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RESEARCH ARTICLE

SELF-INFLICTED INJURY BY THE PATIENT HIMSELF: P-SILI IN ACUTE COVID DESEAS, CASE AND LITERATURE REVIEW

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ABSTRACT

The spontaneous breathing of a conscious patient with pneumopathy has always made it possible to avoid all the complications induced by mechanical ventilation, namely Baro trauma and Volo trauma , nevertheless spontaneous ventilation proved the risk of self-inflicted injury by the patient himself: P-SILI

INTRODUCTION

The spontaneous breathing of a conscious patient with pneumopathy allows us to avoid intubation as well as sedation, thus limiting the dysfunction of the diaphragm and delirium post sedation, allows for easier mobilization and prevents infections and acute weakness in resuscitation service. On the other hand, controlled and invasive mechanical ventilation allows precise breathing, limits air leakage, optimizes PEEP and current volume control, facilitates positioning (Ventral decubitus, Lateral), diagnostic procedures and patient transport. Recently a new concept of patient self-inflicted lung injury (P-SILI) has been proposed that describes all lesions induced by spontaneous ventilation of the patient, The lungs are damaged due to high respiratory power resulting in global/regional pressure/volume changes which aggravates the lung initially reached. In this manuscript, we present a clinical case of a patient with pneumonia covid 19 who presented P-SILI and will discuss the pathophysiology of P-SILI, the types of P-SILI, and their clinical consequences.

Case Report: This is a young patient on 50years old with a lupus complicates thrombocytopenia on Rituximab, and corticosteroids, admitted for management of respiratory distress pneumonia COVID 19, clinical examination finds: intercostal pulling and contraction of the suprasternal muscle, a thoracic CT was made : 20% of the lesions on frosted glass classed cards 4 without other associated CT lesions (Figure 1). put it under the oxygen scope with a flow of 2 liter/min. 5 days later the patient presented a dyspnea (NYHA 4) desaturation in ambient air at 20% with subcutaneous emphysema , Requiring high flow nasal oxygenation with a flow of 60 liter/min and FiO2 at 100%, a thoracic CT angiography objectifying: a worsening of lung damage with an estimated 75%, the presence of a pneumothorax blade on the right and a pneumo mediastinum near to the cervico-thoracic trachea (Figure 2), Without any sign of Pulmonary Embolism, the patient was then put on protective mechanical ventilation (Baby lung ventilation) due to the worsening of her clinical state.

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DISCUSSION

Can spontaneous ventilation cause lung damage?: Two studies in 1988 have already suggested or demonstrated the link between spontaneous ventilation and P-SILI. One was a classic study by Dreyfuss *et al*. (1), showing that the high current volume generated by negative pressure ventilation in animals leads to pulmonary edema observed during pressure ventilations. It was not a spontaneous breathing, but it keeps the same ventilatory profile as those generated by Respiratory Muscles. The other study was by Mascheroni *et al*. (2), which showed that the injecting of sodium salicylate into the sheep magna tank produce spontaneous hyperventilation without mechanical ventilation, which is responsible for a decrease in compliance, hypoxemia and pulmonary morphological lesions

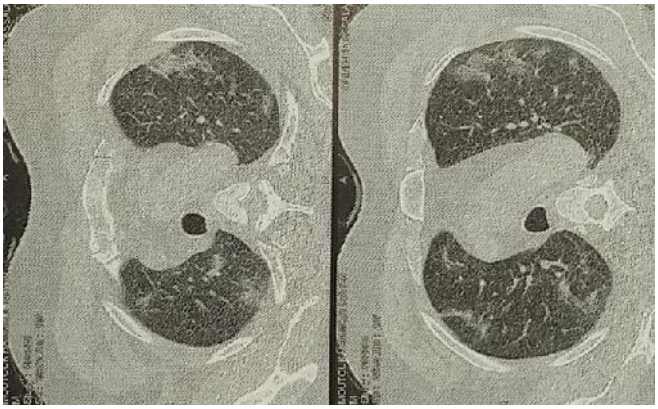

Figure 1: Initial Chest CT

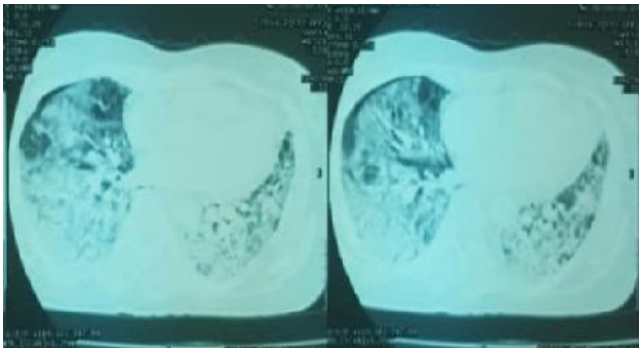

Figure 2. Chest CT with right pneumothorax blade

similar to those observed in VILI. In other words, once the lungs are overloaded, the lung injury can develop regardless of the source of repeated distension. Importantly, this injury does not have to develop after a totally controlled ventilation therefore mechanical ventilation can protect the lungs from P-SILI (3).

P-SILI pathophysiology: During spontaneous breathing, airway pressure is lower than during controlled mechanical ventilation, but this does not result in a lower pressure acting through the lung, which is trans-pulmonary pressure (LP). In spontaneously breathing patients, the primary determinant of the LP is inspiratory effort, which can be measured by negative esophageal pressure deflection (PES). During AHRF and ARDS, respiratory effort is increased this intense inspiratory effort (4) may induce pulmonary damage due to LP fluctuations that abnormally increase pulmonary stress and generate inflation of large volumes in a significantly reduced aerated compartment (baby's lung) (5,6,7)

PEEP typically applied during VNI or HFNC (2-7 cmh2 W) does not prevent shear-induced damage from repeated opening and closing of the alveoli (Atelectraumatism) (8), while muscular effort increases vascular transmural pressure and vascular permeability through deep negativation in pleural pressure, contributing to the appearance of pulmonary oedema at negative pressure (9,10). In addition, differences in lung tissue transmission (solid versus liquid behaviour) diaphragmatic contractions generate heterogeneity in local trans pulmonary pressure, thus improving intra-tidal gas transfer from non dependent pulmonary regions to dependent pulmonary regions, which was recently identified in the early inspiration phase of the spontaneously breathing subjects with ARDS. This phenomenon of pendelluft produces over stretching in the lung regions depending on the size of the current volume inspired (that is, the volume of over stretching comes from the healthy aerated lung and not from the fan), and it has recently been shown to participate in lung damage during spontaneous breathing but not during paralysis and fully controlled ventilation.Finally, the diaphragm can also be damaged by high inspiratory force, due to the development of excessive high mechanical forces. The lesion of the diaphragm is characterized by a rupture of sarcolemma, eventually resulting in a weakness of the diaphragm, which negatively affects the short- and long-term clinical outcomes (11,12,13)

P-SILI Vicious Circle: Patients with pulmonary lesion have more important and hard respiratory function due to impaired respiratory mechanics and hematosis (frequently called respiratory drive).Thus, significant inspirational efforts are accompanied by the risk of over-distension, "pendelluft", atélectrauma and volotrauma of dependent regions, Increased vascular transmural pressure increasing lesion edema and are therefore likely to aggravate pre-existing lesions. All these phenomena were titled By Brochard *et al*. Under the term P-SILI (patient self-inflicted lung injury) (3). P-SILI will aggravate respiratory mechanics and hemostasis, further increasing respiratory function in return and thus further exposing the patient to the risk of P-SILI (Figure 3) (14). It is a sort of vicious Dynamic circle that must be broken. Invasive mechanical ventilation could then be proposed as a means of protection against P-SILI. (14).

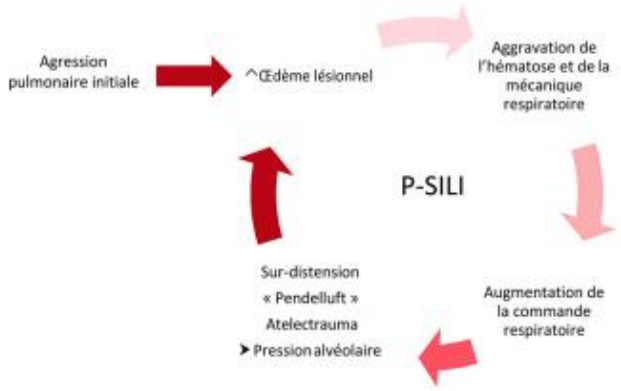

Figure 3. Illustration of the P-SILI vicious circle [14].
P-SILI: patient self-inflicted lung injury

What can be done during P-SILI: The reduction and protection of P-SILI can be achieved through two approaches. The first is the reduction or complete elimination of respiratory control (15). Simple, easy to obtain and effectively, but risk of obtaining against the account of resuscitation neuromyopathy and Obviously, it is not always possible to keep patients in passive respiratory stress depression.This means that a positive ventilation superimposed on the patient's spontaneous effort, that is, assisted ventilation, with an optimization of the ventilation strategy, should be used in this situation, First, a reliable parameter of respiratory mechanics must be used to assess the over-pulmonary stretch at the bedside.Ideally, pulmonary fatigue (pulmonary deformation related to their original size) seems to be the best indicator for assessing pulmonary overstretching.Evaluation of pulmonary tension requires evaluation of pulmonary volume, which is difficult to do in common practice (16).

Alternatively, pulmonary stress (internal force per area experienced by the lungs) can be used as a substitute. Pulmonary stress (tensile stress) is usually scaled by trans-pulmonary pressure, the difference between airway pressure and pleural pressure.Pleural pressure is generally estimated by esophageal pressure, which can be obtained by placing an air-filled catheter in the esophagus (17,18). This technique, although useful, has not been implemented in current practice (19).Clinicians often replace trans-pulmonary pressure with airway pressure to assess pulmonary risk of over-stretching regardless of the variety of mechanical chest wall.With an underestimation, but might be acceptable when the airway pressure is the only external force of lung stretching (during passive ventilation) and impairment of chest wall mechanics is not significant. During spontaneous ventilation, the respiratory muscles apply negative pleural pressure. The enormous variety of this negative pressure makes that the pressure on the airways delivered by the fan is far from representing the Trans pulmonary pressure. For this reason, the difference between maximum airway pressure and PEEP in pressure target mode cannot be considered as an approximation of driving pressure.Direct measurement of airway plateau and motor pressures during spontaneous breathing are possible, but requires fairly relaxed respiratory muscles to perform an end of inspiration.

The second approach is to limit the current volume during spontaneous breathing, we will need to use a volume targeting in this case. The volume-focused mode can adjust the flow by decreasing the airway pressure applied when a patient generates inspiratory effort (negative pleural pressure);While the pressure target mode increases the flow rate to maintain a preset objective pressure (17). In other words, it is better to use a volume mode while monitoring pressures than the other way around.

Detect P-SILI risk during non-invasive strategies : A high current volume (greater than 9–9.5 ml/kg theoretical ideal weight) was identified as a predictive failure volume of non-invasive ventilation in patients with acute respiratory failure (20.21), suggesting the occurrence of P-SILI in this situation. FLORALI (22),In his cohort he objectified a decrease in mortality at j90, with the use of high-throughput nasal oxygen therapy in comparison with the NIV. Explain by the decrease in ventilatory need by a physiological effect, the washing of the anatomical dead space.In practice, the high-throughput nasal oxygen therapy significantly reduces the risk of P-SILI compared to the NAV. However, there is not much publication and research in the literature that describes and detects P-SILI in non-invasive techniques and guides therapeutic choice (23,24).In this case, if the NIV is performed in a patient with acute hypoxemic respiratory failure, it is preferable to monitor the current volume and seek a therapeutic alternative if the current volume exceeds 9–9. 5 ml/kg theoretical ideal weight. In general (14).

Conclusion

The concept of P-SILI opens a wide space of research and study, in order to better know the pathophysiology, clinical repercussions as well as better adapt the so-called protective ventilation strategy that ensures good respiratory control.

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