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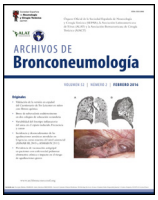

Review

Diaphragm Dysfunction in Mechanically Ventilated Patients☆

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ABSTRACT

Muscle involvement is found in most critical patients admitted to the intensive care unit (ICU). Diaphragmatic muscle alteration, initially included in this category, has been differentiated in recent years, and a specific type of muscular dysfunction has been shown to occur in patients undergoing mechanical ventilation. We found this muscle dysfunction to appear in this subgroup of patients shortly after the start of mechanical ventilation, observing it to be mainly associated with certain control modes, and also with sepsis and/or multi-organ failure. Although the specific etiology of process is unknown, the muscle presents oxidative stress and mitochondrial changes. These cause changes in protein turnover, resulting in atrophy and impaired contractility, and leading to impaired functionality. The term 'ventilator-induced diaphragm dysfunction' was first coined by Vassilakopoulos et al. in 2004, and this phenomenon, along with injury cause by over-distention of the lung and barotrauma, represents a challenge in the daily life of ventilated patients.

Diaphragmatic dysfunction affects prognosis by delaying extubation, prolonging hospital stay, and impairing the quality of life of these patients in the years following hospital discharge. Ultrasound, a non-invasive technique that is readily available in most ICUs, could be used to diagnose this condition promptly, thus preventing delays in starting rehabilitation and positively influencing prognosis in these patients.

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Disfunción diafragmática: una realidad en el paciente ventilado mecánicamente

Palabras clave:
Diafragma
Disfunción diafragmática
Ventilación mecánica
Ecografía

RESUMEN

La afectación muscular del paciente crítico está presente en la mayoría de pacientes que ingresan en el servicio de medicina intensiva (SMI). La alteración, en particular, del músculo diafragmático, inicialmente englobada en esta categoría, se ha diferenciado en los últimos años y se ha demostrado la existencia de una disfunción muscular propia de los pacientes sometidos a ventilación mecánica. En este subgrupo de pacientes encontramos una disfunción muscular que aparece de manera precoz después del inicio de la ventilación mecánica y que se relaciona principalmente con el uso de modalidades control, la presencia de sepsis y/o de fracaso multiorgánico. Aunque se desconoce la etiología concreta que desencadena el proceso, el músculo presenta procesos de estrés oxidativo y alteración mitocondrial que provocan un desequilibrio en la síntesis proteica, con el resultado de atrofia y alteración de la contractilidad y, como consecuencia, una menor funcionalidad. No fue, de hecho, hasta 2004 cuando Vassilakopoulos et al.

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describieron el término «disfunción diafragmática asociada a ventilación mecánica», que, junto a la lesión por sobredistensión pulmonar y por barotrauma, representan un reto en el día a día de los pacientes ventilados.

La disfunción diafragmática tiene influencia en el pronóstico, retardando la extubación, aumentando la estancia hospitalaria y afectando la calidad de vida de estos pacientes en los años siguientes al alta hospitalaria. La ecografía, como técnica no invasiva y accesible en la mayoría de unidades, podría ser de utilidad en el diagnóstico precoz para iniciar, de forma avanzada, la rehabilitación e influir positivamente en el pronóstico de estos enfermos.

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Introduction

Mechanical ventilation (MV) has been used throughout history as a basic tool in the treatment of patients with respiratory failure, and as a means of improving prognosis. Almost 40% of mechanically ventilated patients have difficulties during MV weaning, due to multiple factors. A delay in weaning can prolong the stay in the intensive care unit (ICU) and lead to a poorer prognosis[1–3] and a 12% increase in mortality compared to patients with no weaning problems.[4]

In the last 20 years or so, attention has focused on the study of ventilation-induced diaphragm dysfunction (VIDD)[5] as one of the complications associated with MV. This term refers to diaphragm dysfunction that occurs soon after initiating MV.[6] VIDD worsens prognosis and is associated with extubation failure, which in turn prolongs MV[7–12] and increases the risk of mortality.[13–16] At present, however, diaphragm function is not routinely monitored in many units, suggesting that this entity may be systematically underdiagnosed.[17]

Ventilation-induced Diaphragm Dysfunction

Although respiratory muscle weakness could be considered part of the overall muscle involvement in a critical patient, the last 10 years have seen the emergence of the concept of VIDD.[18] This term refers to diaphragm muscle dysfunction caused by the negative effect of the MV itself, that can occur along with or independently of involvement of the rest of the musculature.

Although earlier studies described how complete diaphragm inactivity induced in patients receiving controlled modality ventilation leads to a rapid, progressive loss of diaphragm function,[19,20] it was not until 2004 that Vassilakopoulos first coined the term VIDD,[18] defined as a progressive loss of diaphragm muscle strength that occurs soon after starting MV.[8,21] The condition affects up to 65% of ventilated patients,[7] and is clinically significant due to its early onset.

Pathophysiology of Ventilator-induced Diaphragm Dysfunction

Several studies have shown that the use of controlled MV (CMV), in which the patient makes no inspiratory effort and the diaphragm is not actively contracted, can lead to contractile dysfunction and diaphragm atrophy within 24 h in both laboratory animals and humans.[20,22,23] Atrophied muscles lose strength and the diaphragm excursion is diminished due to the reduction in the cross-sectional area of the muscle fibers, manifesting as a loss of inspiratory capacity.[7,24] Below we discuss the physiopathological principles associated with VIDD.

Diaphragmatic Atrophy

CMV-induced diaphragm atrophy occurs extremely rapidly.[22] Significant diaphragm atrophy can be observed in laboratory animals within the first 12–18 h of starting CMV, with no sign of peripheral atrophy.[25] Thus, MV-induced diaphragm atrophy is significantly greater than the atrophy caused by skeletal muscle disuse.[22] Levine et al. encountered similar findings in patients receiving CMV for 18–69 h, and showed a significant reduction of around 53%–57% compared to healthy volunteers in both type 1 and type 2 fibers in cross-sectional diaphragm biopsies.[23]

Changes in Muscle Fiber Ultrastructure

CMV causes time-dependent changes in the ultrastructure of the diaphragm muscle fibers.[19,26,27] First, areas containing abnormal myofibrils due to myofibrillar disorganization and Z-band changes appear.[19] This is followed by the appearance of areas of muscle fiber regeneration, with no signs of inflammation.[27] Finally, if MV is prolonged (over 3 days), cytoplasmic lipid vacuoles increase, probably due to an autophagic process.[27–29]

Contractile Dysfunction

In 1994, Le Bourdelles et al. were the first to describe, in an animal model, the appearance of contractile dysfunction 48 h after starting CMV.[30] Prolonged MV causes progressive, time-dependent loss of diaphragm strength.[20] As with atrophy, this reduction in diaphragm strength can be seen within 12 h of CMV.[31,32] Several studies have shown that the maximum inspiratory pressure peak is lower in patients receiving prolonged MV compared to controls.[18]

Changes in Protein Synthesis

Diaphragmatic atrophy and dysfunction associated with the use of CMV occur primarily due to a reduction in protein synthesis and an increase in proteolysis.[23,33] Diaphragmatic protein synthesis can fall by 30% after only 6 h of MV.[33] Increased proteolysis is associated with the activation of protease pathways (calpain, capase-3, and ubiquitin-proteasome system), apoptosis pathways, and activation of autophagy.[34,35] Hooijman et al. analyzed the activity of the ubiquitin-proteasome pathway in diaphragm biopsy samples from patients ventilated during thoracic surgery. Patients with a significant increase in this pathway showed a loss of approximately 25% of both slow and fast twitch diaphragm muscle fibers on cross-sectional biopsy, and reduced contractile strength.[36,37]

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