and SD for continuous variables or calculated them from relevant reported statistical results. For studies reporting medians and interquartile ranges (IQRs), we estimated means and SDs using established methods (12, 13).

We reported the AKI after ARDS incidence using Freeman-Tukey double-arcsine transformations (14). We presented results as risk ratio (RR) and 95% CIs for binary outcomes using pooled effect sizes. Because of the expected substantial clinical heterogeneity among the patient population, setting, study design, and kidney function and respiratory distress metrics across the studies, the effect size was calculated using the random effect model. Adjusted point estimates from each study were consolidated by the generic inverse variance approach of DerSimonian and Laird (15), which designated the weight of each study based on its variance. Quantitative heterogeneity was assessed by performing a formal homogeneity test and evaluating the proportion of total variability attributable to heterogeneity rather than sampling error (I^2). To explore sources of heterogeneity, we performed a subgroup analysis of early vs. late AKI development after ARDS. We reported the variability in studies in forest plots and incorporated them into the standard meta-analysis statistics. We used Review Manager 5.4 (Cochrane Library, Oxford, United Kingdom) and Open Meta software for analyses, with a significance threshold set at p value of less than 0.05.

Risk of Bias Assessment

Risk of bias was evaluated using Cochrane Collaboration risk assessment for nonrandomized studies, Risk of Bias in Observational Studies of Exposures tool (16) and a revised tool for randomized controlled trials, Risk of Bias 2 (17). Small-study effects and publication bias were assessed visually through funnel plots.

Grading the Quality of Evidence

We used the Grading of Recommendations Assessment, Development, and Evaluation (GRADE) (18) was used to assess the overall quality of evidence for each outcome.

RESULTS

Our search strategy initially retrieved 3646 potentially relevant articles for our study. Following the screening stage, we excluded 3559 articles, resulting in 87 articles that underwent full-text review. After the full-text review, an additional 70 articles were excluded, leaving us with 17 studies that met all the eligibility criteria and were included in the final analysis (**Supplemental Fig. S1**, http://links.lww.com/CCX/B307). The agreement between reviewers was substantial (κ = 0.79). Among those studies that described the demographic characteristics, 61% (14 studies) were male, and the average age of the participants was 54 years (95% CI, 49.7–59.4 yr; 15 studies). **Supplemental Table S1** (http://links.lww.com/CCX/B307) summarizes additional information on the countries where the studies were conducted, the definitions for AKI or ARDS, time to follow-up, ICU setting, leading causes of ARDS, and other study characteristics.

Incidence of AKI After ARDS

Of the 17 studies that examined the development of AKI after ARDS, 3287 patients with ARDS were found to develop AKI. This resulted in a pooled incidence rate of 39% (95% CI, 32–46%; 17 studies) (**Fig. 1**). It is important to note that significant heterogeneity was observed among the studies (I^2 = 98%; $p < 0.001$), with the incidence rate ranging from 19% to 68%. A sensitivity analysis was conducted to explore the potential sources of this heterogeneity, considering various factors such as the removal of studies with a high risk of bias due to missing data or potential underestimation of AKI due to unclear definitions of the AKI timing, studies with different definitions of AKI, the AKI timing, ICU setting, and age of the included patient. Despite these efforts, the heterogeneity remained high. This suggests that factors other than those considered in the sensitivity analysis may contribute to the observed variability among the studies.

To address the potential ambiguity arising from the simultaneous occurrence of AKI with ARDS, we conducted a separate analysis focusing on studies that reported the development of AKI at least 48 hours after the initial diagnosis of ARDS (19–23). By excluding cases of AKI within the initial 48 hours, we aimed to investigate AKI incidence beyond the immediate association with ARDS diagnosis. Our analysis revealed a pooled AKI incidence rate of 20% (95% CI, 18–21%; five studies) at least 48 hours after the diagnosis of ARDS. No statistically significant heterogeneity was observed among the included studies (I^2 = 19.5%; p = 0.32) (**Supplemental Fig. S2**, http://links.lww.com/CCX/B307).

ARDS As an Independent Risk Factor for the AKI Development

Only two studies, Darmon et al (1) and Han et al (24), conducted multivariable-adjusted analyses to evaluate whether ARDS is an independent predictor of AKI

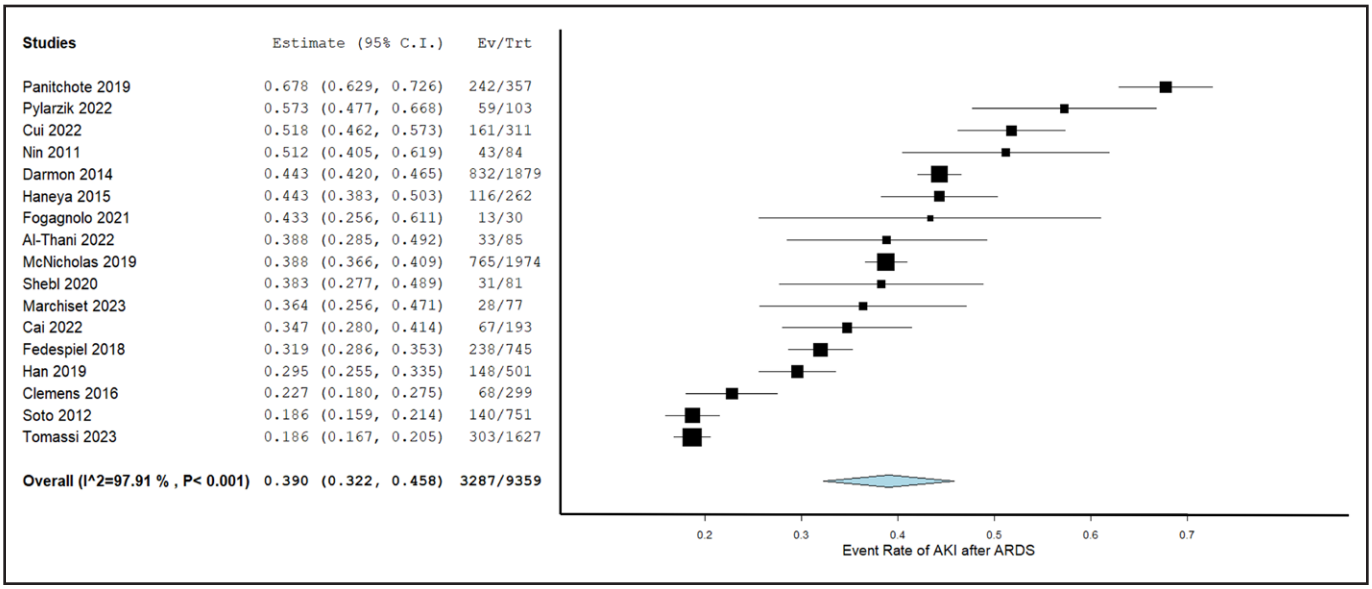

Figure 1. Incidence of acute respiratory distress syndrome (ARDS) patients who developed acute kidney injury (AKI) after diagnosis of ARDS. Ev, Event (number of patients developed AKI after ARDS); Trt, Treatment (Number of patients with ARDS)

development in critically ill patients. The pooled estimate from these two studies yielded an odds ratio (OR) of 2.16 (95% CI, 1.25–3.73; two studies) (**Fig. 2**).

Timing of AKI After ARDS

Five studies reported the timing of AKI development after ARDS (21, 25–28). The pooled estimate for the development of AKI after ARDS was 3.8 days (95% CI, 3.62–4.17; five studies) with no significant heterogeneity (I^2 = 26.6%; p = 0.24) (**Supplemental Fig. S3**, http://links.lww.com/CCX/B307). Three studies did not report the median or mean time of AKI development after ARDS but provided the number of patients who developed AKI at different time intervals. Soto et al (20) reported that out of 465 patients, 70% (n = 333) developed AKI on the day of ARDS (median, 0; IQR, 0–1), 20% (n = 95) developed AKI during the next 7 days after ARDS diagnosis, and the remaining 10% (n = 47) developed AKI after the first week. Nin et al (19) reported that out of 43 AKI patients, 65% (n = 28) developed AKI within 2 days, whereas 35% (n = 15) developed it after 2 days of ARDS diagnosis. Cui et al (23) indicated that out of 161 patients with AKI, 57% (n = 91) developed AKI within 48 hours of ARDS diagnosis, and 43% (n = 70) developed it after 48 hours.

Impact of AKI on Mortality in ARDS

A total of 12 studies reported the mortality of patients who developed AKI after ARDS compared with those who did not develop AKI (19, 21–23, 27–33). The follow-up time to mortality varied in different studies. While most studies reported hospital or 28- to 30-day mortality, Soto et al (20) followed patients for 60 days (Supplemental Table S1, http://links.lww.com/CCX/B307). The pooled estimate for the RR of mortality was 1.93 (95% CI, 1.71–2.18; 12 studies) (**Fig. 3**). However there was substantial heterogeneity (I^2 = 51%; p = 0.02), so we performed prespecified sensitivity analysis to exclude the studies done in different setting (Clemens et al [22] and Al-Thani et al [32] were done at burn ICU and trauma ICU, respectively). The sensitivity analysis showed a pooled RR of 1.84 (95% CI, 1.68–2.02; ten studies) with no significant heterogeneity (I^2 = 22%; p = 0.24) (**Supplemental Fig. S4**, http://links.lww.com/CCX/B307).

To determine if the development of AKI after ARDS is an independent risk factor for mortality, we separately analyzed studies that performed multivariable adjustment analysis (19, 20, 22, 23, 27, 30, 32). The pooled estimate of the OR was 3.69 (95% CI, 2.24–6.09; seven studies) (**Fig. 4**).

Association Between the Timing of AKI Development and Mortality of ARDS Patients

To assess the relationship between the timing of AKI development after ARDS and mortality, we compared mortality rates between early and late AKI. Only two studies (19, 23) provided the relevant data for this analysis. The results indicated that patients with late AKI had a higher mortality rate, with a pooled RR of 1.46 (95% CI, 1.19–1.9; two studies) (**Fig. 5**).

Comparison between AKI before vs. after ARDS showed that here was no difference in mortality between AKI before and AKI after ARDS with a pooled RR (1.32; 95% CI, 0.79–2.2; two studies) (21, 22) (**Supplemental Fig. S5**, http://links.lww.com/CCX/B307).

Risk of Bias and Quality of Evidence Assessment

Risk of bias was identified primarily in missing data and lack of confounder adjustment in observational

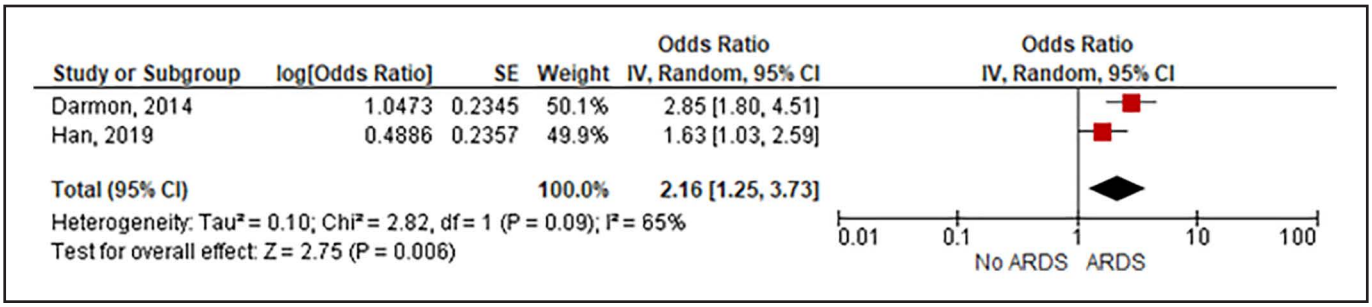

Figure 2. Acute respiratory distress syndrome (ARDS) as an independent risk of developing acute kidney injury. *df* = degrees of freedom.

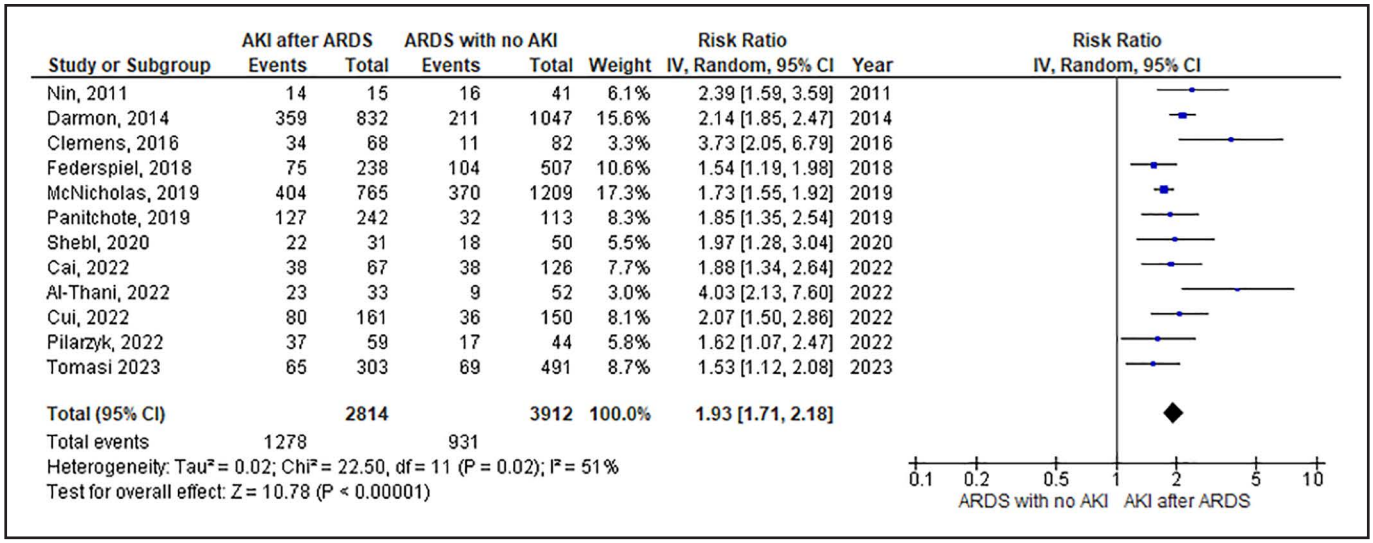

Figure 3. Mortality—acute kidney injury (AKI) after acute respiratory distress syndrome (ARDS) vs. no AKI and ARDS. *df* = degrees of freedom.

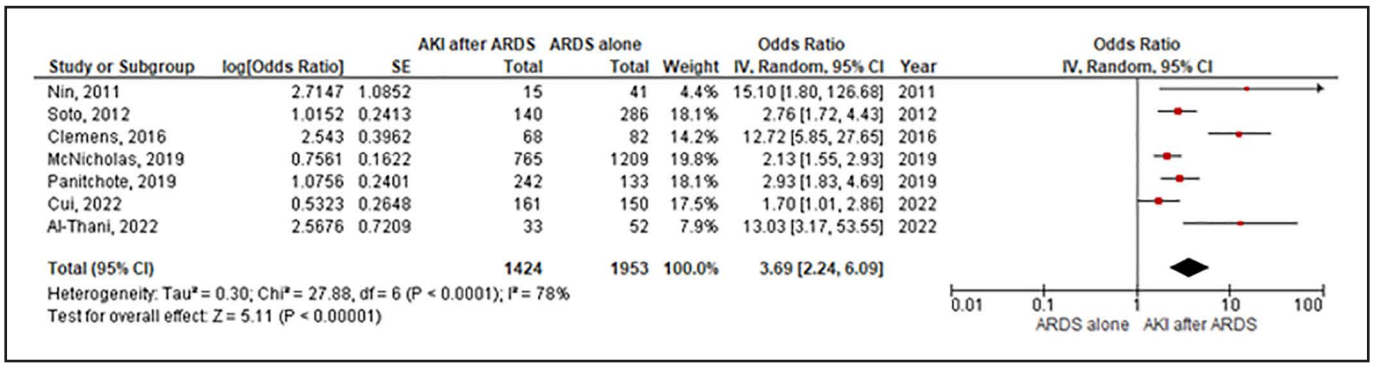

Figure 4. Acute kidney injury (AKI) as an independent risk factor for mortality in acute respiratory distress syndrome (ARDS) patient. *df* = degrees of freedom.

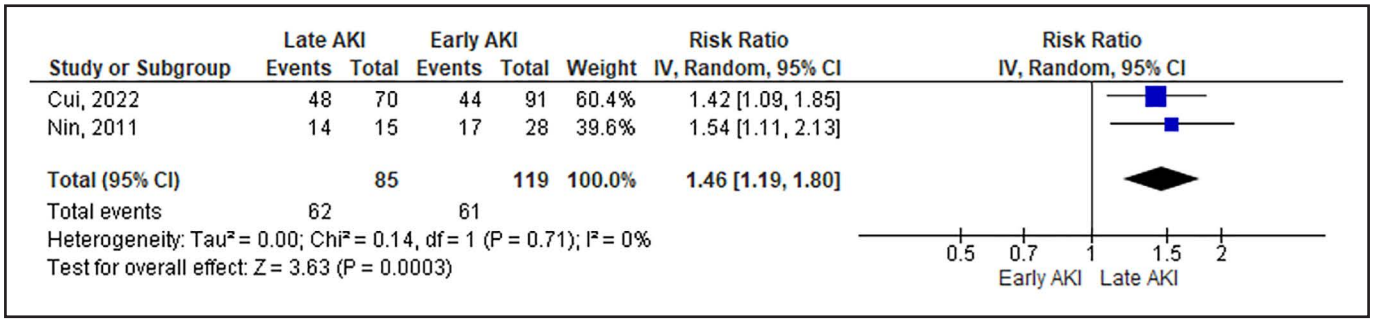

Figure 5. Mortality of patients who developed acute kidney injury (AKI) early vs. late AKI after diagnosis acute respiratory distress syndrome. *df* = degrees of freedom.

studies. No evidence of publication bias was found (**Supplemental Figs. S6** and **S7**, http://links.lww.com/CCX/B307). The quality of evidence, evaluated using the GRADE approach (18) (**Supplemental Table S2**, http://links.lww.com/CCX/B307), was generally low or very low. The incidence of AKI after ARDS had a very low quality of evidence due to the risk of bias in the included studies, inconsistency arising from high heterogeneity, and imprecision indicated by large CIs. However, the quality of evidence for the incidence of AKI at least 48 hours after ARDS was moderate. The timing of AKI development after ARDS had a very low certainty of evidence due to the serious risk of bias associated with study characteristics and the estimation

of mean and SD from the median and IQR. The certainty of evidence that AKI is an independent risk factor for increased mortality after ARDS was moderate. Regarding mortality based on the temporal relationship between AKI and ARDS (AKI prior vs. after ARDS) and mortality of early vs. late AKI, the quality of evidence remained low and very low, respectively.

DISCUSSION

Despite the existing evidence suggesting an interaction between AKI and ARDS, there is a lack of comprehensive studies that have evaluated their temporal relationship. Our systematic review and meta-analysis providing a comprehensive literature summary to date regarding the incidence of AKI after ARDS and its impact on clinical outcomes. We identified significant variability in the reported incidence of AKI following ARDS across the included studies. Our findings suggest that ARDS may independently contribute to the development of AKI. The development of AKI after ARDS was consistently associated with an increased risk of mortality, and AKI served as an independent risk factor for mortality in these patients.

Our study's insights into the relationship between ARDS and AKI echo historical findings from the 70s and 80s. Scholars like Baue et al (34) investigated "horror autotoxicus," a term denoting the progression of multiple organ failure, typically starting with lung failure and progressing to gut/liver and kidney failure. These early studies highlighted the systemic nature of organ response to severe injury or infection, leading to a deeper understanding of multiple organ failure. This historical context not only complements our findings but also underscores the ongoing evolution in comprehending complex organ interactions in critical conditions.

Debates have arisen over the temporal relationship between AKI and ARDS and their impacts on clinical outcomes. The key question revolves around whether ARDS is a true risk factor for subsequent AKI or if common underlying factors contribute to both conditions. A secondary analysis of the Large Observational Study to Understand the Global Impact of Severe Acute Respiratory Failure (LUNG-SAFE) study (30, 35, 36) suggests that ARDS may not substantially influence the subsequent development of AKI. Specifically, no significant associations were found between ventilatory variables, lung injury severity indices, and AKI incidence. Furthermore, risk factors for ARDS differ from those for AKI development. However, most patients in the secondary LUNG-SAFE study analysis experienced early-onset AKI, making it unclear if risk factors for later AKI in ARDS are similar (30, 35). Consequently, it remains unclear whether the risk factors for AKI development later in ARDS are similar. In a separate study, Cui et al (23) found that AKI occurring within 48 hours of ARDS diagnosis was linked to the disease severity and comorbidities, while late-onset AKI might be more associated with drug toxicity or other factors (23). While our analysis suggests that ARDS may independently contribute to AKI development, it is important to note that this conclusion is primarily based on findings from just two studies. Given the limited data, these insights should be considered as preliminary and interpreted with caution. The inclusion of these studies aims to highlight potential areas for further research rather than assert definitive conclusions.

Another challenge in determining the temporal relationship between AKI and ARDS is the diagnostic delay of approximately 24–48 hours when using serum creatinine levels to diagnose AKI (37). This delay makes it difficult to distinguish concomitant from subsequent AKI development after ARDS diagnosis. While the included studies reported AKI developed after ARDS, the timing of AKI development after ARDS was often not clearly described in most studies, making it uncertain whether AKI occurred concomitantly with the development of ARDS or subsequently. Two studies (19, 23), categorized AKI patients as early and late AKI, defined as AKI occurring within or after 48 hours of ARDS diagnosis. However, they did not provide information on patients developing AKI on the same day or within 24 hours of ARDS diagnosis, leading to ambiguity in distinguishing concomitant AKI occurrence. We separately analyzed studies reporting AKI development at least 48 hours after ARDS diagnosis, indicating an increased association of AKI development post-ARDS diagnosis.

Among the included studies, five reported a median time to AKI development following ARDS (1, 21, 25, 26, 38), averaging around 4 days. However, differences in AKI timing definitions in these studies make it challenging to interpret effect estimates with high confidence. Regarding mortality, patients developing AKI after ARDS had a significantly higher mortality rate

than those who did not. Pooled analysis of seven studies (19, 20, 22, 23, 27, 30, 32) conducting multivariable analysis showed that AKI development after ARDS could be an independent risk factor for mortality. Although some heterogeneity existed between studies, all consistently demonstrated AKI as an independent risk factor for significantly increased mortality among ARDS patients. Two studies (19, 23)compared mortality rates between early and late AKI development finding higher mortality in patients with late AKI development. However, the lack of multivariable analysis in included studies limited a robust assessment of late AKI as an independent risk factor for mortality.

The methodological strengths of our review enhance its validity and reliability. Comprehensive search strategies were conducted across multiple databases, guided by an experienced librarian's expertise. Including studies from different periods captured a comprehensive body of evidence for a more comprehensive understanding. A protocol for this review was developed and preregistered to ensure transparency and rigorous methodology. The study selection process involved two independent reviewers to minimize selection bias. Risk of bias in the included studies was assessed using the latest tools and guidelines, ensuring a robust evaluation of the available evidence.

Our study has several limitations. Significant heterogeneities existed, including study design, settings, definitions, timing adjudications, and patient populations. Some studies included patients from trauma or burn ICUs, introducing variability in the primary cause and ARDS severity. While most studies used standardized criteria for AKI and ARDS, others did not explicitly outline their definitions, potentially introducing inconsistencies in case classification. Variability in follow-up duration across studies may introduce biases and limit outcome comparability. In this systematic review, we encountered a notable variation in the definitions of early vs. late AKI development post-ARDS diagnosis across the included studies. However, we acknowledge the absence of a universally accepted standard in this regard, which may influence the interpretation of our results. This study, therefore, synthesizes data based on the heterogeneous definitions as provided by the individual studies. Thus, our findings regarding the timing of AKI development post-ARDS diagnosis should be interpreted with caution, given the variability in definitions across different studies. Limitation of our study is that we were not able to summarize the grade of AKI and rates of renal replacement therapy. The lack of uniform reporting in the primary studies limited our ability to provide a comprehensive analysis on this aspect. Another limitation of the study is that we included only articles that published in English. This was done to ensure the accuracy and consistency of data interpretation.

Our systematic review highlights a significant knowledge gap regarding the temporal relationship between AKI and ARDS. Available evidence is limited, with a scarcity of reliable data focusing on the temporal relationship in patients developing AKI after ARDS. This gap emphasizes the need for further studies to understand AKI incidence during different ARDS stages, identify specific risk factors, and evaluate preventive and management strategies. Investigating the temporal relationship between AKI and ARDS can contribute to recognizing and implementing measures to limit AKI development in this patient population.

CONCLUSIONS

Our systematic review and meta-analysis indicate that AKI frequently develops after ARDS and increases mortality risk. ARDS independently raises AKI risk and AKI post-ARDS is an independent risk factor for mortality. Despite these insights, our study also highlights a substantial knowledge gap in the existing literature regarding the temporal relationship between AKI and ARDS. The low to very low certainty of evidence in our analysis highlights the need for further research to enhance our understanding of this relationship and improve clinical management.

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Dr. Charkviani reviewed and appraised the literature, extracted the data, performed the statistical analyses, drafted and revised

the article, and approved the final version. Drs. Truong and Nikravangolsefid reviewed and appraised the literature, extracted the data, revised the article, and approved the final version. Drs. Reddy and Ninan revised the article and approved the final version. Mr. Prokop performed literature search, revised and approved the final version of article. Dr. Kashani supervised the work, revised the article, and approved the final version. Dr. Domecq Garces supervised the work, revised the article, and approved the final version. All authors have read and agreed to the published version of the article.

Dr. Kashani has reported receiving a grant from the National Institute of Diabetes and Digestive and Kidney Diseases (grant number R01DK131586); he has received consultancy fees and honoraria for delivering lectures from Baxter; and he serves on the advisory board of the Probe-Fluid Trial. The remaining authors have disclosed that they do not have any potential conflicts of interest.

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