Monaldi Archives for Chest Disease

eISSN 2532-5264 https://www.monaldi-archives.org/

Monaldi Arch Chest Dis 2025 [Online ahead of print]

To cite this Article:
Veluthat C, Venkatnarayan K, Selvam S, et al. **Diaphragm ultrasound as a predictor for the need for respiratory support at discharge in patients with exacerbation of chronic obstructive pulmonary disease**. *Monaldi Arch Chest Dis* doi: 10.4081/monaldi.2025.3614

Submitted: 23-06-2025
Accepted: 26-11-2025

Diaphragm ultrasound as a predictor for the need for respiratory support at discharge in patients with exacerbation of chronic obstructive pulmonary disease

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Contributions: CV, KV, UMK, concept and design, drafting of the article and revision; CV, KV, data collection; SS, CV, KV, analysis and interpretation of data; UMK, PR, SS, UD, manuscript editing and review, final approval of the version.

Conflict of interest: the authors declare that they have no competing interests, and all authors confirm accuracy.

Ethics approval and consent to participate: institutional Ethics Committee approved the study protocol and the IEC approval number is 131/2022.

Informed consent: informed consent was obtained from all patients before enrollment.

Patient consent for publication: consent was taken from patients before recruitment.

Availability of data and materials: all data underlying the findings are fully available.

Funding: none.

Acknowledgments: the authors acknowledge all the patients who participated in the study

Abstract
Diaphragm dysfunction during exacerbation of chronic obstructive pulmonary disease (COPD) has prognostic and therapeutic implications. The utility of the latter in predicting continued need for respiratory support at the time of discharge is worth exploring. The present study was carried out in a tertiary care teaching hospital with patients who were admitted to the ward or intensive care unit with exacerbations of COPD. The association between diaphragm function and the need for respiratory support at the time of discharge was assessed. All included participants underwent diaphragm ultrasound within 48-72 hours of admission. Diaphragm ultrasound was performed using the standard protocol wherein diaphragm excursion (DE), measured as the displacement of the diaphragm during inspiration and expiration; diaphragm thickening fraction (DTf), the fractional change of diaphragm thickness between inspiration and expiration; and ratio of inspiratory and expiratory diaphragm thickness (TR) were measured. The need for respiratory support [oxygen alone or oxygen and home non-invasive ventilation (NIV)] at the time of discharge was the outcome measured. Differences between various groups of respiratory support were analyzed using analysis of variance, Kruskal-Wallis, or Chi-square test, as appropriate. A total of 56 patients with exacerbation of COPD were included in the study. The median DE was 2.43 cm (interquartile range: 1.24, 3.33). The mean DTf (in %) was 52.25±34. On comparing the diaphragm function between the three outcome groups, patients requiring both oxygen and NIV at the time of discharge had a lower DTf and TR ($p=0.05$). Patients with an acute exacerbation of COPD requiring home oxygen and NIV support at discharge had a lower DTf and TR compared to those who were discharged without any respiratory support.

Key words: chronic obstructive pulmonary disease, diaphragm, exacerbation, ultrasound.

Introduction

Chronic obstructive pulmonary disease (COPD) remains a major global health concern and is projected to become the fourth leading cause of death by 2040 [1]. It is also the leading cause of respiratory-related mortality in India. Exacerbations of COPD (ECOPD), particularly those requiring hospitalization, contribute substantially to morbidity, mortality and healthcare costs and impose a major financial burden on the family [2]. These events also accelerate decline in lung function and increase the risk of future exacerbations, highlighting the need for early risk stratification and targeted interventions to improve outcomes [2]

Characterization and phenotyping of stable COPD with various predictors like gender, exacerbation status, comorbidities, biomarkers and lung functions have been undertaken in large cohorts of patients [3-7]. However, heterogeneity of COPD exacerbations has not been studied widely. Understanding the profile of disease exacerbations and identifying predictive and prognostic biomarkers are key research areas which would help in attaining this goal [8]. Current guidelines recommend systemic steroids, inhaled bronchodilators and antibiotics as the mainstay of therapy for patients with ECOPD [9]. Optimized treatment can be given by categorizing the patients based on etiology (infective vs non-infective), pathophysiology (inflammatory vs non-inflammatory, eosinophilic vs non eosinophilic) or clinical presentation (frequent exacerbator vs non-frequent exacerbator) [10]. Biomarkers that can predict outcomes such as the need for domiciliary oxygen or ventilatory support at discharge are particularly valuable in optimizing resource allocation and post-discharge planning.

One of the bedside modalities that can be used to assess need for respiratory support post-discharge from hospital is sonographic assessment of diaphragm function. Diaphragm is an important muscle of inspiration and plays a significant role in ventilation. Ultrasound is a non-invasive, bedside test that helps in identifying diaphragm dysfunction [11]. It has emerged as a non-invasive tool for evaluating respiratory muscle function and has been extensively studied in critical care for predicting weaning and extubation failure [12]. Recent evidence highlights its utility in ECOPD for predicting NIV failure and short-term mortality [13,14]. Diaphragm ultrasound has also been shown to be useful in differentiating stable COPD from those in an exacerbation and in studying the impact of rehabilitative interventions [15,16]. Continued need for respiratory support at discharge is a clinically important outcome which has significant implications on patient recovery, healthcare utilization and planning rehabilitative strategies. The potential role of diaphragm ultrasound in defining the above outcome remains to be established.

In this study, we assessed the clinical profile and diaphragm ultrasound characteristics of patients hospitalized with an exacerbation of COPD. We studied the association between diaphragm function, as an imaging biomarker, and the need for home oxygen and/or non-invasive ventilation (NIV) at discharge in a pilot sample of COPD patients.

Materials and Methods

The study was conducted in a tertiary care teaching hospital in South India between March 2022 and April 2023. Ethics approval by the Institutional Ethics Review Board was obtained prior to initiation of the study (Study no.131/2022). Patients with an exacerbation of COPD admitted to the ward and ICU were recruited after obtaining written informed consent. COPD exacerbation was diagnosed based on the GOLD criteria of worsening cough, dyspnea or sputum production within the past 14 days accompanied by tachypnea/ tachycardia. The diagnosis of COPD itself was made based on the history of a risk factor and clinical features of cough, dyspnea and wheeze.

Patients who had clinical conditions mimicking exacerbation of COPD (left ventricular failure, pneumothorax, worsening of underlying COPD secondary to pneumonia, pulmonary embolism and OHS with OSA) were excluded.

Ultrasonographic assessment of diaphragm function was performed by a single operator using Sonosite SII machine. Assessment of diaphragm using ultrasound was done within 48-72 hours of hospital admission in all patients. Diaphragm excursion (DE) was measured in the right subcostal area in semi recumbent posture during tidal breathing. The distance between the crest and trough during the displacement of diaphragm during tidal breathing was measured as DE (Figure 1). Thickening fraction (Figure 2) was measured at the zone of apposition (ZOA). Hypoechoic area between the echogenic peritoneal line and pleural line was identified as the diaphragm at the ZOA. Diaphragm Thickening fraction (DTf) was calculated using the following formula,

Diaphragm thickening fraction = 100 *[(Thickness during inspiration - Thickness during expiration) / Thickness during expiration]

Diaphragm thickening ratio (TR) was measured as the ratio of diaphragm thickness during inspiration and thickness during expiration.

Patients were categorized into three groups based on the need for a respiratory support at the time of discharge as follows:

- Home oxygen group – Patients whose SPO2 was less than 88% at the time of discharge.

- Home oxygen and NIV group – Patients whose SPO2 was less than 88% at the time of discharge and persisting hypercapnia (PaCO2> 55 mm Hg) with a plan to assess later on outpatient basis for the need for long term home NIV
- None – those who did not require either oxygen or NIV at discharge.

Statistical analysis

All data was collected and managed on Microsoft Excel worksheet. Normality was assessed using Q-Q plot. Continuous variables were expressed as mean±standard deviation (SD) or median with 25^{th} and 75^{th} percentile value. Categorical variables were summarized using frequency and percentages. Association between clinical characteristics and discharge outcome (None, Both NIV and O2 and only home O2) was assessed using Pearson's Chi-square tests, ANOVA and Kruskal Wallis test, as appropriate. Multiple comparison test was done using Bonferroni correction and Mann Whitney U test. A two-sided p value of less than 0.05 was considered statistically significant. The software used for analysing was IBM SPSS Statistics (29.0).

Results

Fifty-six patients with exacerbation of COPD were included in the pilot study. Mean age of the patients was 65 years. Three-fourths of the study population were males and 70% and were ever-smokers. The most common co-morbidity was hypertension (50%). Type 1 respiratory failure was seen in 92% while 57% of the patients had type 2 respiratory failure at admission. The median diaphragm excursion (DE) in our study population was 2.43 cm (IQR 1.24, 3.33). The mean thickening fraction was 52.25±34. Table 1 depicts the baseline characteristics of patients with exacerbation of COPD.

Among the 56 patients included in the study, 17 patients were discharged on oxygen support, 20 required both O2 and NIV and 19 did not require any support. A significantly higher proportion of females required both O2 and NIV at discharge whereas a higher proportion of males were discharged with home oxygen alone (p=0.01). Most of the patients who required both O2 and NIV at discharge had type 2 respiratory failure at admission (p<0.001). The mean PaCO2 in this group was significantly higher compared to the other two groups (66.5 mm Hg (p=0.002). On comparing the diaphragm function between the three groups, patients requiring both O2 and NIV at the time of discharge had a lower diaphragm thickening fraction and thickening ratio(p=0.05). Comparison of patient characteristics based on the discharge outcome are summarized in Table 2.

Discussion

In the era of precision medicine, an individualized treatment plan for exacerbation of COPD is the need of the hour [8]. Exacerbation of COPD is an event that is characterized by increased airway inflammation leading to mucus production along with air trapping secondary to bronchoconstriction [9]. In patients with COPD, the diaphragm is at a mechanical disadvantage secondary to dynamic hyperinflation and this is further accentuated during an exacerbation [14].

It is reported that a quarter to one-third of patients presenting to the emergency with exacerbation of COPD have diaphragm dysfunction [11,12]. The indices of diaphragm function measured are diaphragm excursion (DE) during tidal breathing and deep breathing and sniff manoeuvre, diaphragm thickening fraction (DTf) and thickening ratio (TR). Although normative values have been reported in various populations, no definite cut-off values for any of these have been validated in stable COPD or exacerbation [11].

In our study, diaphragm function as measured by diaphragm thickening fraction (DTf) and thickening ratio (TR) was lower in patients who required both home oxygen and NIV at the time of discharge, ($p=0.05$). Although diaphragm excursion (DE) was lesser in patients who were discharged on home oxygen and NIV, the difference was not statistically significant. In addition, 90% of patients who were discharged on both home oxygen and NIV had type II respiratory failure and a higher mean $PaCO_2$ at the time of admission. It is possible that the diaphragm dysfunction may have added to the existing ventilatory impairment in this group.

Studies on exacerbations of COPD have commonly defined diaphragm dysfunction as diaphragm thickening fraction (DTf) less than 20% [11,12]. In our study, however, the median DTf among patients requiring both oxygen and NIV at discharge was 23.52%. Notably, an Indian study reported a DTf cut-off of 35.3% for predicting NIV failure in ECOPD patients [17]. These findings suggest that the current threshold of DTf < 20% may underestimate diaphragm dysfunction during exacerbation of COPD. Establishing an optimal cut-off value could aid in early identification of patients who may need domiciliary respiratory support and diaphragm-targeted rehabilitation strategies.

Diaphragm, being the predominant muscle of inspiration, is increasingly recognised as the target for comprehensive pulmonary rehabilitation. The recommended diaphragm strengthening strategies are inspiratory muscle training, manual therapy and phrenic nerve stimulation. Few studies have shown improvement in mMRC grade of dyspnea, 6-minute walk distance and lung function following diaphragm specific rehabilitation interventions. However, these intervention studies were done on smaller number of patients. Diaphragm ultrasound is an ideal investigation to measure such treatment interventions because of its non-invasive nature and repeatability [18].

Our study had various limitations. In our study, diaphragm assessment was not done at a specific timeline. We defined an arbitrary timeline of 48 – 72 hours within the admission to measure the diaphragm function. Defining a definite timeline for diaphragm assessment or evaluating change in diaphragm function overtime during the hospital stay could provide further insights on its utility in predicting the need for respiratory support at discharge. Left diaphragm assessment was not done as a part of the study as visualisation of left diaphragm is technically challenging. Evaluating both sides might have given a clearer and complete picture. A larger sample size would further validate the results obtained in our study and help in planning targeted interventions.

Conclusions

Patients with an acute exacerbation of COPD requiring home oxygen and non-invasive ventilatory support at discharge, had a lower diaphragm thickening fraction and thickening ratio compared to those who were discharged without any respiratory support. Early identification of diaphragm dysfunction during an exacerbation may enable timely initiation of rehabilitative interventions targeted at improving diaphragm function.

References

1. Foreman KJ, Marquez N, Dolgert A, et al. Forecasting life expectancy, years of life lost, and all-cause and cause-specific mortality for 250 causes of death: reference and alternative scenarios for 2016-40 for 195 countries and territories. Lancet 2018;392:2052-90.
2. Andersson F, Borg S, Jansson SA, et al. The costs of exacerbations in chronic obstructive pulmonary disease (COPD). Respir Med 2002;96:700-8.
3. Fermont JM, Masconi KL, Jensen MT, et al. Biomarkers and clinical outcomes in COPD: a systematic review and meta-analysis. Thorax 2019;74:439-46.
4. Burgel PR, Paillasseur JL, Roche N. Identification of clinical phenotypes using cluster analyses in COPD patients with multiple comorbidities. Biomed Res Int 2014;2014:420134.
5. Foreman MG, Zhang L, Murphy J, et al. Early-onset chronic obstructive pulmonary disease is associated with female sex, maternal factors, and African American race in the COPDGene Study. Am J Respir Crit Care Med 2011;184:414-20.

6. Hurst JR, Han MK, Singh B, et al. Prognostic risk factors for moderate-to-severe exacerbations in patients with chronic obstructive pulmonary disease: a systematic literature review. Respir Res 2022;23:213.
7. Kumar P, Law S, Sriram KB. Evaluation of platelet lymphocyte ratio and 90-day mortality in patients with acute exacerbation of chronic obstructive pulmonary disease. J Thorac Dis 2017;9:1509-16.
8. Leung JM, Obeidat M, Sadatsafavi M, et al. Introduction to precision medicine in COPD. Eur Respir J 2019;53:1802460.
9. Global Initiative for Chronic Obstructive Lung Disease. Global strategy for the prevention, diagnosis and management of COPD: 2025 report. Available from: https://goldcopd.org/2025-gold-report/.
10. Zhou A, Zhou Z, Zhao Y, et al. The recent advances of phenotypes in acute exacerbations of COPD. Int J Chron Obstruct Pulmon Dis 2017;12:1009-18.
11. Tuinman PR, Jonkman AH, Dres M, et al. Respiratory muscle ultrasonography: methodology, basic and advanced principles and clinical applications in ICU and ED patients-a narrative review. Intensive Care Med 2020;46:594-605.
12. Llamas-Álvarez AM, Tenza-Lozano EM, Latour-Pérez J. Diaphragm and lung ultrasound to predict weaning outcome: systematic review and meta-analysis. Chest 2017;152:1140-50.
13. Antenora F, Fantini R, Iattoni A, et al. Prevalence and outcomes of diaphragmatic dysfunction assessed by ultrasound technology during acute exacerbation of COPD: a pilot study. Respirology 2017;22:338-44.
14. Marchioni A, Castaniere I, Tonelli R, et al. Ultrasound-assessed diaphragmatic impairment is a predictor of outcomes in patients with acute exacerbation of chronic obstructive pulmonary disease undergoing noninvasive ventilation. Crit Care 2018;22:109.
15. Crimi C, Heffler E, Augelletti T, et al. Utility of ultrasound assessment of diaphragmatic function before and after pulmonary rehabilitation in COPD patients. Int J Chron Obstruct Pulmon Dis 2018;13:3131-9.
16. An TJ, Yoo YJ, Lim JU, et al. Diaphragm ultrasound is an imaging biomarker that distinguishes exacerbation status from stable chronic obstructive pulmonary disease. Int J Chron Obstruct Pulmon Dis 2022;17:3-12.
17. Patel NB, Jain G, Chauhan U, et al. Evaluating diaphragmatic dysfunction and predicting non-invasive ventilation failure in acute exacerbation of chronic obstructive pulmonary disease in India. Acute Crit Care 2023;38:200-8.

18. Chen Y, Li P, Wang J, et al. Assessments and targeted rehabilitation therapies for diaphragmatic dysfunction in patients with chronic obstructive pulmonary disease: a narrative review. Int J Chron Obstruct Pulmon Dis 2022;17:457-73.

Table 1. Baseline characteristics of patients with exacerbation of COPD

Characteristics (N=56)	**Results**
Age (years) (mean±SD)	65.21±8.40
Sex (males) [n (%)]	43 (76.8)
Ever-smokers [n (%)]	40 (71.4)
Comorbidities [n (%)] • Diabetes mellitus • Hypertension • Ischemic Heart Disease • Cerebrovascular accident	 15 (26.8) 28 (50) 11 (19.6) 2 (3.6)
Frequent exacerbator [n (%)]	26 (46.4)
History of tuberculosis [n (%)]	14 (25)
Type 1 respiratory failure at admission [n (%)]	51 (91.1)
Type 2 respiratory failure at admission [n (%)]	32 (57.1)
Duration of COPD (years) (mean±SD)	5.5±4.5
Admission PaCO2 (mm Hg) (mean±SD)	57.91±20.9
Diaphragm excursion (DE) (cms) (mean±SD)	2.36±1.27
Thickening fraction (DTf) (%) (mean±SD)	51.71±32.13
Thickening ratio (TR) (mean±SD)	1.51±0.32

Table 2. Characteristics of patients based on the discharge outcome

	Home oxygen (n=17)	**Home oxygen and NIV (n=20)**	**None (n=19)**	**p**
Age (years) (mean±SD)	65.7±9.3	67.25±7.9	62.63±7.6	0.223
Sex (males) [n (%)]	15(88.2)	11(55)	17 (89.5)	**0.016**
Ever-smokers [n (%)]	15 (88.2)	10 (50)	15 (78.9)	**0.025**
Frequent exacerbator [n (%)]	9 (52.9)	11 (55.0)	6 (33.3)	0.350
Type 1 respiratory failure at admission [n (%)]	16 (94.1)	19 (100)	16 (84.2)	0.167
Type 2 respiratory failure at admission [n (%)]	8 (47.1)	18 (90)	6 (31.6)	**<0.001**
Duration of COPD (years) [Median (IQR)]	3 (6,10)	4.5 (1,9.2)	4 (0.8,6.5)	0.128
Admission PaCO2 (mm Hg) (mean±SD)	54.85±22.19	66.5±18.18	47.92±19.39	**0.032**
Diaphragm excursion (DE) (cms) [median (IQR)] 95% CI	2.64 (1.52, 3.25) 1.85-3.19	1.46(1.00, 2.74) 1.45-2.9	2.62 (1.75, 3.46) 2.01-3.16	0.120
Thickening fraction (DTf) (%) (median, IQR) 95% CI	52.17 (33.3, 61.1) 39.11-55.87	23.52 (15.15, 61.8) 22.79-61.15	61.1(36.8,80.76) 47.10-80.42	**0.05**
Thickening ratio (TR) (mean±SD) 95% CI	1.47±0.15 1.39-1.54	1.41±0.37 1.25-1.57	1.63±0.34 1.48-1.78	**0.05**

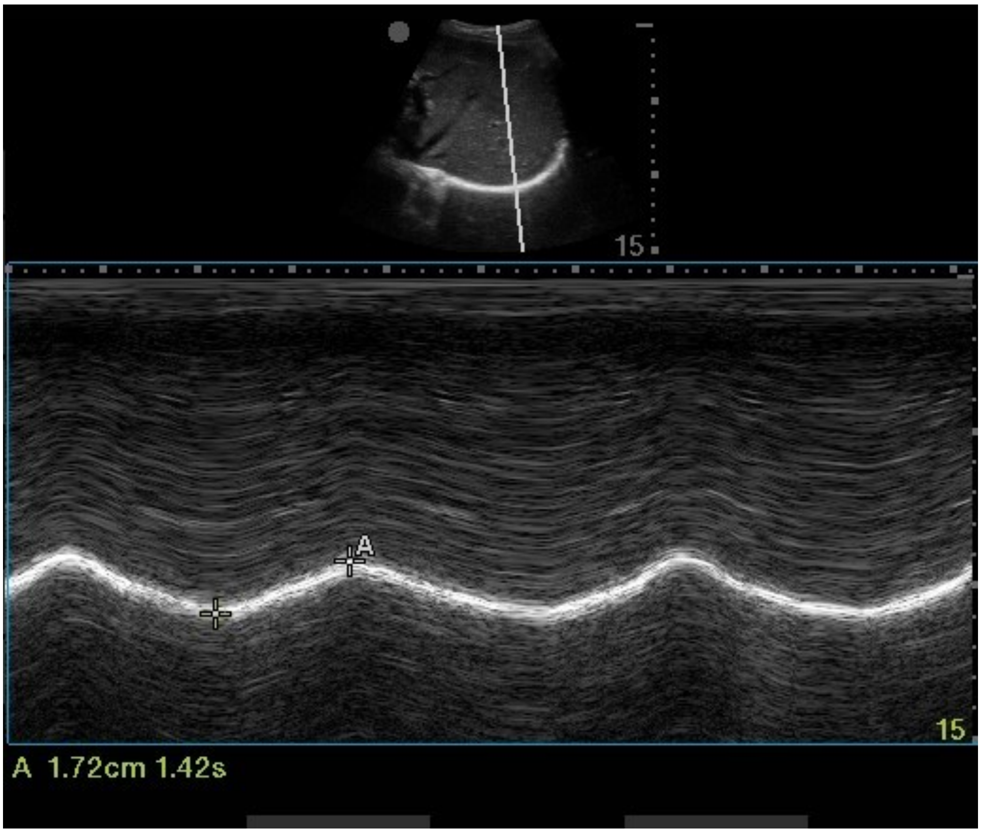

Figure 1. Diaphragm excursion measured using a curvilinear probe in semirecumbent posture during tidal breathing. The distance from crest to trough is measured as the excursion in cm (DE=1.72cm).

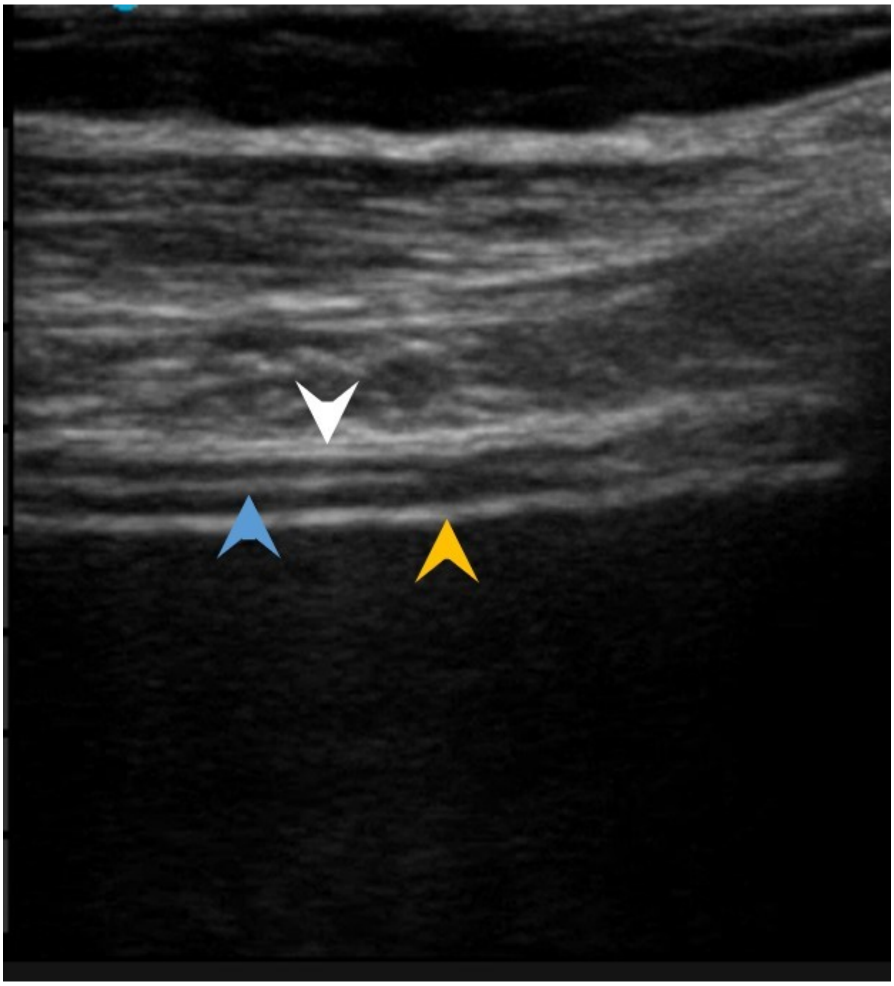

Figure 2. Diaphragm identified as a three-layered structure using linear probe in semirecumbent posture during tidal breathing. Yellow arrowhead: peritoneal line, white arrowhead: pleural line, blue arrowhead: central tendon of diaphragm.