Dicle Tıp Dergisi / Dicle Med J (2026) 53 (2) : 399-406

www.diclemedj.org

Original Article / Özgün Araştırma

Evaluation of the effect of acute kidney injury on weaning from mechanical ventilators in intensive care patients

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Received: 09.01.2026; Revised: 06.05.2026; Accepted: 07.05.2026

Abstract

Background: Acute kidney injury (AKI) affects 5–30% of intensive care unit (ICU) patients and is associated with increased mortality (40–90%), prolonged ICU stays, and higher healthcare costs. While the interplay between lung and kidney injury is increasingly recognized, the impact of AKI on weaning from mechanical ventilation (MV) remains underexplored. This study evaluates the effect of AKI on MV weaning outcomes in ICU patients.

Methods: A retrospective cohort study was conducted in an Internal Medicine ICU (2010–2015). Patients intubated for respiratory failure (≥48 hours) were included, excluding those with chronic kidney disease, chronic lung disease, or heart failure. AKI was defined according to the KDIGO criteria based on serum creatinine levels (an increase of ≥0.3 mg/dL within 48 hours or ≥1.5-fold within 7 days). Due to the retrospective nature of the study, urine output data were not reliably available for all patients and therefore were not included in the AKI definition. AKI was assessed based on data obtained during the intensive care course and was considered to have developed during ICU hospitalization. A total of 100 patients with AKI and 100 without AKI were compared in terms of demographic characteristics, laboratory parameters (creatinine, albumin, hemoglobin, pH, PaO_2, $PaCO_2$), SOFA and APACHE II scores, sepsis status, and weaning outcomes. Statistical analyses included Student's t-test, chi-square test, and multivariable logistic regression. The multivariable model included AKI status, age, APACHE II score, SOFA score, albumin level, and sepsis as covariates.

Results: Overall weaning success was 28% (56/200), with 13% in the AKI group and 43% in the non-AKI group ($p<0.001$) . Patients with AKI had significantly higher creatinine levels (2.99±1.3 vs. 0.78±0.22 mg/dL, $p<0.001$), APACHE II scores (28.2±5.4 vs. 22.3±4.6, $p<0.001$), and SOFA scores (10.1±3.1 vs. 7.7±2.7, $p<0.001$). In multivariable analysis, absence of AKI was independently associated with successful weaning (OR: 2.29, 95% CI: 1.03–5.13, p=0.043). In addition, lower APACHE II score was also independently associated with weaning success (p=0.030). Age showed borderline significance ($p=0.081$), whereas no independent association was found between weaning success and SOFA score, albumin level, or sepsis (all $p>0.05$).

Conclusion: AKI significantly impairs weaning from mechanical ventilation in ICU patients. Even after adjustment for disease severity, AKI remains an independent predictor of weaning failure. Early recognition of AKI may improve clinical management and facilitate successful weaning.

Keywords: Acute kidney injury, mechanical ventilation, weaning, respiratory failure, intensive care

DOI: 10.5798/dicletip.1964534

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Yoğun bakım hastalarında akut böbrek hasarının mekanik ventilatörden ayırma üzerine etkisinin değerlendirilmesi

Öz

Arka plan: Akut böbrek hasarı (AKI), yoğun bakım ünitesindeki (YBÜ) hastaların %5–30'unu etkiler ve artmış mortalite (%40–90), uzamış YBÜ yatışı ve daha yüksek sağlık maliyetleri ile ilişkilidir. Akciğer ve böbrek hasarı arasındaki karşılıklı etkileşim giderek daha fazla tanınmakla birlikte, AKI'nin mekanik ventilasyondan (MV) ayırma sürecine etkisi yeterince araştırılmamıştır. Bu çalışma, YBÜ hastalarında AKI'nin MV'den ayırma sonuçları üzerindeki etkisini değerlendirmektedir.

Yöntemler: Bu retrospektif kohort çalışma, Dahiliye Yoğun Bakım Ünitesi'nde (2010–2015) yürütüldü. Solunum yetmezliği nedeniyle entübe edilen ve en az 48 saat mekanik ventilasyon (MV) altında kalan ≥18 yaş hastalar dâhil edildi. Kronik böbrek hastalığı, kronik akciğer hastalığı veya kalp yetmezliği olan hastalar ile mekanik ventilasyon başladıktan sonra AKI gelişen hastalar çalışma dışı bırakıldı.

AKI, KDIGO kriterlerine göre serum kreatinin düzeylerine dayanarak tanımlandı (48 saat içinde ≥0.3 mg/dL artış veya 7 gün içinde ≥1.5 kat artış). Retrospektif veri yapısı nedeniyle idrar çıkışı verileri tüm hastalar için güvenilir şekilde mevcut olmadığından AKI tanımında kullanılmadı. AKI, yoğun bakım sürecinde elde edilen veriler temel alınarak değerlendirildi ve yoğun bakım yatışı sırasında gelişmiş kabul edildi.

AKI tanısı olan 100 hasta ile AKI olmayan 100 hasta karşılaştırıldı; demografik veriler (yaş, cinsiyet), laboratuvar parametreleri (kreatinin, albümin, hemoglobin, pH, PaO_2, $PaCO_2$), SOFA ve APACHE II skorları, sepsis durumu ve ventilatörden ayırma sonuçları analiz edildi.

İstatistiksel analizlerde Student t-testi, Ki-kare testi ve çok değişkenli lojistik regresyon kullanıldı. Çok değişkenli modelde AKI durumu, yaş, APACHE II skoru, SOFA skoru, albümin düzeyi ve sepsis değişkenleri yer aldı.

Bulgular: Genel MV'den ayırma başarısı %28'di (56/200). AKI grubunda başarı oranı %13 iken AKI olmayan grupta %43 idi (p<0.001). AKI hastalarında kreatinin (2.99±1.3 vs. 0.78±0.22 mg/dL, p<0.001), APACHE II (28.2±5.4 vs. 22.3±4.6, p<0.001) ve SOFA skorları (10.1±3.1 vs. 7.7±2.7, p<0.001) daha yüksekti.

Çok değişkenli lojistik regresyon analizinde, AKI olmaması ventilatörden ayırma başarısı ile bağımsız olarak ilişkili bulundu (OR: 2.29, %95 GA: 1.03–5.13, p=0.043). Ayrıca, daha düşük APACHE II skoru da ventilatörden ayırma başarısı ile bağımsız olarak ilişkiliydi (OR: 0.901, %95 GA: 0.820–0.990, p=0.030). Yaş sınırda anlamlılık gösterirken (p=0.081), SOFA skoru, albümin düzeyi ve sepsis ile ayırma başarısı arasında bağımsız bir ilişki saptanmadı (tüm p>0.05).

Sonuç: AKI, yoğun bakım hastalarında mekanik ventilasyondan ayırma sürecini anlamlı ölçüde olumsuz etkilemektedir. Hastalık şiddeti için düzeltme yapıldıktan sonra dahi, AKI ventilatörden ayırma başarısızlığının bağımsız bir belirleyicisi olarak kalmaktadır. AKI'nin erken tanınması, klinik yönetimi iyileştirebilir ve başarılı ventilatörden ayırmayı kolaylaştırabilir.

Anahtar kelimeler: Akut böbrek hasarı, mekanik ventilasyon, ayırma, solunum yetmezliği, yoğun bakım.

INTRODUCTION

Acute kidney injury (AKI) is a prevalent condition at intensive care units (ICUs), affecting 5–30% of patients and contributing to mortality rates of 40–90%[1]. AKI disrupts acid-base balance, fluid homeostasis, and drug metabolism, which can exacerbate respiratory dysfunction, reduce pulmonary compliance, and increase respiratory workload[2]. These effects may complicate weaning from mechanical ventilation (MV), a critical step in ICU recovery associated with reduced morbidity and costs[3].

Recent studies highlight a bidirectional relationship between lung and kidney injury, with AKI contributing to pulmonary edema, systemic inflammation, and impaired alveolar fluid clearance[4,5]. Experimental models demonstrate that AKI downregulates alveolar sodium and water channels, promoting extravascular lung water accumulation independent of hypervolemia[6]. Additionally, metabolic acidosis in AKI increases respiratory drive, potentially prolonging MV dependence[7].

Despite these insights, few clinical studies have explored AKI's direct impact on MV weaning. Existing research identifies heart failure, advanced age, acid-base disorders, and shock as predictors of weaning failure[8], but AKI's role remains unclear. This study aims to assess the effect of AKI on MV weaning in ICU patients with

respiratory failure, hypothesizing that AKI negatively influences weaning success.

METHODS

Study, Population and Design

This retrospective cohort study was conducted in the General Internal Medicine ICU between 2010 and 2015. Ethical approval was obtained from the local ethics committee (16.12.2016/33). Patients aged ≥18 years who were intubated due to respiratory failure and required mechanical ventilation (MV) for at least 48 hours were included. Patients with chronic kidney disease (CKD), chronic lung disease, heart failure, or those who developed AKI after the initiation of MV were excluded.

Medical records of 200 patients (100 with AKI and 100 without AKI) were retrospectively reviewed using the hospital information system. AKI was defined according to KDIGO criteria based on serum creatinine levels (increase in serum creatinine ≥0.3 mg/dL within 48 hours or ≥1.5 times baseline within 7 days). Urine output criteria were not included due to inconsistent availability of data in this retrospective cohort.

AKI was assessed based on laboratory data obtained during ICU stay and was considered to have developed during ICU hospitalization. Collected data included demographic characteristics (age, sex), laboratory parameters (creatinine, albumin, hemoglobin, pH, PaO_2, $PaCO_2$, SO_2), SOFA and APACHE II scores, sepsis status (defined according to Sepsis-3 criteria [10]), and weaning outcomes. Successful weaning was defined as sustained extubation without the need for reintubation for at least 48 hours.

Statistical Analysis

Data normality was assessed using Shapiro-Wilk and Kolmogorov-Smirnov tests. Normally distributed variables were expressed as mean±SD and compared using Student's t-test. Categorical variables analyzed with the Chi-square test. Univariate and multivariate logistic regression models evaluated factors associated with weaning success. A p-value <0.05 was considered significant and Analyses were performed using SPSS v26.0 (IBM Corp., Armonk, NY).

RESULTS

The study cohort comprised 200 patients (117 men, 83 women) with a mean age of 68.1±18.6 years, admitted to the General Internal Medicine Intensive Care Unit (ICU between 2010 and 2015. Acute kidney injury (AKI) was diagnosed in 100 patients (50%), while the remaining 100 served as non-AKI controls. Sepsis, defined per Sepsis-3 criteria, was present in 37.5% (75/200) of patients. Baseline clinical and laboratory parameters are detailed in Table 1. The mean creatinine level was 1.89±1.45 mg/dL, reflecting the mixed AKI and non-AKI population. Severity of illness was assessed using APACHE II (mean 25.3±5.8) and SOFA scores (mean 8.92±3.1), indicating a critically ill cohort. Other parameters, including albumin (2.3±0.55 g/dL), hemoglobin (9.95±2.55 g/dL), pH (7.39±0.09), $PaCO_2$ (37.9±12.4 mmHg), PaO_2 (123.5±58.2 mmHg), and SO_2 (97.1±2.6%), were consistent with ICU patients requiring mechanical ventilation (MV) for respiratory failure.

Table I: Baseline Clinical and Laboratory Characteristics

Parameter	Value (N=200)
Age (years)	68.1±18.6
Gender (M/F)	117/83
AKI (present/absent)	100/100
Sepsis (present/absent)	75/125
Creatinine (mg/dL)	1.89±1.45
Albumin (g/dL)	2.3±0.55
APACHE II	25.3±5.8
SOFA	8.92±3.1
Hemoglobin (g/dL)	9.95±2.55
pH	7.39±0.09
$PaCO_2$ (mmHg)	37.9±12.4
PaO_2 (mmHg)	123.5±58.2
SO_2 (%)	97.1±2.6

Overall, weaning from MV was successful in 28% (56/200) of patients. The AKI group had a significantly lower weaning success rate of 13% (13/100) compared to 43% (43/100) in the non-AKI group (p<0.001, Table 2). This stark difference underscores AKI's detrimental impact on weaning. Among the 144 patients who failed weaning, 87 (60.4%) were in the AKI group, highlighting a disproportionate burden of weaning failure in this subgroup.

Table II: Weaning Success Rates

Parameter	Number	Percentage
Weaning successful	56	28%
Weaning unsuccessful	144	72%

Comparison of AKI and Non-AKI Groups: Patients with AKI were significantly older (72.6±15.3 vs. 63.6±20.3 years, p<0.001) and exhibited higher disease severity, as evidenced by elevated APACHE II (28.2±5.4 vs. 22.3±4.6, p<0.001) and SOFA scores (10.1±3.1 vs. 7.7±2.7, p<0.001). Creatinine levels were markedly higher in the AKI group (2.99±1.3 vs. 0.78±0.22 mg/dL, p<0.001), consistent with KDIGO-defined AKI. Additionally, pH was lower in AKI patients (7.36±0.09 vs. 7.42±0.07, p<0.001), suggesting a trend toward metabolic acidosis, which may contribute to increased respiratory workload. No significant differences were observed in gender distribution (58M/42F vs. 59M/41F, p=0.88), sepsis prevalence (44% vs. 31%, p=0.58), albumin (2.24±0.51 vs. 2.34±0.58 g/dL, p=0.22), hemoglobin (10.2±2.4 vs. 10.9±2.6 g/dL, p=0.53), $PaCO_2$ (36.7±13.1 vs. 39.0±11.6 mmHg, p=0.192), or PaO_2 (121.6±44.6 vs. 125.4±69.4 mmHg, p=0.641; Table 3). These findings indicate that AKI's impact on weaning is not confounded by differences in gender, sepsis, or respiratory gas exchange parameters.

Table III: Comparison of AKI vs. Non-AKI Groups

Parameter	AKI (n=100)	Non-AKI (n=100)	p-value
Age (years)	72.6±15.3	63.6±20.3	**<0.001**
Creatinine (mg/dL)	2.99±1.3	0.78±0.22	**<0.001**
Gender (M/F)	58/42	59/41	0.88
Sepsis (present/absent)	44/56	31/69	0.58
Albumin (g/dL)	2.24±0.51	2.34±0.58	0.22
APACHE II	28.2±5.4	22.3±4.6	**<0.001**
SOFA	10.1±3.1	7.7±2.7	**<0.001**
Hemoglobin (g/dL)	10.2±2.4	10.9±2.6	0.53
pH	7.36±0.09	7.42±0.07	**<0.001**
$PaCO_2$ (mmHg)	36.7±13.1	39.0±11.6	0.192
PaO_2 (mmHg)	121.6±44.6	125.4±69.4	0.641

Patients who achieved successful weaning were younger (61.7±21.7 vs. 70.6±16.5 years, p=0.007), had lower creatinine levels (1.35±1.39 vs. 2.10±1.42 mg/dL, p=0.001), higher albumin (2.41±0.55 vs. 2.24±0.54 g/dL, p=0.047), lower APACHE II (21.7±4.6 vs. 26.6±5.7, p<0.001), and lower SOFA scores (7.45±2.3 vs. 9.5±3.2, p<0.001). Additionally, pH was higher in the weaned group (7.42±0.07 vs. 7.38±0.09, p=0.001), suggesting better acid-base balance. No significant differences were found in gender (37M/19F vs. 80M/64F, p=0.17), sepsis (20/36 vs. 55/89, p=0.745), hemoglobin (11.2±2.6 vs. 10.3±2.5 g/dL, p=0.32), $PaCO_2$ (38.4±11.9 vs. 37.6±12.6 mmHg, p=0.661), or PaO_2 (121.3±39.9 vs. 124.3±64.1 mmHg, p=0.747). Notably, AKI was significantly less prevalent in the weaned group (13/56 vs. 87/144, p<0.001), reinforcing its role as a key determinant of weaning failure.

Table IV: Comparison of Clinical and Laboratory Data Between Weaning Success and Failure Groups

Parameter	Weaning Success (n=56)	Weaning Failure (n=144)	p-value
Age (years)	61.7 ± 21.7	70.6 ± 16.5	**0.007**
Creatinine (mg/dL)	1.35 ± 1.39	2.10 ± 1.42	**0.001**
Gender (M/F)	37/19	80/64	0.17
AKI (present/absent)	13/43	87/57	**<0.001**
Albumin (g/dL)	2.41 ± 0.55	2.24 ± 0.54	**0.047**
APACHE II	21.7 ± 4.6	26.6 ± 5.7	**<0.001**
SOFA	7.45 ± 2.3	9.5 ± 3.2	**<0.001**
Hemoglobin (g/dL)	11.2 ± 2.6	10.3 ± 2.5	0.32
pH	7.42 ± 0.07	7.38 ± 0.09	**0.001**
$PaCO_2$ (mmHg)	38.4 ± 11.9	37.6 ± 12.6	0.661
PaO_2 (mmHg)	121.3 ± 39.9	124.3 ± 64.1	0.747
Sepsis (present/absent)	20/36	55/89	0.745

A multivariable logistic regression model was constructed to identify independent predictors of weaning success, including AKI status, age, APACHE II score, SOFA score, albumin levels, and sepsis.

In this model, absence of AKI was independently associated with a higher likelihood of successful weaning (odds ratio [OR]: 2.29, 95% confidence interval [CI]: 1.03–5.13, p=0.043). Lower APACHE II score was also independently associated with increased weaning success (OR: 0.901, 95% CI: 0.820–0.990, p=0.030).

Age showed a borderline association with weaning outcomes (OR: 0.984, 95% CI: 0.966–1.002, p=0.081), while SOFA score, albumin level, and sepsis were not significantly associated with weaning success (all p>0.05).

These findings indicate that AKI remains an independent determinant of weaning failure, even after adjustment for disease severity and other clinically relevant variables.

Table V: Multivariate Logistic Regression for Weaning Success

Variable	OR (Exp(B))	95% CI	p-value
AKI (Absent vs Present)	**2.29**	**1.03–5.13**	**0.043**
Age	0.984	0.966–1.002	0.081
APACHE II	**0.901**	**0.820–0.990**	**0.030**
SOFA	0.907	0.772–1.066	0.238
Albumin	1.537	0.267–8.846	0.630
Sepsis (Absent vs Present)	0.85	0.41–1.79	0.675

DISCUSSION

This study provides compelling evidence that acute kidney injury (AKI) significantly impairs weaning from mechanical ventilation (MV) in intensive care unit (ICU) patients with respiratory failure. The weaning success rate was markedly lower in patients with AKI (13% vs. 43%, p<0.001), and multivariate logistic regression confirmed that the absence of AKI was independently associated with a higher likelihood of successful weaning (OR: 2.29, 95% CI: 1.03–5.13, p=0.043)In addition, lower APACHE II score was also independently associated with increased weaning success (p=0.030).These findings underscore AKI as an independent risk factor for weaning failure, even after adjustment for disease severity,

adding to the growing recognition of kidney-lung crosstalk in critical illness[1,2].

The mechanisms underlying AKI's detrimental effect on MV weaning are multifaceted. AKI disrupts fluid homeostasis, often leading to oliguria and a positive fluid balance, which promotes pulmonary edema and reduces lung compliance[3]. Experimental studies have shown that AKI downregulates alveolar sodium and water channels (e.g., epithelial sodium channel, aquaporin-5), increasing extravascular lung water independent of hypervolemia[4]. This alveolar fluid accumulation impairs gas exchange and increases the work of breathing, complicating the weaning process[5]. Additionally, AKI-induced metabolic acidosis, as evidenced by lower pH in our AKI cohort (7.36±0.09 vs. 7.42±0.07, p<0.001), heightens respiratory drive to compensate for reduced serum bicarbonate levels[6]. This increased respiratory workload may exhaust respiratory muscles, prolonging MV dependence. Systemic inflammation, a hallmark of AKI, further exacerbates respiratory dysfunction by promoting cytokine-mediated muscle wasting and reducing diaphragmatic strength, as observed in chronic kidney disease and likely applicable to AKI[7,8]. Our study's finding of higher SOFA scores in AKI patients (10.1±3.1 vs. 7.7±2.7, p<0.001) supports the role of systemic inflammation, as SOFA scores reflect multi-organ dysfunction driven by inflammatory cascades[9].

Our results align with prior clinical studies linking AKI to adverse ICU outcomes. Vieira et al. reported a strong association between elevated creatinine levels and prolonged MV (p<0.001), with AKI patients exhibiting delayed weaning due to fluid overload and acid-base imbalances[10]. Similarly, Ostermann et al. found that AKI increased MV duration by 2–4 days in critically ill patients, attributing this to pulmonary-renal interactions[11]. Our study extends these findings by specifically quantifying AKI's impact on weaning success, a critical yet underexplored outcome. The higher APACHE II (28.2±5.4 vs. 22.3±4.6, p<0.001) and SOFA scores in our AKI group also corroborate studies identifying disease severity as a predictor of weaning failure[12,13]. Notably, APACHE II and SOFA scores, traditionally used for mortality risk stratification, emerged as robust markers of weaning potential in our cohort, suggesting their utility in guiding clinical decision-making beyond mortality prediction[14].

The association between older age and AKI in our study (72.6±15.3 vs. 63.6±20.3 years, p<0.001) is consistent with epidemiological data highlighting age-related renal vulnerability[15]. Elderly patients are more susceptible to AKI due to comorbidities (e.g., hypertension, diabetes), reduced renal reserve, and exposure to nephrotoxic agents during critical illness[16]. This age-AKI interaction may amplify weaning challenges, as older patients often have diminished respiratory muscle strength and lower physiological reserve[17].

The clinical implications of our findings are significant. Early recognition of AKI, potentially through routine monitoring of creatinine and urine output, could prompt interventions to optimize fluid balance (e.g., targeted diuresis, ultrafiltration) and correct acid-base disturbances, thereby facilitating weaning[18]. Moreover, our results highlight the need for tailored ventilator strategies in AKI patients, such as minimizing positive end-expiratory pressure (PEEP) to reduce intrathoracic pressure and renal venous congestion, which can exacerbate AKI[19]. The strong association between AKI and weaning failure also suggests that AKI severity (e.g., KDIGO stage) could be integrated into weaning readiness protocols, alongside traditional criteria like respiratory rate and oxygenation indices[20-22].

Prospective, multicenter studies are needed to validate our findings and explore AKI's mechanistic role in weaning failure using

advanced biomarkers (e.g., neutrophil gelatinase-associated lipocalin [NGAL], cystatin C) to detect AKI earlier[23]. Investigating AKI severity and its dose-response relationship with weaning outcomes could refine risk stratification. Randomized trials evaluating interventions like early RRT, fluid restriction, or anti-inflammatory therapies in AKI patients could provide actionable strategies to improve weaning success[24]. Additionally, integrating machine learning models to predict weaning outcomes based on AKI status, SOFA/APACHE II scores, and respiratory parameters could enhance personalized ICU care[25]. Finally, longitudinal studies tracking post-weaning recovery and functional outcomes in AKI survivors could elucidate the broader implications of our findings.

CONCLUSION

This study establishes AKI as a significant barrier to successful MV weaning in ICU patients, driven by fluid overload, acid-base imbalances, and systemic inflammation. Early AKI detection and targeted management may improve weaning outcomes, reduce MV duration, and alleviate ICU burden. These findings call for heightened awareness of kidney-lung interactions and the integration of AKI into weaning protocols to optimize critical care delivery.

Limitations

Several limitations of this study should be acknowledged. First, the retrospective design may introduce selection bias and limits the ability to establish causal relationships. Second, urine output data were not consistently available, and AKI was defined based solely on serum creatinine levels obtained at ICU admission. Third, AKI staging according to KDIGO classification was not evaluated, as the primary aim of the study was to assess the impact of AKI presence rather than severity; therefore, potential differences in weaning outcomes according to AKI stage could not be analyzed. Fourth, renal replacement therapy requirement was not specifically evaluated, as the study focused on comparing patients with and without AKI; however, RRT may reflect AKI severity and influence weaning outcomes. Fifth, although major chronic comorbid conditions such as chronic kidney disease, chronic lung disease, and heart failure were excluded, a standardized comorbidity index was not calculated, which may have limited adjustment for residual confounding. Finally, the study was conducted in a single center with a relatively limited sample size, which may limit the generalizability of the findings.

Ethics Committee Approval: Ethical approval was obtained from the local ethics committee (16.12.2016/33).

Conflict of Interest: The author(s) declare that there is no financial conflict of interest related to this article.

Financial Disclosure: The authors have stated that no financial support was provided for this study.

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