Clinical and Pharmaceutical Sciences Journal

Review article

Diaphragmatic Dysfunction across the COPD Continuum: From Pathophysiology to Rehabilitation and Mechanical Ventilation

Marwa Mohammed[1,2*], Taha Hussein Musa[3]
[1]Faculty of Physical Therapy, Beni-Suef University, Egypt
[2]Faculty of Allied Medical Sciences, Department of Physiotherapy, Applied Science Private University, Amman, Jordan
[3]School of Medicine, Darfur University College, Nyala, Sudan

*Corresponding Author: m_elbelbessy@asu.edu.jo

Received: 20 April 2026 | Revision: 19 June 2026 | Accepted: 30 June 2026

Abstract

Chronic obstructive pulmonary disease (COPD) remains a leading cause of morbidity and mortality worldwide, frequently diagnosed at advanced stages and often accompanied by significant extrapulmonary complications. Amongst these, diaphragmatic dysfunction plays a central yet often underrecognized role in disease progression, respiratory failure, and poor clinical outcomes. This review synthesises current evidence on diaphragmatic dysfunction in COPD and mechanically ventilated (MV) patients, evaluates diagnostic techniques, and explores therapeutic interventions, particularly pulmonary rehabilitation. A structured literature search of PubMed, Scopus, and Web of Science was conducted between January 2000 and March 2026. Two independent reviewers screened titles, abstracts, and full texts against predefined eligibility criteria. Studies addressing diaphragmatic dysfunction in patients with COPD or MV were included. Findings were synthesised narratively and supplemented by the GOLD 2026 report. No formal risk of bias assessment was performed, consistent with narrative review methodology. Diaphragmatic dysfunction in COPD is characterised by muscle fibre atrophy, fibre-type shifting, hyperinflation-induced shortening, and reduced force-generating capacity, leading to dyspnea, respiratory failure, and increased mortality. Diagnostic approaches include transdiaphragmatic pressure measurement (gold standard), ultrasound, chest radiography, computed tomography (CT), Magnetic resonance imaging (MRI), and fluoroscopy. Pulmonary rehabilitation improves diaphragmatic strength, exercise tolerance, dyspnea, and quality of life. GOLD 2026 supports home-based and tele-rehabilitation as effective alternatives to centre-based programs. However, significant barriers persist, including underdiagnosis, insufficient funding, limited provider awareness, and inequitable access to rehabilitation services. Diaphragmatic dysfunction is an important yet underdiagnosed complication of COPD and MV. Early recognition using appropriate diagnostic tools, combined with targeted rehabilitation, can improve functional outcomes. Future research should optimise stimulation techniques, standardise tele-rehabilitation protocols, and expand evidence to post-exacerbation and multimorbid populations.

Keywords: Chronic obstructive pulmonary disease; diaphragmatic dysfunction; mechanical ventilation; pulmonary rehabilitation; ventilator-induced diaphragmatic dysfunction.

Introduction

The diaphragm is a musculotendinous structure that separates the thoracic cavity from the abdominal cavity and serves as the primary muscle of respiration. It has a dome-like shape and is covered by the pleura on its thoracic surface and by the peritoneum on its abdominal surface. Structurally, the diaphragm consists of a peripheral muscular portion, which attaches to the ribs, and a more central tendinous portion [1]. The diaphragm serves as the primary inspiratory muscle and the principal respiratory muscle during quiet breathing. Diaphragmatic dysfunction can be defined as a partial or complete loss of diaphragm function, potentially affecting one or both hemidiaphragms [2]. In individuals with COPD, diaphragm dysfunction most commonly presents as unilateral diaphragmatic paralysis, which is often asymptomatic. Functionally, COPD patients with diaphragmatic impairment typically exhibit reduced diaphragmatic strength, altered endurance capacity, decreased mobility, and muscle fatigue.

Chronic obstructive pulmonary disease (COPD) is an important breathing disorder resulting from exposure to noxious particles or gases, manifested by persistent respiratory symptoms and irreversible airflow limitation [3]. COPD

occurs from pathological changes and structural remodelling of lung tissues, which progressively lead to airway obstruction [4]. COPD is one of the pathologies that lead to diaphragmatic dysfunctions, which impair its ability to perform essential respiratory functions. Clinically, diaphragmatic dysfunction primarily manifests with respiratory symptoms, including dyspnea (on exertion or, in severe cases, at rest), orthopnea, sleep-disordered breathing, and hypersomnia. Due to the nonspecific nature of these symptoms, the true incidence of diaphragmatic dysfunction is difficult to estimate; however, it is likely an underdiagnosed condition. Nonetheless, diaphragmatic dysfunction has significant implications for patients 'quality of life (QOL) and, in severe cases, for survival [1].

According to the World Health Organisation (WHO), COPD is recognised as the third leading cause of death worldwide [5]. COPD is the fifth leading cause of global disease burden and a major cause of healthcare costs worldwide. Beyond its detrimental effects on the airways, alveoli, and pulmonary vasculature, COPD exerts systemic effects, including impairment of the circulatory, nervous, and musculoskeletal systems, leading to considerable extrapulmonary consequences [6]. As noted in the Global Strategy for Prevention, Diagnosis and Management of COPD (GOLD), COPD is a preventable and treatable disease and frequently arises from lifestyle factors such as cigarette smoking, along with exposure to air pollution and occupational or domestic environmental hazards [7].

GOLD 2026 further emphasises the importance of non-pharmacological interventions, positioning them as essential pillars of disease modification rather than adjunctive options. The 2026 report clearly identifies smoking cessation, vaccination, structured patient education, self-management support, and regular physical activity as core interventions that should be made available to nearly all individuals with COPD [7]. In this review, we describe diaphragmatic dysfunction in patients with COPD and in those receiving mechanical ventilation (MV), along with the diagnostic approaches used to assess this condition. We also discuss pulmonary rehabilitation, as well as the challenges and recommendations for improving the care of COPD patients.

Materials and Methods

Materials

The materials used in this narrative review consisted of scientific literature retrieved from PubMed/MEDLINE, Scopus, Web of Science, Cochrane Library, and Google Scholar. Peer-reviewed articles, systematic reviews, meta-analyses, clinical practice guidelines, and official reports, including the GOLD 2026 report and American Thoracic Society (ATS)/European Respiratory Society (ERS) statements, published in English between January 2000 and March 2026, were considered. The review focused on literature related to diaphragmatic dysfunction in patients with COPD and/or those receiving MV.

Methods

A systematic literature search was conducted across the following electronic databases: PubMed/MEDLINE, Scopus, Web of Science, Cochrane Library, and Google Scholar. The search was limited to articles published in English from January 2000 to March 2026, with an emphasis on more recent publications where available. The following search terms and their combinations were used, employing Boolean operators (AND, OR): "chronic obstructive pulmonary disease" OR "COPD"; "diaphragm dysfunction" OR "diaphragmatic dysfunction" OR "diaphragm weakness"; "ventilator-induced diaphragmatic dysfunction" OR "VIDD"; "mechanical ventilation" OR "MV"; "inspiratory muscle training" OR "IMT"; "pulmonary rehabilitation" OR "PR"; "pursed-lip breathing" OR "diaphragmatic breathing"; "phrenic nerve stimulation" OR "PNS"; "transdiaphragmatic pressure" OR "Pdi"; "diaphragm ultrasound" OR "diaphragm imaging"; and "GOLD 2026".

Inclusion and exclusion criteria

Studies were included if they involved adult patients (aged ≥18 years) diagnosed with COPD at any stage according to GOLD criteria and/or patients receiving invasive or non-invasive mechanical ventilation in intensive care or other clinical settings. Eligible studies investigated diaphragmatic dysfunction, including its pathophysiology, risk factors, and clinical consequences, evaluated diagnostic techniques for diaphragmatic function such as ultrasound, transdiaphragmatic pressure measurement, and imaging modalities, or assessed therapeutic interventions, including pulmonary rehabilitation, inspiratory muscle training, pursed-lip breathing, diaphragmatic breathing, manual diaphragm release, muscle energy techniques, and phrenic nerve stimulation. Studies reporting management strategies from GOLD reports were also considered. To be eligible, studies were required to report at least one relevant outcome, including diaphragmatic function parameters (e.g., strength, endurance, thickness, excursion, and transdiaphragmatic pressure), respiratory muscle strength (e.g., maximal inspiratory pressure and sniff nasal inspiratory pressure), exercise capacity, dyspnea severity, QOL, lung function, weaning outcomes from MV, hospitalisation rates, or mortality. Eligible study designs included randomised controlled trials, non-randomised controlled trials, cohort studies, case-control studies, cross-sectional studies, diagnostic accuracy studies, systematic reviews, meta-analyses, and clinical practice guidelines.

Only peer-reviewed original articles, reviews, and official guidelines, such as GOLD reports and ATS/ERS statements, published in English between January 2000 and March 2026 were included.

Studies were excluded if they involved patients younger than 18 years of age or focused on diaphragmatic dysfunction resulting from conditions other than COPD or MV, such as traumatic phrenic nerve injury, congenital diaphragmatic abnormalities, diaphragmatic hernia, or neuromuscular diseases, unless COPD was the primary focus. Studies focusing exclusively on pharmacological treatments without assessment of diaphragmatic function or respiratory mechanics, as well as studies investigating surgical interventions for COPD without diaphragmatic assessment, were also excluded. In addition, studies that did not report diaphragmatic or respiratory muscle-related outcomes, case reports, or case series involving fewer than five patients, editorials, commentaries, letters to the editor, conference abstracts, and opinion papers without original data were excluded. Non-English publications, duplicate publications, studies with unavailable full texts, dissertations, and other materials were also excluded from the review.

Selection process

The study selection process was conducted following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines to enhance reporting clarity, although the present study is a narrative review. Titles and abstracts of all identified records were independently screened by two reviewers. Full texts of potentially relevant articles were subsequently retrieved and assessed against the eligibility criteria. Disagreements between reviewers at either stage were resolved through discussion.

The initial database search yielded 1,847 records. After the removal of duplicates (n = 423), 1,424 records underwent title and abstract screening. Of these, 1,201 records were excluded because they were irrelevant to the research question (e.g., non-diaphragmatic topics, animal studies without clinical translation, or editorials without original data). The remaining 223 full-text articles were assessed for eligibility. Following full-text review, 168 articles were excluded for the following reasons: unavailable full text (n = 31), non-English language without available translation (n = 18), lack of original data (n = 45), or insufficient relevance to diaphragmatic dysfunction in COPD or MV (n = 74). Ultimately, 55 studies were included in this narrative review. A PRISMA-style flow diagram summarising the study selection process is presented in Figure 1.

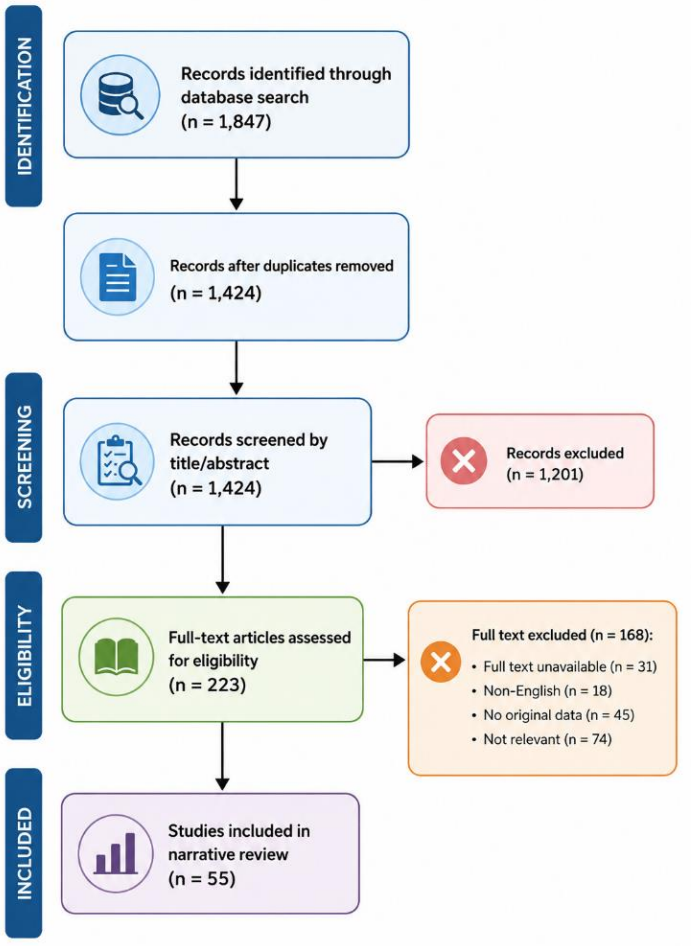

Figure 1. PRISMA-style flow diagram summarizing the study selection process.

Data analysis

Data from the included studies were extracted and synthesized narratively. Information related to study characteristics, patient populations, diagnostic approaches, pathophysiological mechanisms, therapeutic interventions, and clinical outcomes was systematically reviewed and summarized. The findings were subsequently grouped into major thematic categories, including the pathophysiology of diaphragmatic dysfunction, diagnostic modalities, clinical consequences, and therapeutic interventions in patients with COPD and those receiving MV.

A descriptive comparison of findings across studies was performed to identify similarities, differences, and emerging trends in the current evidence. Particular attention was given to outcomes related to diaphragmatic function, respiratory muscle strength, exercise capacity, dyspnea severity, QOL, weaning outcomes, hospitalization, and mortality. Recommendations from the GOLD 2026 report were integrated to contextualize the findings within current clinical practice. Owing to the heterogeneity of study designs, populations, interventions, and outcome measures, no quantitative meta-analysis or formal risk-of-bias assessment was conducted.

Results and Discussion

Diaphragmatic Dysfunction in COPD Patients

Diaphragmatic dysfunction is noticeable crossways entirely stages of COPD progression [8], and maximum inspiratory pressure has been identified as an independent predictor of survival in those with severe COPD. Nevertheless, dyspnea may manifest during physical exertion or when lying supine. Diaphragmatic dysfunction directly contributes to the development of dyspnea and respiratory failure [9] and is further related to a higher risk of hospitalization and increased mortality resulting from acute exacerbations of COPD.

Diaphragmatic strength is largely determined by the cross-sectional area of muscle fibres, their type of composition, and initial fibre length [10]. In patients with COPD, the cross-sectional area of diaphragmatic muscle fibres is reduced, with a shift from fast-twitch toward slow-twitch fibres. Concurrently, pulmonary hyperinflation shortens the resting length of the diaphragm, together contributing to diminished diaphragmatic strength. Although this fibre-type transition enhances adenosine triphosphate (ATP) production capacity, ATP utilisation efficiency declines, resulting in reduced specific force and impaired pressure-generating ability. In COPD, elevated airway resistance and airflow limitation increase the mechanical load imposed on the diaphragm and the overall work of breathing. More recently recognised contributors to diaphragmatic weakness include structural remodelling, oxidative stress, and a decrease in myosin filament content stemming from reduced protein synthesis and increased muscle cell apoptosis [11].

Transdiaphragmatic pressure (Pdi) serves as a more direct measure of diaphragmatic strength. In COPD patients, Pdi is reduced by approximately 40%, and the force generated by the diaphragm is about 35% lower than that observed in healthy individuals [12]. Moreover, the maximum Pdi values in those with severe COPD reach only 60% of the levels seen in healthy controls [13]. Increased lung volumes and alterations in chest wall geometry contribute to diaphragmatic shortening. On average, the diaphragm in COPD patients is 28% shorter than in healthy subjects. This shortened state places the diaphragm at a suboptimal length, impairing the effective conversion of diaphragmatic tension into transdiaphragmatic pressure and thereby reducing pressure-generating capacity. Dynamic hyperinflation further compromises diaphragmatic function by shortening the diaphragm to a mechanically disadvantageous length, diminishing its curvature, and reducing the area of contact between the diaphragm and the chest wall. Together, these factors increase the mechanical burden on the diaphragm while decreasing its power output [14]. In addition, pulmonary hyperinflation shortens the operating length of the diaphragm and disrupts the mechanical coupling among its segments, ultimately weakening diaphragmatic strength [13].

The diaphragm also influences cardiac and autonomic function. In patients with COPD, diaphragmatic muscle fibres exhibit lower protein content and are shortened due to an inspiratory posture [15]. Compared with healthy individuals, the diaphragm occupies a more caudal position, which reduces its radius in the zone of apposition and places it at a biomechanical disadvantage [16]. Magnetic resonance imaging (MRI) has revealed asynchronous movement of the two hemi-diaphragmatic domes in COPD, a phenomenon that becomes more pronounced in patients with greater disease severity (GOLD stage IV) [17]. Neuropathic damage to the phrenic nerve, particularly on the left side, has been observed, with more extensive lesions correlating with greater degrees of hyperinflation [15]. Overall, diaphragmatic mobility is globally reduced, and non-physiological adaptations are detectable in all COPD patients, irrespective of disease stage [18].

Diaphragmatic Dysfunction in Mechanically Ventilated (MV) Patients

Diaphragmatic dysfunction may lead to alveolar hypoventilation and, in severe cases, progress to respiratory failure necessitating MV [19]. Among patients admitted to the intensive care unit, an estimated 40–80% develop at least one form of neuromuscular dysfunction [20]. Such dysfunction encompasses a broad spectrum of conditions, including critical illness polyneuropathy, critical illness myopathy, combined critical illness polyneuromyopathy, and ventilator-

induced diaphragm dysfunction (VIDD) [21]. VIDD is defined by a reduced capacity of the diaphragm to generate force [22] and may occur either alongside ICU-acquired weakness (ICUAW) or independently of it [23]. This condition frequently coexists with diaphragm atrophy resulting from MV in the intensive care unit (ICU), a combination referred to as critical illness-associated diaphragm weakness [23].

VIDD refers to a decline in the diaphragm's force-generating capacity that is specifically attributable to MV, accompanied by diaphragmatic muscle unloading and inactivity [22]. In critically ill patients, diaphragm weakness exhibits a rapid onset. The structural damage incurred during MV follows a time-dependent pattern: the duration of MV correlates directly with the extent of muscle fibre injury and proteolysis [24]. A previous study showed that more than 50% loss of diaphragmatic muscle fibre cross-sectional area occurs after only 18 to 69 hours of MV [25]. Diaphragmatic unloading leads to contractile dysfunction and a marked reduction in force production [26]. Over-assistance myotrauma comprising disuse atrophy and VIDD, results from excessive respiratory support, which diminishes respiratory drive and effort, thereby promoting disuse atrophy and functional impairment. Regarding the time course of VIDD development, half of intubated ICU patients do not use their diaphragm following intubation, and some require more than five days before resuming diaphragmatic activity [27].

There is currently no validated, universally accepted diagnostic standard for VIDD in mechanically ventilated ICU patients. Although measurement of Pdi following phrenic nerve stimulation remains the gold standard for diaphragmatic strength assessment, this technique is invasive (requiring oesophageal and gastric balloon catheters), time-consuming, and impractical for routine bedside use in critically ill patients [28]. Consequently, ultrasound measurement of diaphragm thickness and thickening fraction has emerged as a popular surrogate. However, the correlation between ultrasound-derived parameters and directly measured diaphragmatic force generation in ventilated ICU patients has not been rigorously validated. Some studies report moderate correlations [29], while others suggest that thickening fraction may reflect effort rather than intrinsic strength [30]. Furthermore, inter-operator reliability of diaphragm ultrasound in critically ill patients who may have variable oedema, positioning, and acoustic windows is less well established than in healthy volunteers. Clinicians should therefore interpret ultrasound findings with caution and recognise that "normal" thickness does not definitively exclude weakness, nor does "reduced" thickness invariably confirm clinically significant VIDD.

A common assumption in the studies and in clinical practice is that reduced diaphragm thickness (atrophy) equates to reduced force-generating capacity (weakness). However, the evidence for this relationship is more complex [31], notably demonstrating rapid loss of muscle fibre cross-sectional area in brain-dead organ donors following 18–69 hours of MV, but these patients had no respiratory drive whatsoever, an extreme scenario not representative of most ICU patients. Subsequent studies in critically ill patients with preserved respiratory drive have shown only modest correlations between thickness and strength [29]. Importantly, some patients develop significant diaphragmatic weakness without measurable atrophy, possibly due to contractile dysfunction at the myofilament level or impaired neural activation. Conversely, others maintain force generation despite measurable thinning. This dissociation suggests that VIDD encompasses at least two distinct phenotypes: an atrophic form (predominantly disuse-mediated) and a non-atrophic myopathic form (potentially driven by inflammation, oxidative stress, or sepsis). Current clinical assessment tools do not reliably distinguish these phenotypes, yet they may have different prognoses and treatment responses.

The prevailing narrative, reinforced by influential animal studies and the Levine organ donor data, is that MV rapidly and uniformly induce diaphragmatic dysfunction. However, human data from non-brain-dead critically ill patients suggest a more nuanced picture. Several studies have documented substantial inter-individual variability in the rate and magnitude of diaphragm thinning during MV [29]. Some patients on prolonged ventilation (≥7 days) exhibit minimal or no atrophy, while others develop marked thinning within 48–72 hours. Factors that may modulate susceptibility include baseline diaphragm function (pre-existing COPD-related weakness may increase vulnerability), severity of systemic inflammation, nutritional status, use of neuromuscular blocking agents, and preserved spontaneous breathing efforts. This variability raises an important clinical question: Which patients are at the highest risk for VIDD, and can we predict who will be protected? Current evidence does not provide clear answers, representing a significant knowledge gap.

In subsequent years, surgically implantable devices designed for controlled electrical stimulation of the phrenic nerve emerged as a promising approach to facilitate weaning from MV in spinal cord injury patients with chronic respiratory insufficiency [32]. Regarding the potential therapeutic application of phrenic nerve stimulation, current research efforts are directed toward identifying both the preventive and therapeutic effects of various stimulation techniques in critically ill individuals. A preventive strategy could involve early use of diaphragmatic stimulation to avert muscle atrophy during the initial phase of critical illness and MV, analogous to early mobilisation of skeletal muscle. Preliminary data indicate that temporary phrenic nerve stimulation (PNS) can sustain continuous diaphragmatic activity during MV [33]. The underlying rationale is that preventing diaphragmatic disuse helps preserve muscle mass and function, thereby reducing atrophy and dysfunction. Limiting diaphragmatic unloading may lead to earlier liberation from MV and consequently reduce associated adverse events. An additional benefit of maintaining diaphragmatic

contraction is the preservation of ventilated lung volume, which is typically lost following deep sedation. This effect could enhance gas exchange and lower the risk of ventilator-induced lung injury (VILI). From a therapeutic perspective, diaphragmatic muscle training may be employed to shorten the weaning duration in patients with established diaphragmatic weakness.

Diaphragmatic activation via PNS holds potential for use in electro phrenic respiration, which is a form of negative pressure ventilation that enables more physiological breathing patterns and may thereby alleviate the adverse effects associated with positive pressure ventilation. Specifically, PNS-induced ventilation (electrophenic respiration) could help prevent VILI through two mechanisms: (1) by reducing the overall duration of MV, or (2) by improving the benefit-to-risk ratio of MV through avoidance of its maximal utilisation. As a therapeutic strategy in critically ill patients, PNS could be titrated according to the patient's own diaphragmatic electrical activity, synchronised with spontaneous breathing efforts, and delivered alongside non-invasive ventilation, high-flow nasal oxygen, or pressure support ventilation [33].

Early rehabilitation is both feasible and beneficial for mitigating diaphragm dysfunction resulting from prolonged MV, as well as for facilitating weaning from the ventilator and promoting extubation in patients receiving MV. Diaphragm ultrasound (US) is recommended for mechanically ventilated patients in the ICU [34]. Consequently, exercise may improve diaphragmatic dysfunction by reducing adverse factors that negatively affect diaphragmatic structure and function overall. Beyond its direct positive effects on the diaphragm, exercise interventions can also train the accessory muscles of the abdomen, back, and limbs. These muscles provide enhanced support during respiration, thereby reducing diaphragmatic fatigue and improving diaphragmatic function.

Although COPD-related diaphragmatic dysfunction and VIDD differ in terms of their underlying mechanisms and temporal progression, both conditions ultimately lead to impaired diaphragmatic function and respiratory muscle weakness. Moreover, patients with advanced COPD frequently require MV, creating a clinical continuum in which COPD-related dysfunction may coexist with or predispose patients to VIDD. The key distinctions and rationale for integrating these two conditions in the present review are summarized in Table 1.

Table 1. Comparison of COPD-related diaphragmatic dysfunction and ventilator-induced diaphragmatic dysfunction (VIDD) and the rationale for their integration

Feature	COPD-related dysfunction	VIDD	Rationale for combining
Primary Cause	Chronic overload (excessive work of breathing)	Acute unloading (diaphragmatic inactivity)	Clinical continuum: COPD progression may lead to respiratory failure requiring mechanical ventilation, thereby predisposing patients to VIDD.
Time Course	Months to years	Hours to days	Temporal sequence: VIDD may develop following the initiation of mechanical ventilation in patients with advanced COPD.
Mechanism	Hyperinflation, muscle fiber-type shift (Type I to Type II), and structural remodeling	Proteolysis, muscle atrophy, and mitochondrial oxidative stress	Convergent pathways: both conditions ultimately result in diaphragmatic weakness and impaired respiratory function.
Therapeutic implication	Reduction of respiratory load and pulmonary rehabilitation	Restoration of diaphragmatic activity and prevention of disuse	Shared therapeutic goal: maintaining or restoring diaphragmatic activity may improve outcomes in both conditions.

Diagnosis of Diaphragmatic Dysfunction

Several static and dynamic imaging modalities are available for evaluating patients with suspected diaphragmatic dysfunction. Static techniques, including chest radiography, ultrasound, CT, and static MRI, assess diaphragm position, shape, and dimensions. Dynamic techniques, such as fluoroscopy, evaluate diaphragmatic motion in one or more directions [35]. In addition, magnetic stimulation of the phrenic nerve provides a functional assessment.

Chest radiography (CT)

Chest radiography is used to determine the position of each hemidiaphragm. Elevation of one hemidiaphragm suggests unilateral phrenic nerve palsy; however, this finding is nonspecific and may also occur in atelectasis, pneumonia, lobectomy, or pulmonary fibrosis [36]. The sensitivity of plain chest radiographs for correctly identifying

unilateral diaphragm paralysis is as low as 66.6%, with a specificity of only 44%. Additional limitations include moderate interobserver agreement in detecting unilateral hemidiaphragm elevation [36]and difficulty identifying diaphragm elevation in bilateral paralysis unless prior radiographs are available for comparison [37]. Chest radiography provides limited information on diaphragm morphology [37] and is not a practical option for repeated assessments. Other images are used for assessment, but they carry hazards, as fluoroscopy is untrustworthy and involves ionising radiation [11]. Although CT and MRI offer superior image quality, they are costly, and CT also exposes patients to radiation [38], limiting their use in serial examinations.

Ultrasonography

Ultrasound effectively evaluates diaphragmatic dysfunction in COPD patients [39]. Diaphragm ultrasound is an emerging, real-time, noninvasive tool increasingly adopted in clinical practice. It can assess muscle structure, including diaphragm thickness, excursion, activity (thickness fraction), and function (maximal thickening fraction) [40]. The reproducibility of this technique has been widely validated in healthy individuals [41] and outpatients and critically ill patients [42]. To measure diaphragm thickness, the ultrasound probe is placed longitudinally parallel to the body's long axis, typically between the eighth and tenth intercostal spaces at the anterior axillary line or midway between the anterior and midaxillary lines [41]. Sonographic methods include measuring diaphragmatic thickening in the zone of apposition during contraction, observing downward movement of the left portal vein during inspiration, and directly visualising the diaphragm using the liver or spleen as acoustic windows [27]. Major limitations of these sonographic approaches include dependence on patient cooperation and limited comprehensive validation.

Computed tomography (CT)

CT imaging obtained at different lung volumes has been used to assess diaphragm position [43] and dimensions, including thickness, surface area, and volume [44]. CT has also been proposed for measuring crural diaphragm thickness in ventilated patients and those with suspected diaphragm paralysis [45]. However, no consensus exists regarding which muscle region to measure or at which lung volume [45], leaving the role of CT for crural diaphragm measurement uncertain. Spiral CT has been applied to calculate diaphragm volume [44], but its accuracy for this purpose has not been validated.

Static magnetic resonance imaging (MRI)

Static MRI performed at various lung volumes allows assessment of diaphragm shape, position, thickness, and surface area [46]. It provides detailed information on the entire diaphragm's surface area and positioning within the thorax, enabling correlation between changes in diaphragm position and lung volume [47] and helping to elucidate the diaphragm's role in pulmonary disease conditions [47]. Moreover, MRI images can be sufficiently detailed to detect clinically meaningful changes in muscle thickness, potentially serving as a useful tool for assessing diaphragm atrophy [47].

Fluoroscopy

During a sniff manoeuvre, fluoroscopy provides two-dimensional information on the movement of the diaphragm's central tendon. However, fluoroscopy can yield misleading results, particularly in patients with hemidiaphragm paresis or bilateral paralysis [35] and involves radiation exposure.

Magnetic stimulation of the phrenic nerve

The current gold standard for assessing diaphragmatic dysfunction is measurement of transdiaphragmatic pressure following magnetic stimulation of the phrenic nerve [48]. Magnetic stimulation simplifies localisation of the phrenic nerve, though it may inadvertently stimulate adjacent structures such as the brachial plexus. Although this test is invasive and time-consuming, it has the advantage of being independent of patient cooperation.

Although magnetic stimulation of the phrenic nerve with Pdi measurement remains the gold standard for assessing diaphragmatic dysfunction, its invasive nature and limited availability restrict its routine clinical use. Several alternative diagnostic modalities are currently available, each with specific strengths and limitations. The main characteristics, indications, and limitations of these modalities are summarized in Table 2.

Table 2. Comparison of diagnostic modalities for the assessment of diaphragmatic dysfunction: advantages, disadvantages, clinical indications, and limitations

Modality	Advantages	Disadvantages	Clinical indications	Limitations
Chest radiography	Widely available; low cost; quick to perform	Low sensitivity (66.6%) and specificity (44%); poor interobserver agreement; exposure to ionizing radiation; inability to assess bilateral paralysis without previous films	Initial screening for suspected unilateral diaphragmatic elevation	Nonspecific findings (e.g., atelectasis or pneumonia may mimic diaphragmatic dysfunction); provides no functional information; unsuitable for serial assessments
Ultrasonography (US)	Noninvasive; real-time assessment; radiation-free; portable and suitable for bedside use in the ICU; assesses diaphragm thickness, excursion, and thickening fraction; validated in healthy, outpatient, and critically ill populations	Operator dependent; requires patient cooperation; limited acoustic windows in obesity and severe COPD; incomplete validation for some parameters	Monitoring ventilator-induced diaphragmatic dysfunction (VIDD) in ICU patients; assessment in COPD outpatients; evaluation during weaning from mechanical ventilation; screening for diaphragm atrophy	Cannot directly measure transdiaphragmatic pressure; poor visualization in certain body habitus; requires operator training
Computed tomography (CT)	Excellent anatomical detail; allows assessment of diaphragm thickness, surface area, and volume; can evaluate the crural diaphragm in ventilated patients	Exposure to ionizing radiation; expensive; not portable; requires transportation of critically ill patients; lacks functional assessment	Anatomical evaluation when ultrasonography is inconclusive; pre-surgical planning; research applications	No consensus regarding measurement location or lung volume; diaphragm volume measurements remain insufficiently validated; unable to assess dynamic function
Static magnetic resonance imaging (MRI)	Superior soft-tissue contrast; no ionizing radiation; evaluates diaphragm shape, position, thickness, and surface area; capable of detecting clinically significant atrophy	Expensive; not portable; requires patient cooperation (breath-holding); contraindicated in some patients (e.g., pacemakers, claustrophobia); limited availability	Detailed anatomical mapping; research evaluating diaphragm–lung volume relationships; repeated imaging when radiation exposure is undesirable	Does not provide dynamic functional assessment unless dynamic MRI is performed; time-consuming; cannot be performed at the bedside
Fluoroscopy	Dynamic assessment of diaphragmatic motion; real-time visualization during the sniff maneuver	Exposure to ionizing radiation; two-dimensional imaging only; potentially misleading results in partial or bilateral paralysis; operator dependent	Sniff test for suspected unilateral phrenic nerve palsy (historical use)	Limited diagnostic accuracy for diaphragmatic paresis; increasingly replaced by ultrasonography; cannot quantify force generation
Magnetic stimulation of the phrenic nerve with transdiaphragmatic pressure (Pdi) measurement	Current gold standard; independent of patient cooperation; objectively quantifies diaphragmatic force-generating capacity	Invasive (requires esophageal and gastric balloon catheters); time-consuming; requires specialized equipment and expertise; may cause patient discomfort	Definitive diagnosis when imaging findings are inconclusive; research settings requiring precise force measurements; medicolegal evaluation	Unsuitable for serial bedside monitoring; requires specialized training; may inadvertently stimulate adjacent structures such as the brachial plexus

Pulmonary Rehabilitation (PR) for Diaphragmatic Dysfunction in COPD Patients

PR is defined as a comprehensive intervention based on a thorough patient assessment followed by patient-tailored therapies that include, but are not limited to, exercise training, education, and behaviour change, designed to improve the physical and psychological condition of people with chronic respiratory disease and to promote the long-term adherence to health-enhancing behaviours [49]. It constitutes a fundamental component of integrated care for individuals with chronic respiratory diseases.

PR has been shown to confer physiological, symptom-relieving, psychosocial, and health-economic benefits across multiple outcome domains for patients with chronic respiratory diseases [49]. Accordingly, it should be regarded as a standard of care, alongside established treatments such as pharmacotherapy, supplemental oxygen, or noninvasive ventilation. PR emphasises the stabilisation and/or reversal of extrapulmonary manifestations and comorbidities associated with chronic respiratory disease, as well as the critical role of behaviour change [50]. It serves as an essential element of an integrated, continuous healthcare model that spans the entire illness trajectory and coordinates across various healthcare providers and settings [49].

Exercise training serves as the foundational element of PR. Concurrent behavioural strategies such as enhancing self-efficacy and teaching collaborative self-management skills are equally essential for optimising patient outcomes [51]. Upon completing a PR program, patients benefit from continued engagement in home-based, community-based, or program-based maintenance exercise regimens that support the persistence of positive exercise behaviours [51]. **Table 3** summarises the evidence quality rating.

Table 3. Summary of evidence quality, clinical effects, and recommendations for non-pharmacological interventions in diaphragmatic dysfunction

Intervention	Evidence quality	Effect size	Clinical recommendation strength	Key limitations
Inspiratory muscle training (IMT)	Moderate to high (supported by multiple randomized controlled trials and meta-analyses)	Moderate (PImax increase of approximately 20–30 cm H_2O)	Grade B (recommended for selected patients)	Predominantly short-term studies; 20–30% of patients may not respond; optimal training protocol remains unclear
Pursed-lip breathing (PLB)	Low to moderate (based mainly on small randomized trials without sham controls)	Small to moderate (6-minute walk distance increase of approximately 30–50 m)	Grade C (reasonable as an adjunctive intervention)	Lack of long-term follow-up; unblinded study designs; inconsistent effect sizes across studies
Diaphragmatic breathing (DB)	Very low (absence of standalone randomized controlled trials)	Unknown (effects cannot be isolated from PLB interventions)	Grade D (not recommended as a standalone intervention)	No independent evidence available; potential adverse effects in patients with severe hyperinflation
Manual diaphragmatic release	Very low (small, unblinded studies without sham controls)	Uncertain (improvements generally below the minimal clinically important difference for the 6-minute walk test)	Grade I (insufficient evidence)	Lack of standardized protocols; potential publication bias; absence of long-term outcome data

Challenges and Recommendations

Underdiagnosis and case finding

Despite years of advancement in understanding COPD mechanisms and developing treatments, COPD continues to be detected late in its course and remains insufficiently treated. Its management often occurs in a disorganised fashion that typically overlooks the disease's biological diversity and frequent coexistence with other conditions [63]. Against this milieu, successive restatements of the GOLD strategy literature have become the worldwide reference for COPD diagnosis and treatment. The most recent version, the GOLD 2026 report, stands out for its refined clinical pathways and the introduction of several novel concepts relevant to everyday practice [64]. This new framework moves beyond

simply alleviating symptoms and assessing conventional "future risk." It shapes the revised management cycle, the restructured ABE classification, and the approach to therapeutic decisions covering treatment escalation, de-escalation, and the use of advanced agents such as biologics.

Underdiagnosis of COPD remains common, with many patients unaware of their underlying airflow obstruction. The GOLD 2026 report distinguishes between targeted case finding, which it supports, and population screening, which it does not recommend. It consolidates evidence endorsing the use of structured questionnaires, handheld devices, and opportunistic testing in high-risk clinical settings. These recommendations align with emerging epidemiological data showing that early physiological abnormalities, particularly emphysema and small-airway disease detected by CT, precede spirometric decline by several years, thereby offering a theoretical window for "activity suppression" interventions [64].

GOLD 2026 adopts a "multimorbidity framework" that moves away from treating COPD in isolation. Within this framework, diaphragmatic dysfunction should be recognised as a common and prognostically important comorbidity or, more accurately, a systemic manifestation of COPD. This reframing encourages clinicians to screen for diaphragm weakness in all patients with moderate-to-severe COPD (GOLD stages II–IV) and to consider diaphragm-directed rehabilitation as part of comprehensive multimorbidity management.

Non-pharmacological interventions

GOLD 2026 places greater emphasis on non-pharmacological interventions, positioning PR, vaccination, smoking cessation, oxygen/non-invasive ventilation (NIV) therapy, and structured post-exacerbation care as core disease-modifying strategies rather than optional adjuncts [24]. Evidence from controlled trials indicates that these interventions reduce hospital readmissions and improve survival, with effect sizes comparable to pharmacological escalation in selected patient groups [28]. This elevation of rehabilitation in the treatment order provides a policy rationale for increased compensation and resource allocation for diaphragm-specific interventions, including IMT and ultrasound-guided breathing retraining. The GOLD report explicitly supports home-based and tele-rehabilitation as effective alternatives to centre-based programs, which is particularly relevant for IMT, which is well-suited to home-based delivery with remote monitoring of device usage and PImax measurements

Barriers to PR utilisation

Despite its well-documented benefits, PR remains significantly underutilised and frequently inaccessible to patients worldwide. It is often absent from integrated care models for chronic respiratory disorders. Contributing factors include insufficient funding, limited resources for PR programs, inadequate health system reimbursement, lack of awareness among healthcare professionals, payers, patients, and caregivers regarding the processes and benefits of PR, suboptimal referral of suitable patients, and limited training opportunities for PR professionals. These barriers widen the gap between the scientific evidence supporting PR and its actual delivery [33]. Persistent limitations, such as suboptimal referral, uptake, and completion rates, stem from limited provider awareness, logistical challenges, and inadequate funding, all contributing to inequities in access [65]. In addition to underutilization of diaphragm ultrasound, lack of clinician training, an absence of standardised IMT protocols, and patient-specific contraindications such as diaphragmatic breathing in severe hyperinflation

Telerehabilitation and ongoing challenges

Tele-rehabilitation has emerged as a strategy to increase access to pulmonary rehabilitation while reducing barriers related to travel, cost, and time. For diaphragm-specific rehabilitation, tele-rehabilitation offers several unique applications and challenges. Although telerehabilitation alleviates some of these challenges, concerns remain regarding protocol standardisation, patient selection, and long-term benefits. A 2026 randomised controlled trial of 123 patients with chronic respiratory diseases provides the strongest evidence to date for telerehabilitation [66]. Patients assigned to a 6-month Internet of Things (IoT)-based remote respiratory rehabilitation program demonstrated significantly greater improvements in diaphragm thickness compared to conventional rehabilitation (between-group difference: 6.79 mm, 95% CI 4.68–8.90, $p < 0.001$). The study group also showed significantly greater improvements in the six-minute walk test(6MWT) (between-group difference: 63.74 m, exceeding the minimal clinically important difference of 30 m), forced expiratory volume at 1st second(FEV1) (0.68 L), and forced vital capacity(FVC) (0.54 L). Rehospitalisation rates were substantially lower in the study group (18.33% vs. 36.51%). This study used IoT-enabled devices for remote monitoring and real-time data transmission, representing a higher level of technological integration than simple telephone monitoring [66].

Moreover, the current evidence base, derived largely from stable COPD populations, remains limited with respect to post-exacerbation care, comorbid conditions, and outcomes beyond program completion. In addition, challenges to remote IMT delivery, remote diaphragm ultrasound, and digital coaching for breathing techniques [64]. The GOLD 2026 update highlights the importance of structured discharge bundles, including PR referral, early follow-up within

one month, and reassessment at 12–16 weeks, to reduce readmissions and optimise maintenance therapy. The 2026 update expands eligibility to include all individuals with exertional dyspnea, deconditioning, or recent exacerbations, irrespective of spirometric severity [64].

Home-based and telerehabilitation

GOLD 2026 also acknowledges a growing body of evidence supporting home-based and telerehabilitation programs. When delivered with comparable intensity and supervision, these programs achieve outcomes like centre-based rehabilitation in terms of functional capacity, QOL, and dyspnea, while demonstrating higher completion rates. However, recognising heterogeneity in program design, patient selection, and outcome durability, the report continues to regard supervised centre-based rehabilitation as the preferred standard, positioning remote modalities as alternatives for patients unable to access in-person care [64].

ATS/ERS policy statement on PR

The *ATS/ERS* statement on PR [49] adopts a different focus, offering policy recommendations aimed primarily at expanding the provision of PR to suitable individuals globally. This document represents a consensus among international PR experts, primary care specialists, and global patient advocates. It provides recommendations addressing key processes central to enhancing the implementation, use, and delivery of PR, including increasing awareness and knowledge among healthcare professionals, payers, and patients; improving patient access to PR; and ensuring the quality of PR programs. The statement also proposes actionable items to foster the implementation of these policy recommendations, notably including strategies to increase PR implementation, utilisation, and delivery.

Artificial intelligence (AI) applications

The report further describes AI applications for remote monitoring and self-management, including smart inhalers, wearable sensors, telerehabilitation platforms, and AI-supported patient interfaces such as chatbots. Additional potential benefits include improved integration of multimorbidity data within electronic health records, support for guideline-aligned care, and reduction of risks associated with polypharmacy [64].

AI and machine learning (ML) techniques have begun to be applied to diaphragm assessment in COPD patients. It is important to emphasise that these applications remain experimental, and none are currently approved for routine clinical use. However, several proof-of-concept studies have reported promising preliminary results that warrant further investigation. In a prospective study involving 316 patients with COPD conducted in 2025, an interpretable ML model integrating multimodal ultrasound features, including lung, diaphragm, and quadriceps assessments, was developed to facilitate bedside diagnosis of acute exacerbations [67]. The support vector machine (SVM) model achieved an area under the receiver operating characteristic curve (AUC) of 0.9302 in the test set, with diaphragmatic dysfunction identified by Shapley additive explanations (SHAP) analysis as one of the most influential predictors. The final model incorporated six routinely obtainable variables, five of which were ultrasound-derived. The authors acknowledge that multicenter validation is required before clinical implementation.

An automated tool for quantifying diaphragmatic configuration from chest CT images was evaluated in 8,431 participants from the COPD Gene study [68]. The tool calculated a diaphragm index, defined as the ratio of diaphragm surface area to projected surface area, as a measure of diaphragmatic flattening. The diaphragm index decreased significantly with increasing GOLD stages, ranging from 1.83 ± 0.16 in never-smokers to 1.54 ± 0.11 in patients with GOLD stage 4 disease ($p < 0.001$). Furthermore, the diaphragm index demonstrated moderate correlations with FEV_1 % predicted ($r = 0.44$) and emphysema score ($r = -0.36$). Technical limitations included incomplete diaphragm segmentation in 9.2% of cases. The authors suggested that this tool may be useful for evaluating treatment efficacy following lung volume reduction procedures; however, this potential application has not yet been validated.

Despite these promising findings, several important barriers continue to hinder the clinical implementation of AI for diaphragm assessment. All currently available studies have been conducted in single-centre settings with relatively limited sample sizes, and no AI algorithm has yet been prospectively validated in multicenter cohorts. Furthermore, no system has received regulatory approval from the Food and Drug Administration (FDA) for diagnostic use. Challenges related to the integration of AI tools with existing electronic health record systems and picture archiving and communication systems (PACS) also remain unresolved. Importantly, no study has demonstrated that AI-assisted diaphragm assessment improves patient outcomes compared with standard care. Therefore, until these challenges are addressed, AI-based diaphragm assessment should be considered a research tool rather than a routine clinical application.

This review has several notable strengths. First, to our knowledge, it is among the first reviews to incorporate recommendations from the recently published GOLD 2026 report, which introduces important concepts such as disease activity as a treatment target, an expanded emphasis on non-pharmacological interventions, formal recognition of home-based and tele-rehabilitation, and a multimorbidity framework. These developments have not been comprehensively

addressed in earlier reviews. Second, unlike previous reviews that have generally considered COPD-related diaphragmatic dysfunction and VIDD as separate entities, the present review adopts a clinical continuum perspective by highlighting how pre-existing diaphragmatic weakness in patients with COPD may increase susceptibility to VIDD during MV. Third, this review provides a comprehensive comparison of currently available diagnostic modalities, including their advantages, disadvantages, clinical indications, and limitations, thereby offering a practical resource for clinicians and researchers. In addition, this review critically addresses several unresolved issues in the field, including limitations of current diagnostic standards, the dissociation between diaphragmatic atrophy and weakness, inter-individual variability in treatment responses, optimal respiratory effort targets, and the still experimental role of phrenic nerve stimulation. Finally, in line with GOLD 2026 recommendations, this review places particular emphasis on rehabilitation and other non-pharmacological strategies while also identifying important barriers to their implementation in routine clinical practice.

Nevertheless, several limitations should be acknowledged. Although a structured literature search was employed to ensure comprehensive identification of relevant evidence, this study was conducted as a narrative review, and therefore the findings were synthesized narratively rather than quantitatively. This approach, sometimes referred to as a systematic narrative review, combines the transparency of a structured search strategy with the interpretative flexibility of narrative synthesis. However, the absence of a formal risk-of-bias assessment and quantitative data pooling may limit the ability to draw definitive conclusions. Furthermore, as with all narrative reviews, there remains the potential for author bias in study selection and interpretation of the findings.

Conclusion

Diaphragmatic dysfunction is a clinically important yet frequently underrecognized complication of COPD and MV, contributing substantially to respiratory impairment, poor functional status, and adverse clinical outcomes. Early identification using appropriate diagnostic modalities, particularly diaphragm ultrasound, together with timely implementation of pulmonary rehabilitation and other non-pharmacological interventions, may improve diaphragmatic function, exercise capacity, and QOL. The GOLD 2026 report further highlights the importance of integrated, patient-centred care approaches, including home-based and tele-rehabilitation, to expand access to evidence-based interventions. Future research should focus on optimizing diagnostic strategies, standardizing rehabilitation protocols, and improving equitable access to effective interventions across diverse patient populations.

Acknowledgment

The authors would like to express their sincere gratitude to all individuals and institutions who provided support during the preparation of this review. The authors also acknowledge the valuable contributions of colleagues and reviewers whose insights and constructive feedback helped improve the quality of this manuscript.

Declarations

Author contribution	:	M.M. conceptualized the review topic, designed the review framework, developed the search strategy, and supervised the overall study process. M.M. also conducted the literature search, screened the identified studies, interpreted the findings, and prepared the original manuscript draft. T.H.M. contributed to data curation, assisted in study screening and data extraction, critically reviewed the included literature, and contributed to the interpretation and synthesis of the findings. T.H.M. also performed critical revision and editing of the manuscript for important intellectual content. Both authors reviewed, revised, and approved the final version of the manuscript and agreed to be accountable for all aspects of the work.
Funding statement	:	This research received no external funding. No specific grant was obtained from funding agencies in the public, commercial, or not-for-profit sectors.
Conflict of interest	:	The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper. The authors further declare that there are no conflicts of interest regarding the publication of this manuscript.
Ethics Declaration	:	Ethical approval and informed consent were not required because this study is a narrative review based exclusively on previously published literature and did not involve human participants, animals, or the use of identifiable personal data.

Additional information : The authors confirm that all relevant data and information supporting the findings of this review are included within the manuscript and the cited references. The authors are solely responsible for the content and interpretation presented in this review.

Informed Consent Statement

Not applicable. This study did not involve human participants or the collection of identifiable personal information.

Data Availability Statement

No new data were generated or analyzed in this study. All information presented in this review was derived from previously published studies, which are cited appropriately throughout the manuscript.

Declaration of Generative AI and AI-Assisted Technologies in the Writing Process

During the preparation of this manuscript, the authors used generative artificial intelligence (AI) tools solely to improve the language, grammar, and readability of the manuscript. All AI-assisted outputs were carefully reviewed, revised, and validated by the authors. The authors take full responsibility for the accuracy, integrity, and originality of the content presented in this article. No AI tools were used for study design, data collection, data analysis, interpretation of findings, or the formulation of scientific conclusions.

ORCID for Authors

Marwa Mohammed[1,2], ORCID: https://orcid.org/0000-0003-1666-2167
Taha Hussein Musa[3], ORCID: https://orcid.org/0000-0003-4452-1943

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