PNEUMOMEDIASTINUM SECONDARY TO BAROTRAUMA PRODUCED BY NON-INVASIVE MECHANICAL VENTILATION IN COVID19

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June 15, 2020

Introduction:

Pneumomediastinum produced by barotrauma is a possible complication from the mechanical ventilation, since the positive pressure produces alveolar rupture. The incidence is variable, and increases according to the indication of mechanical ventilation (1). In a prospective study with 5183 ICU patients, the overall incidence was 2.9%, reaching 6.5% in patients with acute respiratory distress syndrome (ARDS)(2). The incidence is higher in invasive ventilation than in non-invasive ventilation (NIV) or continuous positive airway pressure (CPAP). Barotrauma risk can be diminished with protective measures, such as using low support pressure and reduced tidal volumes (1,3).

During the health crisis caused by the COVID-19 coronavirus pandemic, a high percentage of the severe patients (up to 15.6% in a review of 1099 patients in Wuhan, China) had respiratory distress (7). In the context of respiratory failure the first step is conventional oxygen therapy. Secondly, if it’s available, the High-flow Nasal Cannulas (HFNC). And finally invasive or non invasive ventilation (NIV). This last one should not postpone intubation (8).

We present the case of a patient with COVID19 positive who, after starting an NIV, suffered a pneumomediastinum and subcutaneous emphysema.

Case Report:

The patient was a 60-year-old male with a history of hypothyroidism and metastatic rectum adenocarcinoma (diagnosed in 2018) with retroperitoneal, iliac and mediastinal adenopathies and pulmonary affection (a 5mm pulmonary nodule in the inferior right lobe), in active chemotherapy treatment.

He went to the emergency room with a clinic of cough and expectoration with two weeks of evolution, and fever with progressive dyspnea (finally at rest) in the last week. On the pulmonary auscultation presented roncus and diffuse crackles in the left hemithorax.

Analytically the patient had a PCR 67mg/L, a PCT 0.17ng/mL, a leukopenia (1,75x10⁹) with lymphopenia (0,26x10⁹ [14.9%]) and a D-dimer of 2133ng/mL. In the chest X-ray was possible to observe an interstitial patron, with increased diffuse density in lower left hemithorax. Given the high suspicion of coronavirus infection as a cause of the condition of the patient (fever + lymphopenia + pneumonia) a PCR for COVID-19 was requested, being positive.

In the arterial blood gas was observed a respiratory alkalosis and an acute respiratory distress (pH 7.51, PaCO₂ 26mmHg, PaO₂ 51mmHg, and a calculated PaO₂/FiO₂ of 60). Due to the palliative state of the patient, it was decided to treat the acute respiratory insufficiency with CPAP, excluding an ICU assistance.
During hospitalization, the patient increased respiratory work and the oxyhemoglobin saturation was \(<90\%\) despite a FiO$_2$ of 50\% with Ventimask. It was decided to place a CPAP of 12 cmH$_2$O, and the patient presented a clinical improve with a respiratory frequency of 18/min and a 94\% saturation with an additional oxygen of 15 lpm.

After 24 hours of CPAP treatment, the patient presented a sudden clinical deterioration, with important desaturation, (SaO$_2$ 72\%), with an extensive subcutaneous emphysema in the anterior thorax, up to the neck and both arms.

Given the suspicion of pneumothorax secondary to barotrauma, the ventilation was interrupted and an urgent chest X-ray was requested, which didn’t confirm the suspicion. The chest CT scan confirmed a severe emphysema of thoracic soft parts, important neumomediastinum, right apex pneumothorax and ground-glass lung opacities in all lung fields, compatible with COVID-19 infection.” (Figure 1)

A pleural drainage was placed in the small right pneumothorax. The patient didn’t present any clinical improvement, and maintained saturations below 80\% with oxygen therapy with Ventimask at FiO$_2$ of 50\% and 15 lpm. Due to increased dysneic sensation, and poor prognosis, it was started a sedation with morphine, and the exitus occurred in the following hours.

**Discussion:**

The incidence of pneumomediastinum secondary to NIV is low (1). This pathology should be suspected in case of subcutaneous emphysema or dyspneic sensation, mostly if there’s a concomitantly pneumothorax. The risk increases, according to the cause for which mechanical ventilation is required, in the patient exposed it was secondary to an ARDS infection by COVID-19.

Due to the vital risk that pneumomediastinum can produce, especially when accompanied by pneumothorax, it’s important to suspect it early and confirm urgently using imaging techniques. Pneumomediastinum is associated with increased morbidity and mortality, so avoiding it’s decisive.

To prevent it is crucial to carry out protection strategies during ventilation, such as maintaining pressure Plateau \(<30\text{cmH}_2\text{O}\) (only possible in patients with invasive mechanical ventilation) or maintaining reduced tidal volumes (between 6 to 8 mL/kg based on the ideal body weight), or low support pressures (3).

In our patient, COVID-19 positive, even performing mechanical ventilation with protection strategies, such as keeping volumes reduced, and being in CPAP mode at 12 cmH$_2$O, it was produced a pneumomediastinum with its consequent worst outcome (6).

Therefore, in addition to the increased risk of pneumomediastinum due to ARDS caused by the COVID-19 infection, we cannot rule out if this infection may cause respiratory damage that may increase the risk of barotrauma, and if those patients should be closely monitored.

There’re only two reported cases of spontaneous pneumomediastinum in a positive COVID patient, who were not submitted to ventilation (4,5).

We still don’t have enough information available about all the phisiopatology of the coronavirus infection and we will have to wait for more publications and case series studies.

**Figure:**
Figure 1.- Thoracic CT scan: abundant emphysema of thoracic soft parts and neck base. An important neumomediastinum, without compromise of the airway or large vessels. A 12 mm right apex pneumothorax. Ground-glass lung opacities in all lung fields, compatible with COVID-19 infection.

References:


