The role of Point of Care Ultrasound (POCUS) and focused echocardiography in optimization of non-invasive mechanical ventilation: from diaphragmatic functionality to hemodynamic monitoring

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Abstract

This case shows the use of ultrasound guidance to optimize non-invasive mechanical ventilation for a 62-year-old patient with a complex medical history. Point-of-care ultrasound (POCUS) was used to assess diaphragmatic function and hemodynamics, leading to adjustments in ventilator setting. The approach improved gas exchange, resolved respiratory acidosis, and enhanced hemodynamics, providing a promising strategy for ventilator management in complex clinical cases.

Keywords: Non-Invasive Mechanical Ventilation, Point-of-Care Ultrasound, Diaphragmatic Ultrasound, Focused Echocardiography, Ventilator-Induced Diaphragmatic Dysfunction, Hemodynamics.

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Introduction

In recent years, Point-of-Care Ultrasound (POCUS) and Focused Cardiac Ultrasound (FOCUS) have emerged as valuable tools for the management of patients in mechanical ventilation. These ultrasound techniques provide real-time imaging at the bedside, avoid the use of radiation and allow clinicians to assess lung and cardiac function promptly. Diaphragmatic ultrasound (DUS) is an application of POCUS that facilitates the assessment of diaphragm dynamics during mechanical ventilation while FOCUS plays a pivotal role in evaluating cardiac function, identifying signs of hemodynamic instability, and guiding fluid management strategies.
The integration of both techniques, DUS and FOCUS, into mechanical ventilation practices enhances diagnostic accuracy and contributes to more informed decision-making in the critical care setting.

The significance of this case lies in the integration of ultrasound techniques to fine-tune NIV in complex clinical scenarios, potentially preventing ventilator-induced diaphragmatic dysfunction (VIDD) and ensuring optimal ventilation settings. This approach is not standardized in the literature and offers a promising avenue for future research and clinical practice.

Case

A 62-year-old man with a history of hypertension, severe pulmonary emphysema, chronic obstructive pulmonary disease (COPD), type II diabetes mellitus, severe dilated cardiomyopathy with implanted ICD- Pacemaker presented to the emergency department with fever, dyspnea, and significant hypoxemia.

A chest computerized tomography (CT) scan revealed a large lobar pneumonia in the right lower lobe, bilateral pleural effusion, and diffuse centrolobular and paraseptal emphysema in both lungs.

The patient was admitted to our sub-intensive respiratory department, where sputum cultures, blood cultures, and broad-spectrum antibiotic therapy were initiated along with diuretic therapy.

Upon admission to the department, a point-of-care ultrasound (POCUS) was performed, showing a significant consolidation area in the right PLAPS point (posterolateral alveolar or pleural syndromes), a reduced diaphragm motility, the presence of focal B-lines associated with irregular and thickened pleura bilaterally, sparing areas, a 11 mm inferior vena cava (IVC) with a collapsibility index (CI) >50%. Focused echocardiography (FOCUS) showed a dilated and diffusely hypokinetic left ventricle with severe reduction in ejection fraction (EF), normal right chambers with good right ventricular systolic function with a Tricuspid annular plane systolic excursion (TAPSE) of 22, no evidence of elevated pulmonary pressure and a mild, concentric pericardial effusion without hemodynamic significance.

Due to worsening of general clinical conditions in the 48 hours following admission and the development of septic shock, it became necessary to initiate supportive therapy with steroids, inotropes, and vasopressors. Additionally, respiratory support with high-flow oxygen (HFNC) was initiated, maintaining good blood gas compensation. After a few hours, due to a worsening trend in PaCO2 levels, the onset of respiratory acidosis, and signs of respiratory fatigue, non-invasive mechanical ventilation (NIV) was started.

A double-tube circuit (with one inspiratory and one expiratory line) connected to a full-face mask was used. No catheter mount was used to minimize resistance and dead space. The ventilation mode used was spontaneous pressure support ventilation (PSV). The expiratory trigger was set at 40% of the peak inspiratory flow, and the inspiratory trigger was adjusted using the highest sensitivity while avoiding auto-triggering. Inspiratory rise time was set to the most rapid settings to match the patient's demand. Backup respiratory rate (BURR) was set at 14 breaths/minute. The target pressure support (PS) was set at 14 cm H_2O, and the expiratory pressure (PEEP) was set at 7 cm H_2O. The obtained tidal volume (V_T) was approximately 6 ml/kg. The recorded leaks were <10%. FiO_2 was set at 28%. The patient was compliant with ventilation, showing good ventilator monitoring curves, adequate volumes, SpO_2 of 95% and a spontaneous respiratory rate of about 16 breaths per minute. Blood gas compensation was achieved after a few hours of NIV.

Subsequently, a weaning process from mechanical ventilation was initiated using an alternating strategy between NIV and HFNC. After approximately 72 hours, the alternation between HFNC and NIV became increasingly difficult as the patient showed progressive deterioration in general conditions, sarcopenia, and motor strength.

Due to the development of respiratory acidosis during HFNC treatment and signs of respiratory fatigue, a prolonged session of NIV followed by a difficult weaning process was initiated. The effectiveness of the ventilation strategy was not as clear this time, as the respiratory acidosis did not completely improve even after several hours of ventilation, despite multiple attempts to modify ventilator settings. Hemodynamic instability with marked hypotension (MAP <65 mmHg) and reduced diuresis also occurred, which was not entirely explainable by the patient's sepsis.

Given the poor response to NIV, a new modification of the ventilator parameters was attempted, this time using ultrasound guidance to carefully assess diaphragmatic excursions and hemodynamic parameters in response to changes in ventilator pressures.

Specifically, the ventilator parameters used up to that point (PSV mode with PS 14, PEEP 7, FiO_2 28%; BURR 15, V_T approximately 6 ml/kg) resulted in a severe reduction in diaphragmatic excursion during ventilation, falling below the threshold values defined in the literature as indicative of severe diaphragmatic dysfunction/paralysis, [1] and indicative of over-assistance of the ventilator to the patient. Additionally, the application of FOCUS revealed a reduction in the Left Ventricular Outflow Tract Velocity Time Integral (LVOT-VTI), likely due to excessive PEEP.

The patient had a peripheral SpO_2 of 94% on NIV enriched with 28% FiO_2 (SpO_2/FiO_2 ratio of 335). Before modifying the ventilator parameters, an arterial blood gas analysis was performed on NIV with FiO_2 28%, which showed a pH of 7.28, $PaCO_2$ of 76, PaO_2 of 69, HCO_3 of 36, and lactate of 0.4.

A diaphragmatic ultrasound was then performed using a convex probe with a frequency of 3.5-5 MHz, scanning the right subcostal ascending region at the level of the hemi-clavicular line. M-Mode was used to assess the excursions of the right hemidiaphragm during the patient's respiratory cycles. During the delivery of pressure support (PS) of 14 cmH_2O, the right hemidiaphragm had an inspiratory excursion of 0.9 mm (Figure 1). The progressive reduction of the PS value to 10 cmH_2O, guided by ultrasound measurements of diaphragmatic excursions, resulted in an increase in the right hemidiaphragm excursion to 12.9 mm (Figure 2). This was achieved while maintaining good lung volumes (approximately V_T 450- 500 ml), a spontaneous respiratory rate of 17 breaths per minute, and a peripheral SpO_2 of 93%, despite reducing FiO_2 to 21% (SpO_2/FiO_2 ratio of 442).

Later, a FOCUS was performed using a 5 MHz phase array cardiac probe, aimed at measuring the LVOT-VTI. During the delivery of positive end-expiratory pressure (PEEP) of 7 cmH_2O, the measured LVOT-VTI was 18 cm, which was at the lower limits of normal (Figure 3). PEEP was then progressively reduced, with serial echocardiographic measurements of LVOT-VTI. The reduction of PEEP values delivered by the ventilator to 5 cmH_2O resulted in maintaining peripheral SpO_2 values at 93%, an increase in LVOT-VTI from 18 cm to 21 cm (Figure 4), and an increase in MAP > 65 mmHg, improving the patient's hemodynamics and recovery of diuresis.
A blood gas analysis was performed 2 hours after modifying the ventilation parameters (PS 10, PEEP 5, FiO_2 21%), yielding the following results: pH 7.35, $PaCO_2$ 63, PaO_2 62, SaO_2 93%, Lactate 0.5.

In conclusion, the modification of non-invasive mechanical ventilation parameters based on ultrasound evaluation of diaphragmatic excursions and LVOT-VTI improved gas exchange, resolved uncompensated respiratory acidosis, reduced CO2 levels, improved diaphragmatic excursions, and enhanced patient hemodynamics while maintaining stable peripheral oxygen saturation values and lactate levels.

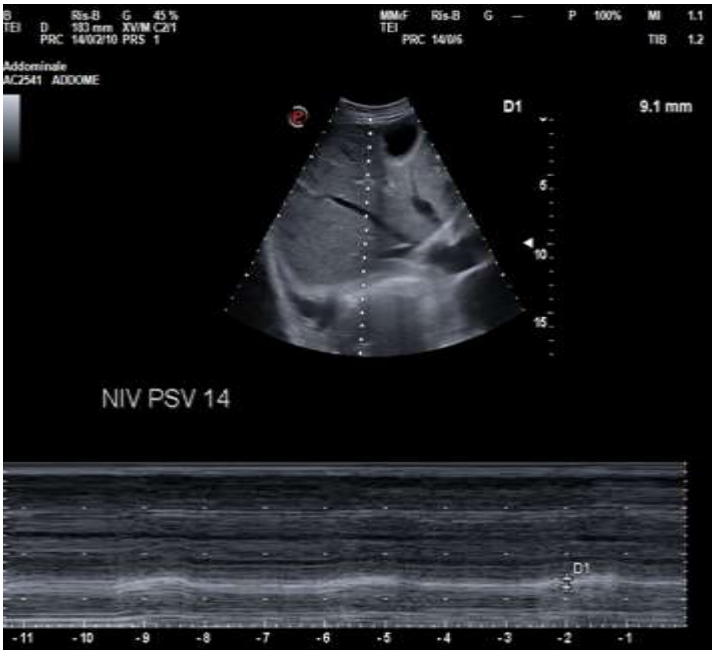

Figure 1. M-Mode Ultrasonographic evaluation of right hemidiaphragm excursions during NIV with a pressure support (PS) of 14 cmH_2O shows a measurement of 0.9 cm, a threshold value suggestive of diaphragmatic paralysis

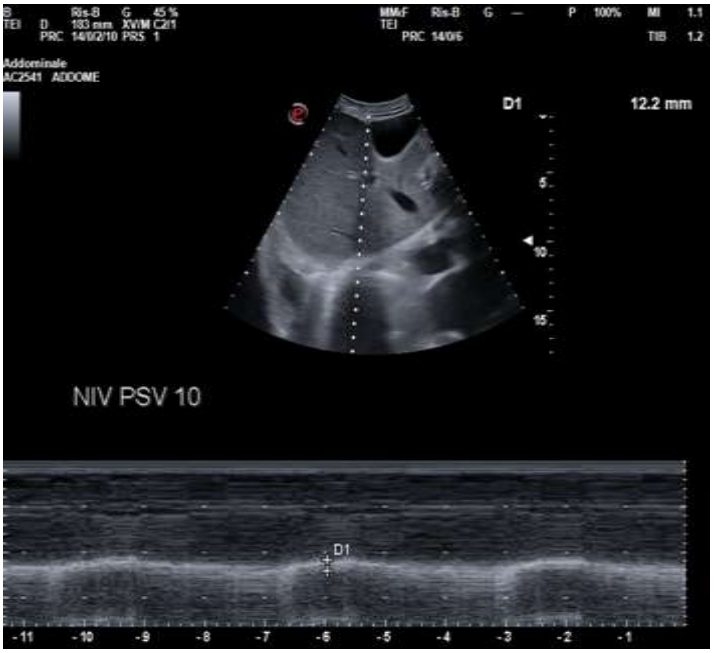

Figure 2. M-Mode Ultrasonographic evaluation of right hemidiaphragm excursions during NIV with a pressure support (PS) of 10 cmH_2O shows a measurement of 1.22 cm, a value within the normal range.

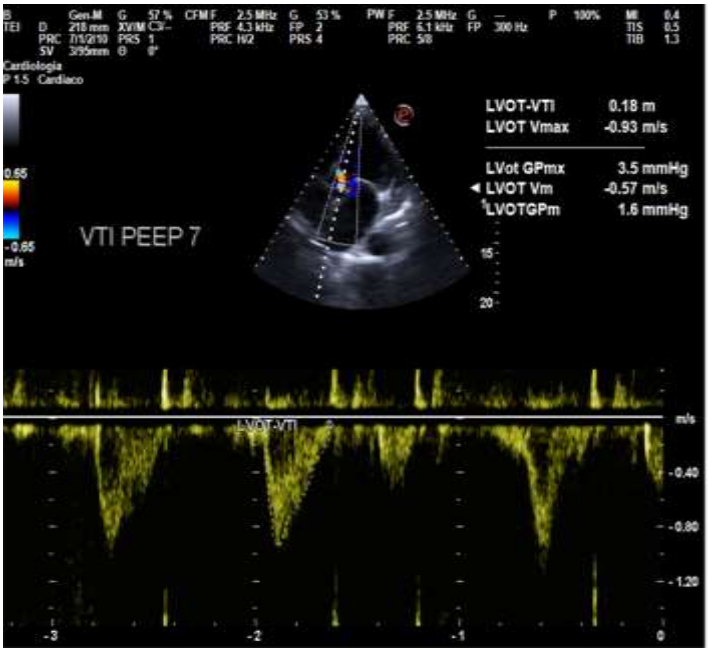

Figure 3. Left Ventricular Outflow Tract - VTI (LVOT-VTI) measured during PEEP 7 cmH_2O: the measured value is 18 cm, at the lower limits of the normal range.

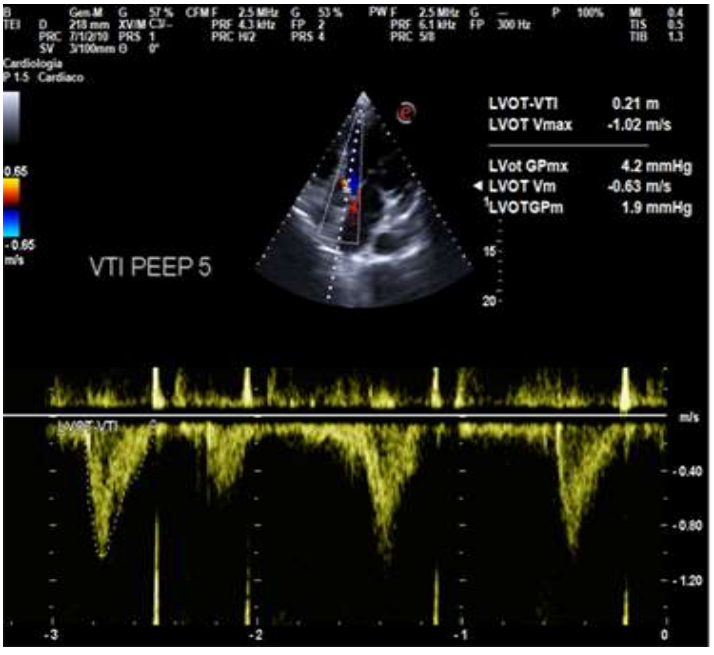

Figure 4. LVOT (Left Ventricular Outflow Tract) measured after reducing PEEP to 5 cmH_2O, the measured value is 21 cm, increased compared to the previous assessment

Discussion

The case presented herein illustrates a situation where the use of POCUS allowed for optimizing ventilator settings and improving overall patient management by addressing refractory respiratory acidosis. Non-invasive mechanical ventilation is a well-established and increasingly utilized method in emergency departments, intensive care units, and sub-intensive respiratory therapy for treating patients with acute type I and particularly type II respiratory failure related to COPD exacerbations or pneumonia [2-4]. In patients responsive to NIV treatment, improvements in pH values or respiratory rate have been demonstrated within the first 4 hours of mechanical ventilation. [2]

NIV has been shown to be useful in improving gas exchange, reducing the likelihood of endotracheal intubation, and improving survival in these patients. [5-6] Conversely, patients who do not respond to initial NIV treatment and require endotracheal intubation have worse outcomes. [7]

Despite its benefits and positive aspects, mechanical ventilation can contribute to atrophy of the diaphragm muscle fibers in ventilated patients, leading to a reduction in the muscle's ability to generate contractile strength, a condition known as "ventilator-induced diaphragmatic dysfunction (VIDD)," which can occur after just 18 hours of mechanical ventilation. [8-9] Recent evidence has also shown that diaphragmatic dysfunction is frequently associated with weaning failure [10-11] and is associated with a poor prognosis at the time of ventilation withdrawal. [12-13]

Another aspect to consider regarding the use of NIV and its potential risks is the effect on hemodynamics during mechanical ventilation. Specifically, the literature has shown that positive end-expiratory pressure (PEEP), by increasing transpulmonary pressure, leads to a simultaneous increase in right atrial pressure. This effect results in a reduction in pressure gradient, right ventricular filling, and consequently, stroke volume (SV). [14-16] This aspect is particularly relevant in patients with right atrial pressure <10 mmHg, which correlates with a high degree of inferior vena cava collapsibility and is closely related to circulatory volume. Patients with hypovolemia, with higher PEEP applications, will experience a greater reduction in SV. [17-18]

In recent years, the use of point-of-care ultrasound (POCUS) for the management of critically ill patients has become increasingly widespread among intensivists and emergency physicians. The most commonly used POCUS applications in the intensive care setting include bedside cardiac, pulmonary, and abdominal ultrasounds. [19-20] One of the rapidly growing applications of POCUS is diaphragmatic ultrasound (DUS), a useful tool for assessing diaphragmatic morphology and function, which has gained significance in the critical care patient population due to its numerous advantages. These include the simplicity of the machines required to perform DUS, which allows for execution at the patient's bedside, and the standardization of the ultrasound technique, enabling rapid, precise, repeatable, and reproducible assessments. [21-23]

Mastery of this technique allows for the rapid diagnosis and assessment of respiratory muscle dysfunction in critical patients, those undergoing mechanical ventilation, and those with newly onset dyspnea. Furthermore, it can be used to assess patient-ventilator interaction and weaning failure. Regarding hemodynamic monitoring in critically ill patients, the measurement of stroke volume (SV) and cardiac output (CO) are useful parameters for assessing tissue perfusion and oxygen distribution. [24] Although there are multiple valid methods for performing these measurements, the use of bedside transthoracic echocardiography (TTE), also known as FOCUS, is gaining more acceptance and use among intensivists and emergency physicians.

While the pulmonary artery catheter (PAC) is currently considered the gold standard for hemodynamic monitoring of critically ill patients, its utility has been questioned due to an unfavorable risk-to-benefit ratio, often leading to the abandonment of this method. [25-26] To address this issue, the latest guidelines from the American Society of Echocardiography recommend using both transthoracic echocardiography (TTE) and/or transesophageal echocardiography (TEE) to assess SV and CO in determining responses to medical and surgical therapies. The evaluation of these hemodynamic parameters via TTE is considered a non-invasive, cost-effective, bedside procedure, free of ionizing radiation, continuous or rapidly repeatable, reproducible, and reliable during various pathophysiological states. [27] Additionally, TTE provides the opportunity to correlate SV or CO with the causative factors, such as hypovolemia, the presence of cardiac dysfunction, cardiac tamponade, acute cor pulmonale, or vasodilation. [28]

Using TTE, SV is calculated as the product of the cross-sectional area (CSA) of the left ventricular outflow tract (LVOT) and the velocity-time integral

(VTI) of the LVOT. CO is the result of the product of SV and heart rate (HR). However, since this measurement can be subject to potential inaccuracies, an approach that can be executed during FOCUS to overcome such inconveniences has been proposed, especially when calculating LVOT-CSA. Considering the LVOT-CSA as constant, any change in SV and CO depends strongly on variations in LVOT-VTI. Therefore, the calculation of LVOT-VTI alone is an excellent surrogate for calculating SV and is extremely useful for SV monitoring. This approach, used in clinical practice, is useful for assessing the patient's response to therapeutic interventions such as the administration of inotropic agents and fluids. [29-30] LVOT-VTI variation has also been studied in mechanically ventilated critically ill patients with different levels of PEEP, demonstrating that changes in LVOT-VTI values between PEEP 10 cmH2O and PEEP 0 cmH_2O are useful for predicting fluid responsiveness in patients. [31]

In the present case, the combined use of both POCUS methodologies allowed for the optimization of ventilator management by objectively assessing the patient's response to changes in ventilator settings. Currently, there is no standardized and integrated approach in the literature for the ultrasound monitoring of diaphragmatic and hemodynamic responses in mechanically ventilated patients.

The use of DUS and FOCUS in the case we presented could be beneficial in optimizing mechanical ventilation settings, especially in critically ill patients.

Firstly, DUS performed at the patient's bedside during mechanical ventilation documented a state of over assistance by the ventilator, with extremely reduced diaphragmatic excursions, approaching diaphragmatic paralysis. The progressive reduction of delivered PS resulted in increased diaphragmatic excursion up to 12.9 mm, a value associated with NIV success and reduced $PaCO_2$ values after one hour in the literature ,[32] as demonstrated by the actual improvement in blood gas analysis in our patient.

Secondly, serial measurements of LVOT-VTI via FOCUS allowed for titration and progressive reduction of PEEP delivered to the patient, objectively achieving the "best PEEP" in light of improved SV, hemodynamics and blood gas parameters.

Conclusions

The combined ultrasound approach of DUS and FOCUS for LVOT-VTI assessment can be useful for setting ventilator parameters in complex patient ventilation cases. Specifically, in our case, we dealt with a sarcopenic COPD patient with diaphragmatic weakness and a high risk of VIDD, associated with severe heart disease, where NIV could play a crucial role in worsening respiratory mechanics and hemodynamics.

The use of these techniques, which are useful for monitoring diaphragmatic function during ventilation, can lead to the development of ventilatory strategies aimed at preventing VIDD and protecting the diaphragm during mechanical ventilation, primarily based on the titration of appropriate levels of inspiratory effort (to avoid over- and under-assistance). Such strategies are not yet standardized in the literature.

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