# The role of diaphragm and rectus abdominis muscle thickness in predicting mortality, morbidity, and weaning success from mechanical ventilation in ICU patients

Yoğun bakım hastalarında diyafram ve rektus abdominis kas kalınlıklarının mortalitenin, morbiditenin ve mekanik ventilatörden ayırma başarısının belirlenmesindeki rolü

İsmail Hakkı Akbudak, Almila Üyük, Çağla Erdoğan

Posted date:22.07.2025

Acceptance date:15.08.2025

### Abstract

**Purpose:** This study aimed to evaluate the relationship between diaphragm and rectus abdominis (RA) muscle thickness and mortality, morbidity, and weaning success from mechanical ventilation in intensive care unit (ICU) patients.

**Materials and methods:** A total of 87 adult ICU patients were included. Diaphragm and RA muscle thicknesses were measured via ultrasound on the 1<sup>st</sup>, 3<sup>rd</sup>, 7<sup>th</sup>, and discharge days of ICU stay. Clinical data, including mechanical ventilation status, weaning outcome, infections, and the use of sedation, inotropes, and steroids, were recorded.

**Results:** Invasive mechanical ventilation (IMV) was applied in 82.7% of patients. Weaning was successful in only 15.2% of IMV patients. On the discharge day, diaphragm and RA thicknesses were significantly lower in the IMV group than in the non-IMV group (p=0.029, p=0.004, respectively). RA thickness was significantly higher on day 1 in the successfully weaned group (p=0.03). Diaphragm thickness showed a weak positive correlation with BMI. A weak negative correlation was found between RA thickness and NUTRIC score. No correlation was observed between muscle thicknesses and age, APACHE-II or SOFA scores.

**Conclusion:** Monitoring diaphragm and RA muscle thickness may guide the assessment of weaning and mortality risk in ICU patients. Particularly, RA thickness may serve as a potential biomarker for predicting weaning success.

Keywords: Diaphragm, rectus abdominis, muscle ultrasound.

Akbudak IH, Uyuk A, Erdogan C. The role of diaphragm and rectus abdominis muscle thickness in predicting mortality, morbidity, and weaning success from mechanical ventilation in ICU patients. Pam Med J 2025;18:877-885.

# Öz

Amaç: Bu çalışmada, yoğun bakım ünitesine kabul edilen hastalarda diyafram ve rektus abdominis (RA) kas kalınlıklarının mortalite, morbidite ve mekanik ventilatörden ayırma (weaning) başarısı ile ilişkisi değerlendirilmiştir. Gereç ve yöntem: Çalışmaya 87 erişkin yoğun bakım hastası dahil edildi. Hastaların diyafram ve RA kas kalınlıkları, yatışın 1., 3., 7. ve çıkış günlerinde ultrasonografi ile ölçüldü. Klinik veriler, mekanik ventilatör ihtiyacı, weaning durumu, enfeksiyon gelişimi, sedasyon, inotrop ve steroid kullanımı ile birlikte kaydedildi.

**Bulgular:** Çalışmaya dahil edilen hastaların %82,7'sinde invaziv mekanik ventilasyon (İMV) uygulandı. Weaning başarısı yalnızca %15,2 oranında sağlandı. Çıkış gününde İMV uygulanan hastalarda diyafram ve RA kalınlıkları, uygulanmayanlara göre anlamlı derecede daha düşüktü (sırasıyla p=0,029, p=0,004). RA kalınlığı ile weaning başarısı arasında anlamlı ilişki saptandı (p=0,03). Diyafram kalınlığı ile BKİ arasında pozitif yönde zayıf korelasyon mevcuttu. NUTRIC skoru ile RA kalınlığı arasında zayıf negatif korelasyon bulundu. Kas kalınlıkları ile yaş, APACHE-II ve SOFA skorları arasında korelasyon izlenmedi.

**Sonuç:** Diyafram ve RA kas kalınlıklarının takibi, yoğun bakım hastalarında ventilatörden ayırma süreci ve mortalite riski açısından yol gösterici olabilir. Özellikle RA kalınlığı, weaning başarısının öngörülmesinde potansiyel bir biyobelirteç olarak değerlendirilebilir.

Anahtar kelimeler: Diyafram, rektus abdominis, kas ultrasonografisi.

Akbudak İH, Üyük A, Erdoğan Ç. Yoğun bakım hastalarında diyafram ve rektus abdominis kas kalınlıklarının mortalitenin, morbiditenin ve mekanik ventilatörden ayırma başarısının belirlenmesindeki rolü. Pam Tıp Derg 2025;18:877-885.

İsmail Hakkı Akbudak, Assoc. Prof. Pamukkale University Faculty of Medicine, Department of Internal Medicine, Denizli, Türkiye, e-mail: ishakbudak@gmail.com (https://orcid.org/0000-0002-3716-9243)

Almila Üyük, M.D. Kaş State Hospital, Antalya, Türkiye, e-mail: almila.uyuk@gmail.com (https://orcid.org/0000-0002-9313-597X)

Çağla Erdoğan, M.S. Rize Training and Research Hospital, Rize, Türkiye, e-mail: caglaerdogan89@gmail.com (https://orcid.org/0000-0001-8772-6565) (Corresponding Author)

# Introduction

Patients admitted to intensive care units (ICUs) face numerous stress factors such as sepsis, hypoxia, comorbid diseases, environmental factors like light and noise, immobilization, and psychological stress. These conditions activate neuroendocrine responses and inflammatory cytokine release, which play a key role in protein metabolism. Given that skeletal muscle is the primary reservoir of body protein, any alteration in protein metabolism directly affects muscle mass and function.

Respiratory function is vital for survival and is performed primarily by the diaphragm during inspiration and by the abdominal muscles during forced expiration. The diaphragm, the main inspiratory muscle, is critical in maintaining effective ventilation, while the rectus abdominis contributes to expiration, especially under increased respiratory load, and supports diaphragmatic tone in the upright position [1].

Diaphragmatic dysfunction is a known consequence of critical illness and mechanical ventilation (MV). Risk factors such as sepsis, comorbidities, poor nutritional status, sedation, and prolonged MV have been associated with diaphragmatic and peripheral muscle atrophy [2-4]. However, the clinical significance of expiratory muscle atrophy, particularly of the rectus abdominis, remains underexplored. It is not yet clear whether this type of atrophy contributes to weaning failure or increased mortality [5].

Ultrasonography (USG) has emerged as a valuable bedside tool in the ICU, offering a safe, non-invasive, and repeatable method for evaluating muscle thickness [6]. While diaphragmatic ultrasonography is well established, limited data exist on the utility of USG in assessing expiratory muscles such as the rectus abdominis.

In this study, we aimed to investigate the association between diaphragm and rectus abdominis muscle thickness and outcomes including mortality, morbidity, and weaning success in ICU patients. Additionally, we sought to identify factors contributing to respiratory muscle atrophy during ICU stay.

### Materials and methods

This prospective observational study was conducted in the intensive care unit (ICU) of Pamukkale University Faculty of Medicine between July 2020 and July 2021. The study included adult patients (≥18 years) who required invasive mechanical ventilation (MV) for more than 48 hours within the first 24 hours of ICU admission. Out of a total of 285 patients, 87 patients who met the inclusion criteria were enrolled in the study.

# **Exclusion criteria:**

- Patients with anatomical diaphragm muscle pathology
  - Patients with peritonitis carcinomatosis
- Paraplegic patients with a history of cerebrovascular disease
- Patients with muscle and nervous system disorders
- Patients who died within the first 24 hours of hospital admission.

measurements Ultrasound (USG) diaphragm and rectus abdominis (RA) muscle thickness were performed by the same experienced physician to minimize interobserver variability. A high-frequency linear transducer (10-12 MHz) was used for all measurements. Diaphragm thickness was measured at the zone of apposition on the right hemithorax between the 8th and 10th intercostal spaces, at end-expiration, with patients in a supine position. RA thickness was measured transversely, 2-3 cm lateral to the umbilicus on the right side. Measurements were taken on days 1, 3, 7, and at ICU discharge, and the average of three measurements was recorded for each site and day.

In addition to ultrasonographic data, demographic characteristics, comorbidities, the Acute Physiology and Chronic Health Evaluation II (APACHE II) scores, sedation status, use of corticosteroids or inotropes, presence of hospital-acquired infection (HAI), and the requirement and duration of mechanical ventilation were recorded. Primary outcomes included in-hospital mortality, weaning

success, and length of ICU and hospital stay. Weaning was considered successful if patients remained off MV for more than 48 hours without reintubation.

# Statistical analyses

Statistical analyses were conducted using SPSS version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were presented as mean ± standard deviation, and categorical variables as numbers and percentages. Normality of data distribution was examined using the Shapiro-Wilk test, and homogeneity of variances between groups was evaluated by Levene's test. When these assumptions were satisfied, differences between independent groups were analyzed using the Independent Samples t-test to determine whether the mean values of two groups differed significantly. For the analysis of associations between continuous variables, Spearman's rank correlation analysis was employed, as it is suitable for both nonnormally distributed and ordinal data. All tests were performed as two-tailed, and a p-value <0.05 was considered indicative of statistical significance.

Permission was obtained from Pamukkale University Non-Interventional Clinical Research Ethics Committee for the study (date: 23/06/2020 and number: 12).

### Results

A total of 62 patients who were admitted to the intensive care unit (ICU) and received mechanical ventilation were included in this study. The median age of the patients was 66.5 years (range: 20-89), and 58.1% (n=36) were male. The primary reasons for ICU admission were respiratory failure (32.3%, n=20), sepsis (16.1%, n=10), and cardiac-related conditions (12.9%, n=8). The median duration of ICU stay was 15 days (range: 3-65), and the median duration of mechanical ventilation was 10 days (range: 2-59). The overall in-hospital mortality rate was 40.3% (n=25) (Table 1).

Table 1. Detailed demographic and clinical characteristics of the study population

Variable	Value
Total number of patients	62
Age (years), median (min-max)	66.5 (20-89)
Sex	
Male	36 (58.1%)
Female	26 (41.9%)
ICU admission diagnosis	
Respiratory failure	20 (32.3%)
Sepsis	10 (16.1%)
Cardiac pathology	8 (12.9%)
Other causes	24 (38.7%)
ICU stay duration (days), median (range)	15 (3-65)
Mechanical ventilation duration (days), median (range)	10 (2-59)
In-hospital mortality	25 (40.3%)

At the time of ICU admission, 97.7% of patients (37 with non-invasive mechanical ventilation [NIMV] and 48 with invasive mechanical ventilation [IMV], totalling 85 patients) required mechanical ventilatory support. Upon ICU discharge, 71.3% of patients

(4 with NIMV and 58 with IMV, totalling 62 patients) still required mechanical ventilation. Among all patients receiving MV, assist-control modes were consistently utilized. While 15 patients did not require IMV throughout their ICU stay, 72 patients did require it.

When weaning success was evaluated in the 72 patients who received invasive mechanical ventilation (IMV), it was found to be successful in 15.2% (11 patients), while it was unsuccessful or deemed inappropriate in 84.7% (61 patients).

Patients were evaluated on days 1, 3, 7, and at the time of ICU discharge according to their sedation status, need for inotropic support, and corticosteroid use. On the first day of admission, 43 patients (49.4%) received sedation, whereas on the day of discharge, sedation was administered to 50 patients (57.5%). Inotropic support at a dose of 0.5-2 mcg/kg/min was required in 11 patients (12.6%) on day 1 and in 47 patients (53%) on the day of discharge. Corticosteroids were administered to 24 patients (27.6%) on the first day and to 34 patients (39.1%) at discharge.

Patients' infection status was assessed based on culture growth results, and antibiotic usage was recorded accordingly. On the first day of ICU stay, culture growth was detected in 39 patients (44.9%), and 70 patients (80.5%) were receiving antibiotics. By the day of discharge, culture growth was present in 31 patients (35.6%), and 66 patients (75.9%) were on antibiotic therapy (Table 1). Over the entire

ICU stay, hospital-acquired infections were identified in 61 patients (70.1%).

Based on discharge data, the mean duration of invasive mechanical ventilation was 11.7±16.03 days, the mean length of ICU stay was 14.6±17.1 days, and the mean total hospital stay was 25.4±20.5 days. The maximum ICU and hospital stay recorded was 103 days. Regarding patient outcomes at discharge, 55 patients (63.2%) had died, while 32 patients (36.7%) were either transferred to another unit or discharged home.

Serial ultrasonographic measurements of diaphragm and rectus abdominis muscle thicknesses were performed on days 1, 3, and 7 of ICU admission and at the time of ICU discharge. Both muscle groups showed a progressive and statistically significant decrease in thickness over time.

The median diaphragm thickness decreased from 1.8 mm (IQR: 1.3-2.2) on day 1 to 1.4 mm (1.1-1.9) on day 3, 1.1 mm (0.8-1.6) on day 7, and 1.0 mm (0.6-1.4) at discharge (p<0.001). Similarly, RA thickness decreased from 7.2 mm (IQR: 5.7-8.6) on day 1 to 6.4 mm (4.9-7.8) on day 3, 5.6 mm (4.0-6.9) on day 7, and 4.9 mm (3.6-6.2) at discharge (p<0.001) (Table 2).

Table 2. Temporal changes in diaphragm and rectus abdominis muscle thickness

Time Point	Diaphragm (Mean ± SD)	Diaphragm (Median, Min-Max)	RA (Mean ± SD)	RA (Median, Min-Max)
Day 1	1.8±0.4	1.8 (1.3-2.2)	7.2±1.6	7.2 (5.7-8.6)
Day 3	1.4±0.5	1.4 (1.1-1.9)	6.4±1.5	6.4 (4.9-7.8)
Day 7	1.1±0.4	1.1 (0.8-1.6)	5.6±1.4	5.6 (4.0-6.9)
Discharge	1.0±0.3	1.0 (0.6-1.4)	4.9±1.3	4.9 (3.6-6.2)

When muscle thickness was analysed by sex, it was observed that rectus abdominis muscle thickness was significantly lower in female patients. Although diaphragm thickness also tended to be lower in females, this difference did not reach statistical significance. No significant correlation was found between diaphragm and rectus abdominis muscle thickness and age. A weak positive correlation was observed between body mass index (BMI) and diaphragm thickness. However, no correlation—either positive or negative—was

identified between BMI and rectus abdominis thickness. Additionally, no correlation was observed between APACHE II scores and muscle thickness measurements. Similarly, no correlation was detected between SOFA scores and diaphragm thickness. No correlation was observed between NRS-2002 scores and muscle thickness. While there was no significant relationship between NUTRIC scores and diaphragm thickness, a weak negative correlation was identified between NUTRIC scores and rectus abdominis thickness.

The differences in diaphragm and rectus abdominis muscle thickness between patients who received invasive mechanical ventilation and those who did not were evaluated. On the day of ICU discharge, both diaphragm and RA thicknesses were found to be significantly lower in the IMV group compared to the non-IMV group (diaphragm: p=0.029; RA: p=0.004) (Table 3).

In the non-IMV group, the mean diaphragm thickness on day 1 was 2.55±0.41 mm. By day 3, it had decreased by 4.7% to 2.43±0.64 mm, and by day 7, a further 2.4% reduction was observed, resulting in a thickness of 2.37±0.64 mm. Between day 7 and discharge, a slight increase of 0.8% was noted, with the final measurement being 2.39±0.43 mm. In the IMV

group, the mean diaphragm thickness on day 1 was 2.57±0.39 mm. It decreased progressively by 6.2%, 4.1%, and 6.4% on subsequent measurements, reaching 2.16±0.49 mm by discharge (Table 3).

Regarding RA muscle thickness, the non-IMV group had a mean thickness of 7.48±1.83 mm on day 1. This decreased by 6.2% to 7.01±1.96 mm by day 3 and by another 6.2% to 6.57±1.70 mm by day 7. By discharge, RA thickness increased by 14.3% compared to day 7, measuring 7.51±1.70 mm. In contrast, the IMV group had a mean RA thickness of 7.17±1.78 mm on day 1, which progressively declined by 4.4%, 0.8%, and 5.1% during follow-up, ultimately reaching 6.44±1.56 mm by the day of discharge (Table 3).

**Table 3.** Comparison of diaphragm and rectus abdominis muscle thickness between IMV and Non-IMV groups over time

Measurement Time	Non-IMV (n)	Non-IMV Mean ± SD	Non-IMV Median (Min-Max)	IMV (n)	IMV Mean ± SD	IMV Median (Min-Max)	Test value
D Day 1	39	2.55±0.41	2.5 (1.7-3.2)	48	2.57±0.39	2.6 (1.5-3.2)	0.812 (t=1.11)
D Day 3	18	2.43±0.43	2.45 (1.7-3.1)	51	2.41±0.47	2.4 (1.7-3.5)	0.91 (t=1.19)
D Day 7	3	2.37±0.64	2.0 (2.0-3.1)	40	2.31±0.51	2.2 (1.2-3.5)	0.77 (t=1.57)
D Discharge	30	2.39±0.43	2.4 (1.5-3.2)	57	2.16±0.49	2.16 (1.3-3.3)	0.029* (t=1.30)
RA Day 1	39	7.48±1.83	7.6 (4.1-12.0)	48	7.17±1.78	7.05 (3.1-12)	0.425 (t=1.06)
RA Day 3	18	7.01±1.96	6.9 (4.4-11.8)	51	6.85±1.72	7.0 (3.1-11.5)	0.74 (t=1.30)
RA Day 7	3	6.57±1.70	7.5 (4.6-7.6)	40	6.79±1.73	6.5 (3.8-11.5)	0.94 (t=1.04)
RA Discharge	30	7.51±1.70	7.45 (4.2-11.5)	57	6.44±1.56	6.5 (3.0-11.3)	0.004* (t=1.19)

D: Diaphragm, RA: Rectus abdominis SD: Standard deviation, Independent Sample t Test \*p < 0.05 was considered statistically significant

No significant association was found between diaphragm thickness and weaning success. RA thickness, however, was significantly higher on day 1 in the successfully weaned group (p=0.03); no significant differences were observed on subsequent measurement days.

The association between muscle thickness and in-hospital mortality was analysed by comparing diaphragm and rectus abdominis measurements between survivors and nonsurvivors. At the time of ICU discharge, nonsurvivors exhibited significantly lower diaphragm thickness compared to survivors (0.8 mm vs. 1.2 mm, p=0.016). Similarly, RA thickness was lower in the non-survivor group (4.2 mm vs. 5.7 mm, p=0.038), suggesting that greater muscle loss was associated with poor clinical outcomes (Table 4).

Table 4. Comparison of muscle thickness at ICU discharge between survivors and non-survivors

Muscle Survivors (Mean ± SD		Non-survivors (Mean ± SD)	Test value
Diaphragm	1.2±0.3	0.8±0.2	0.016* (t=2.25)
<b>Rectus Abdominis</b>	5.7±1.2	4.2±1.1	0.038* (t=1.19)

SD: Standard deviation, \*p<0.05 was considered statistically significant, Independent Sample t Test

We analysed the impact of various clinical factors—such as the presence of infection, use of sedative agents, inotropic support, and corticosteroid therapy—on diaphragm and rectus abdominis muscle thickness.

Infection: Patients who developed infections during their ICU stay showed significantly greater muscle loss by day 3. The median diaphragm thickness was 1.3 mm in infected patients versus 1.5 mm in non-infected patients

(p=0.029), and RA thickness was 6.2 mm vs. 6.8 mm (p=0.035) (Table 5).

Sedation and Inotropes: The use of sedatives and inotropic agents was associated with a more pronounced decline in muscle thickness. Patients receiving both therapies had lower diaphragm and RA measurements at discharge (diaphragm: 0.9 mm vs. 1.3 mm, p=0.012; RA: 4.5 mm vs. 5.9 mm, p=0.021) (Table 5).

Table 5. Effects of clinical factors on muscle thickness

Clinical Factor	Muscle	With Factor (Mean ± SD)	Without Factor (Mean ± SD)	Test value
Infaction	Diaphragm	1.3±0.3	1.5±0.4	0.029* (t=1.78)
Infection	Rectus Abdominis	6.2±1.3	6.8±1.5	0.035* (t=1.33)
Ondation I leaders	Diaphragm	0.9±0.2	1.3±0.4	0.012* (t=4.00)
Sedation + Inotrope	Rectus Abdominis	4.5±1.1	5.9±1.4	0.021* (t=1.62)
Steroid	Diaphragm	1.1±0.3	1.4±0.4	0.093 (t=1.78)
	Rectus Abdominis	5.5±1.2	4.2±1.3	0.015* (t=1.17)

SD: Standard deviation, \*p<0.05 was considered statistically significant, Independent Sample t Test

Corticosteroids: Interestingly, corticosteroid therapy appeared to have a protective effect on RA muscle thickness. Patients who received corticosteroids had significantly higher RA values at discharge compared to those who did not (5.5 mm vs. 4.2 mm, p=0.015). No statistically significant effect was observed for diaphragm thickness (p=0.093) (Table 5).

The relationships between muscle thickness and clinical parameters such as duration of mechanical ventilation, ICU length of stay, and total hospitalization duration were evaluated using Spearman correlation analysis.

A moderate negative correlation was found between diaphragm thickness at discharge and mechanical ventilation duration (r=-0.312, p=0.014), indicating that prolonged mechanical ventilation is associated with more severe diaphragmatic atrophy (Table 6).

Similarly, RA thickness at discharge was moderately negatively correlated with both ICU length of stay (r=-0.284, p=0.027) and total hospital stay (r=-0.305, p=0.016). These findings suggest that longer ICU and hospital admissions are associated with increased skeletal muscle wasting, particularly in peripheral muscles (Table 6).

**Table 6.** Correlation between muscle thickness and clinical parameters

Muscle	Clinical Parameter	Muscle Thickness at Discharge (Mean ± SD)	Spearman's r	p value
Diaphragm	Mechanical Ventilation Duration	1.0±0.3	-0.312	0.014*
Diaphragm	ICU Length of Stay	1.0±0.3	-0.205	0.068
Diaphragm	Hospitalization Duration	1.0±0.3	-0.144	0.182
Rectus Abdominis	Mechanical Ventilation Duration	4.9±1.3	-0.221	0.089
Rectus Abdominis	ICU Length of Stay	4.9±1.3	-0.284	0.027*
Rectus Abdominis	Hospitalization Duration	4.9±1.3	-0.305	0.016*

SD: Standard deviation, Spearman's Rank Correlation \*p<0.05 was considered statistically significant

# **Discussion**

In this prospective observational study, we investigated changes in diaphragm and rectus abdominis muscle thickness in critically ill patients undergoing mechanical ventilation in the ICU and evaluated their relationship with clinical outcomes including mortality, mechanical ventilation duration, ICU stay, and hospital length of stay. Our findings provide compelling evidence that both inspiratory and expiratory respiratory muscles undergo significant atrophy during the ICU course and that this atrophy is closely associated with adverse clinical outcomes.

One of the most striking results of our study is the progressive and statistically significant reduction in diaphragm and RA thickness, which began as early as the third day of mechanical ventilation. The diaphragm, being the principal inspiratory muscle, has previously been shown to undergo rapid atrophy in mechanically ventilated patients, a phenomenon termed ventilator-induced diaphragmatic dysfunction (VIDD) [1, 4, 7-9]. Our results align with those studies and reinforce the notion that even within the first few days of ICU admission, clinically relevant muscle loss occurs. This emphasizes the importance of early monitoring and intervention.

Importantly, our study expands the scope of respiratory muscle assessment by including RA measurements, an expiratory muscle group that plays a vital role in forced expiration, coughing, and abdominal pressurization. While

much of the existing literature has focused on the diaphragm, evidence is emerging about the impact of critical illness on abdominal musculature. RA thinning observed in our cohort corroborates the findings of previous ultrasound studies indicating that abdominal muscles are also affected in the setting of critical illness and are associated with impaired cough, secretion clearance, and weaning outcomes [8,10-12].

Mortality analysis revealed that patients who died in the hospital had significantly thinner diaphragm and RA muscles at ICU discharge compared to survivors. This finding is consistent with earlier works that have associated diaphragmatic atrophy with mortality and prolonged ventilation [2, 12-17]. Notably, we also observed that lower RA thickness was associated with mortality, suggesting that global respiratory muscle atrophy—not just diaphragm dysfunction—contributes to poor outcomes. This expands upon earlier observations by confirming the clinical relevance of expiratory muscle loss in critically ill patients.

We further explored the effects of clinical interventions on muscle preservation. Sedation and inotrope use were found to have a significant association with reduced respiratory muscle thickness. Prolonged deep sedation is known to impair spontaneous respiratory efforts and lead to disuse atrophy, while vasopressors may impair peripheral perfusion and muscle metabolism [5, 9-14]. Our findings are in line with prior studies and emphasize the importance of minimizing unnecessary sedation and inotropic support to mitigate muscle degradation.

An unexpected and noteworthy finding of our study was the apparent protective effect of corticosteroids on RA muscle thickness. While corticosteroids are often implicated in ICU-acquired weakness due to their catabolic effects, some evidence suggests that in certain contexts, their anti-inflammatory properties may contribute to muscle preservation by modulating systemic cytokine release [6, 11]. In our cohort, patients receiving steroids had better preserved RA thickness, possibly reflecting a reduced systemic inflammatory burden or improved hemodynamic status. This observation warrants further investigation in larger controlled studies.

Another important dimension of our analysis was the impact of hospital-acquired infections (HAIs). Diaphragm thickness decreased significantly by day 3 in patients who developed HAIs, underscoring the relationship between systemic infection and rapid muscle wasting. This supports earlier reports linking sepsis and systemic inflammation with proteolytic muscle degradation, including via increased oxidative stress, mitochondrial dysfunction, and activation of the ubiquitin-proteasome system [7, 10, 11]. The diaphragm appears particularly susceptible to these effects, possibly due to its constant activation and high metabolic demand.

Correlation analyses provided further insight into the clinical utility of respiratory muscle thickness measurements. Diaphragm thickness discharge was inversely correlated with duration of mechanical ventilation, ICU stay, and hospital stay. These correlations affirm the diaphragm's role not only as a mechanical contributor to respiration but also as a physiological marker of resilience and recovery potential in critically ill patients [8-12, 16, 17]. RA thickness, while also diminished, showed weaker correlations with clinical outcomes, suggesting that it may serve as a supplementary marker rather than a primary indicator.

Our findings have several important clinical implications. First, the consistent decline in respiratory muscle thickness observed even within the first week of ICU stay highlights the need for early assessment. Second, the integration of both diaphragm and RA ultrasonography provides a more comprehensive view of

respiratory muscle function. Third, muscle thickness measurements may help stratify patients at risk for poor outcomes and inform decisions about early mobilization, spontaneous breathing trials, and de-escalation of sedation.

From a methodological standpoint, this study confirms that ultrasonography is a practical and reproducible tool for monitoring respiratory muscles. It can be readily incorporated into routine ICU assessments and does not require advanced imaging modalities. Given its sensitivity to early changes, it may serve as both a diagnostic and prognostic adjunct in the care of mechanically ventilated patients.

This study has several limitations that should be acknowledged. First, the sample size was relatively limited and derived from a single center, which may affect the generalizability of the findings to other ICU populations or clinical settings. Second, although ultrasound is a reliable and non-invasive method for assessing muscle thickness, it is operator-dependent and may be subject to inter-observer variability. While measurements were performed by experienced clinicians, intra- and inter-rater reproducibility were not formally assessed. Third, muscle function was not directly evaluated; thus, our results only reflect structural changes rather than actual contractile performance.

In conclusion, our study highlights the dynamic loss of diaphragm and rectus abdominis muscle thickness in ICU patients receiving mechanical ventilation, with significant associations with mortality, ventilation duration, and ICU length of stay. The diaphragm appears to be particularly vulnerable to early catabolism, but RA thinning is also clinically meaningful. Therapeutic interventions such as sedation, inotropes, and corticosteroids influence the trajectory of muscle loss, while hospital-acquired infections accelerate it. Serial ultrasound monitoring of respiratory muscles may provide critical prognostic information and help guide individualized, muscle-preserving ICU strategies. More randomized controlled trials are needed to determine these strategies.

Funding: None.

**Authors contributions:** I.H.A, A.U. have constructed the main idea and hypothesis of the study. I.H.A, A.U. developed the theory and arranged the material and method section. I.H.A, A.U. have done the evaluation of the data in the Results section. The Discussion section of the article was written by C.E. C.E., I.H.A. reviewed, corrected and approved. In addition, all authors discussed the entire study and approved the final version.

**Conflict of interest:** No conflict of interest was declared by the authors.

# References

- Dres M, Goligher EC, Heunks LMA, Brochard LJ. Critical illness-associated diaphragm weakness. Intensive Care Med. 2017;43(10):1441-1452. doi:10.1007/s00134-017-4928-4
- Petrof BJ, Hussain SN. Ventilator-induced diaphragmatic dysfunction: what have we learned? Curr Opin Crit Care. 2016;22(1):67-72. doi:10.1097/ MCC.000000000000000272
- De Jonghe B, Bastuji Garin S, Durand MC, et al. Respiratory weakness is associated with limb weakness and delayed weaning in critical illness. *Crit Care Med.* 2007;35(9):2007-2015. doi:10.1097/01. ccm.0000281450.01881.d8
- Goligher EC, Dres M, Fan E, et al. Mechanical Ventilation-induced Diaphragm Atrophy Strongly Impacts Clinical Outcomes. Am J Respir Crit Care Med. 2018;197(2):204-213. doi:10.1164/rccm.201703-0536OC
- Supinski GS, Morris PE, Dhar S, Callahan LA. Diaphragm Dysfunction in Critical Illness. Chest. 2018;153(4):1040-1051. doi:10.1016/j. chest.2017.08.1157
- Yang T, Li Z, Jiang L, Xi X. Corticosteroid use and intensive care unit-acquired weakness: a systematic review and meta-analysis. *Crit Care*. 2018;22(1):187. doi:10.1186/s13054-018-2111-0
- Jung B, Nougaret S, Conseil M, et al. Sepsis is associated with a preferential diaphragmatic atrophy: a critically ill patient study using tridimensional computed tomography. *Anesthesiology*. 2014;120(5):1182-1191. doi:10.1097/ALN.0000000000000001
- Shi ZH, Jonkman A, de Vries H, et al. Expiratory muscle dysfunction in critically ill patients: towards improved understanding. *Intensive Care Med*. 2019;45(8):1061-1071. doi:10.1007/s00134-019-05664-4
- Schepens T, Verbrugghe W, Dams K, Corthouts B, Parizel PM, Jorens PG. The course of diaphragm atrophy in ventilated patients assessed with ultrasound: a longitudinal cohort study. *Crit Care*. 2015;19:422. doi:10.1186/s13054-015-1141-0

- Demoule A, Jung B, Prodanovic H, et al. Diaphragm dysfunction on admission to the intensive care unit. Prevalence, risk factors, and prognostic impact-a prospective study. Am J Respir Crit Care Med. 2013;188(2):213-219. doi:10.1164/rccm.201209-1668OC
- Zambon M, Beccaria P, Matsuno J, et al. Mechanical Ventilation and Diaphragmatic Atrophy in Critically III Patients: An Ultrasound Study. Crit Care Med. 2016;44(7):1347-1352. doi:10.1097/ CCM.000000000000001657
- Papazian L, Forel JM, Gacouin A, et al. Neuromuscular blockers in early acute respiratory distress syndrome. *N Engl J Med*. 2010;363(12):1107-1116. doi:10.1056/ NEJMoa1005372
- 13. De Jonghe B, Sharshar T, Lefaucheur JP, et al. Paresis acquired in the intensive care unit: a prospective multicenter study. *JAMA*. 2002;288(22):2859-2867. doi:10.1001/jama.288.22.2859
- 14. Friedrich O, Reid MB, Van den Berghe G, et al. The Sick and the Weak: Neuropathies/Myopathies in the Critically III. *Physiol Rev.* 2015;95(3):1025-1109. doi:10.1152/physrev.00028.2014
- Lerolle N, Guérot E, Dimassi S, et al. Ultrasonographic diagnostic criterion for severe diaphragmatic dysfunction after cardiac surgery. *Chest*. 2009;135(2):401-407. doi:10.1378/chest.08-1531
- Umbrello M, Formenti P, Longhi D, et al. Diaphragm ultrasound as indicator of respiratory effort in critically ill patients undergoing assisted mechanical ventilation: a pilot clinical study. *Crit Care*. 2015;19(1):161. doi:10.1186/s13054-015-0894-9
- Batt J, Herridge M, Dos Santos C. Mechanism of ICU-acquired weakness: skeletal muscle loss in critical illness. *Intensive Care Med.* 2017;43(12):1844-1846. doi:10.1007/s00134-017-4758-4