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Tension pneumothorax in a patient with covid 19 maintained on non invasive positive pressure ventilation

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Abstract

COVID 19 is the name given to disease caused by the Novel Coronavirus of 2019, SARS COV 2 that started in the city Wuhan of China as an epidemic has soon become a pandemic. SARS COV 2 as the name suggests causes severe acute respiratory syndrome ARDS that is responsible for the tremendous morbidity and mortality. It also has been observed to cause pneumothorax. We describe a case of a 60 years old male, who presented to the ER with COVID pneumonia on April 16th. He was maintained on a BIPAP device as he was in severe acute hypoxemic respiratory failure and a month into admission on May 13th the patient developed right-sided pneumothorax. A chest tube was placed and the patient showed clinical improvement with resolution of pneumothorax. During his stay, he started developing increasing needs for oxygen and a high work of breathing but he continued to refuse invasive mechanical ventilation. On 1st June he developed severe dyspnea despite the BIPAP device with supplemental oxygen and a chest X-ray done showed right-sided tension pneumothorax and pneumomediastinum with worsening of bilateral opacities. He was taken emergently to the Interventional Radiology for the placement of another chest tube and was subsequently transferred to ICU. The subsequent imaging showed lung expansion. He was then switched to a high flow nasal cannula. 2 weeks later he was discharged to acute rehabilitation with 50 liters on high flow and a mild persistent pneumothorax. We postulate the use of positive pressure ventilation even noninvasive like BI-PAP can predispose the COVID patients to pneumothorax so there needs to be more research in comparing various noninvasive mechanical ventilation strategies to better help these patients.

Keywords

Pneumothorax; SARS COV2; Pneumomediastinum; BIPAP.

Background

SARS COV 2 induced respiratory illness that is responsible for causing ARDS in many cases is the cause of concern all over the world right now [1]. COVID-19 can induce a spectrum of respiratory illnesses including pneumonia progressing to complications like ARDS and pneumothorax [2]. Pneumothorax is defined as the presence of air in pleural space which can be spontaneous or iatrogenic. Spontaneous pneumothorax can happen without an inciting factor. We will discuss here the development of spontaneous pneumothorax in a patient with COVID pneumonia maintained on BIPAP [3]. There have been some reports of patients in the literature that show the development of pneumothorax with COVID-19, the pathogenesis could be the rupture of bullae [4,5]. We hereby describe a case where a patient positive with SARS COV 2 came into respiratory failure and developed recurrent pneumothoraces while being maintained on Non-Invasive Positive Pressure Ventilation. We have very limited literature saying that BIPAP and positive airway pressure ventilation can predispose to spontaneous pneumothorax. As in our case, BIPAP seemed to be the causative factor because the patient did not get further complications when he was switched to a high flow nasal cannula. There is some evidence that NIPPV can increase the risk of pneumothorax development in patients with some underlying diseases like cystic fibrosis and in patients where the exposure is prolonged. Patients with COVID-19 infection can have increased risk for these complications with positive pressure ventilation that can be avoided with alternative therapies to prevent the comorbidity [6,7].

Case Presentation

We describe the case of a 60 years old male with Past Medical History of Hypertension, Atrial Fibrillation, Congestive Heart failure with an AICD presented on April 16th, 2020 with 2 weeks of cough and shortness of breath. He was tested positive for SARS COV on April 8th and was staying at home. He was tested as he started having a cough a couple of days before. In the Emergency Room, the Vitals of the patient were Blood Pressure 138/78, Heart Rate 104, Oral Temperature 102.8 F, Respiratory Rate 28, and Pulse oximetry showed 96% O_2 saturation on 100% Non-Rebreather. He was desaturating and was hypoxic so he was placed on 100% Non-Rebreather by the paramedics. His Laboratory data showed Leukocytosis with a White Cell Count of 17000 and a bandemia of 12%, Lactate of 6.2, Ferritin 805, Procalcitonin 5.68, Blood Glucose of 468, although he was not a diagnosed diabetic. The patient was admitted to the Intensive Care Unit for acute hypoxemic respiratory failure and was started on Non-Invasive Mechanical Ventilation NIPPV (BIPAP).

He was started on hydroxychloroquine with little effect. He continued spiking temperatures and his oxygen status did not improve so he stayed on a BIPAP device. At this point, he was also given a trial of steroids. On day 5 into his admission, his fever improved but his respiratory status worsened. He was offered mechanical ventilation which he refused as he did not want to be intubated. His labs showed a worsening of leukocytosis. The ferritin levels though started to come down from 1991 in the beginning down to 1779 on the 3rd day and 1398 by the 7th day. They correlated with the resolution of fever. The patient was then transferred to the telemetry unit. His respiratory status had stabilized with BIPAP and he was not on escalating doses of oxygen (Figure 1) April 24th CXR.

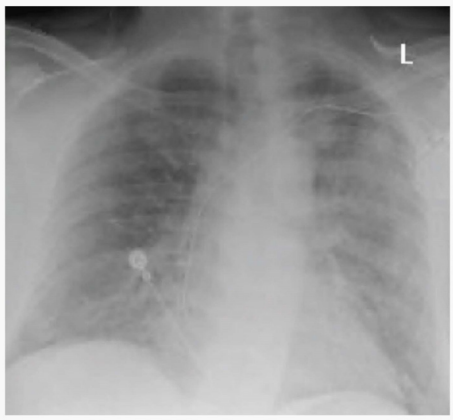

Figure 1: April 24th CXR 1.

The chest X-ray showed some improvement of bilateral infiltrates although he kept requiring BI-PAP.

On May 13th a rapid response was called as the patient developed chest pain and worsening hypoxia so a chest X-ray was ordered that showed the interval development of right-sided pneum mothorax.

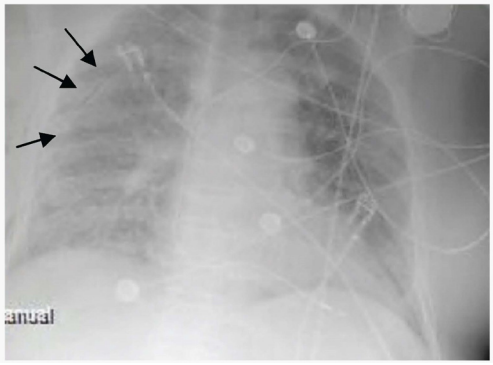

Figure 2: May 13th CXR Pneumothorax, Arrows depicting lung and pneumotothorax border.

A cardiothoracic surgery consultation was called and a right-sided chest tuube was placed. During all this time the patient stayed on a BIPAP device with 80% FiO_2, rate of 14, and 90% oxygen saturation. The subsequent chest X-rays showed the resolution of pneumothorax. Since the respiratory status did not improve much another chest X-ray was done that showed a mass at the left lung base. The CXR and CT scan from the date of May 24th are shown here.

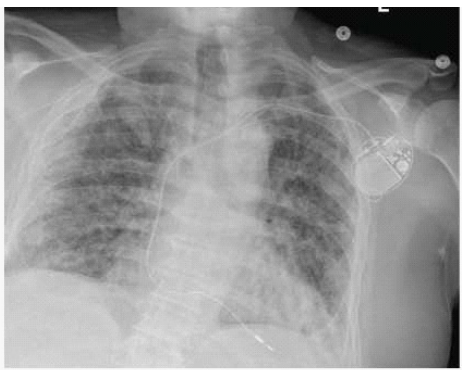

Figure 3a: CXR May 24th.

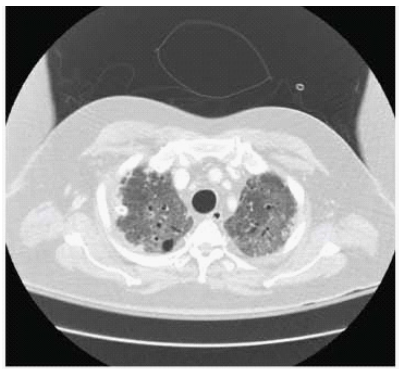

Figure 3b: CT chest May 24th.

May 28th CXR shows the changes in pulmonary fibrosis. Since the respiratory status started to improve a decision was made to take the chest tube out. The chest tube was clamped a day before the date of

removal but the patient could not tolerate it being clamped and he became very tachypneic, dyspneic and severely hypoxic. As a result of the worseninng of his clinical status he was evaluated and a chest X ray was done. The chest X ray showed that there was an interval development of tension pneumothorax on the right side (June 1).

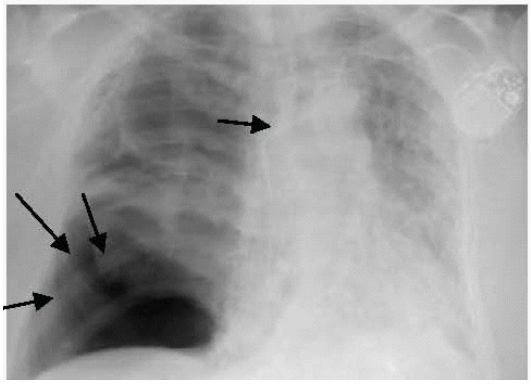

Figure 4a: Tension pneumo, June 01.
Arrow shows tension pneumothorax and shifting of the trachea.

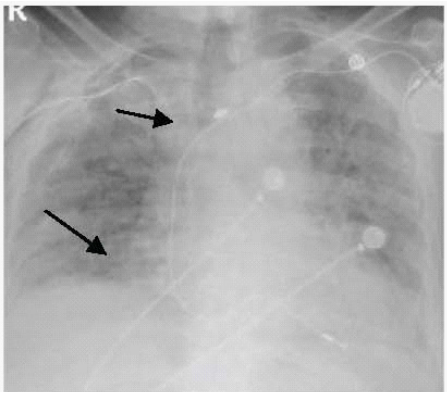

Figure 4b: CXR June 01.
Arrow shows tension pneumothorax and shifting of the Arrows show resolution of tension pneumo with resolution of the trachea tracheal shift as well after chest tube placement.

Chest X-ray confirmed tension pneumothorax and also showed persistent patchy bilateral opacities with a small left effusiion. A chest tube was placed emergently by the interventional radiology and that resolved the tension pneumothorax. The patient started to improve but still requiring very high oxygen conc centrations. But this time he was switched from BIPAP to high flow nasal cannula. The following images are the CT scans taken in the subsequent days of his hospital stay. These CT scans show the extensive fibrosis with some persistent pneumothorax and pneumomediastinum on the right side.

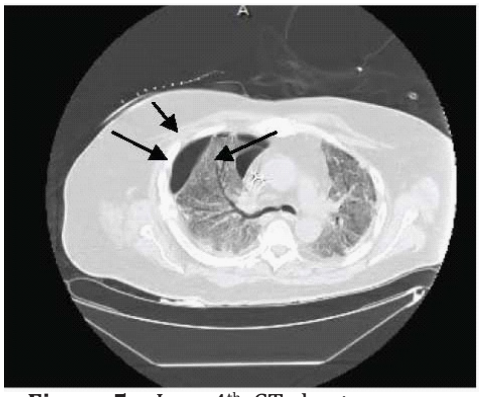

Figure 5a: June 4th CT chest.

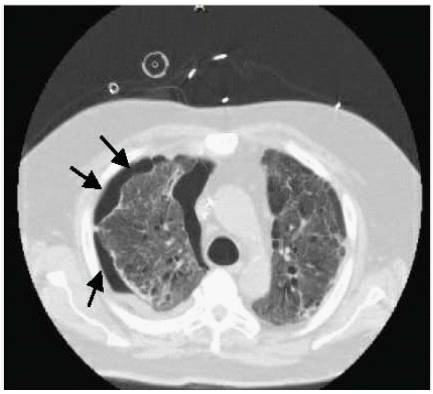

Figure 5b: June 11th CT chest 1. The arrows shows the persistent pneumothorax and pneumomediatinum.

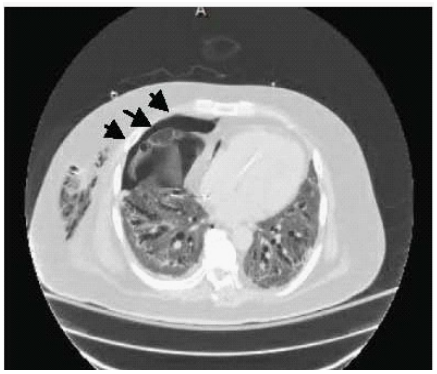

Figure 6a: CT chest June 17th. CT chest Arrows showing pneumothorax.

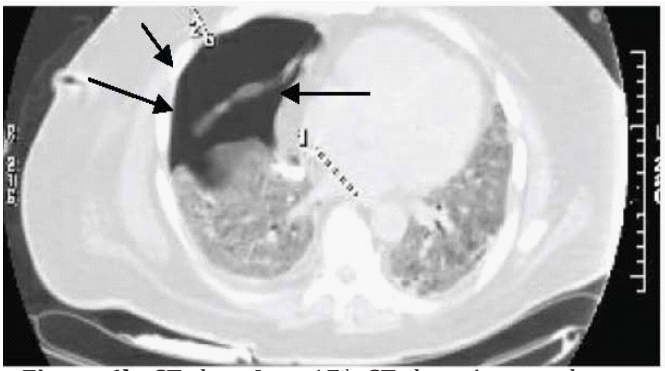

Figure 6b: CT chest June 17th. CT chest Arrows show pneumothorax

He still had 2 chest tubes on the right side but after being switched to HFNC (High Flow Nasal Cannula) and his respiratory status started getting better and no further new pneumothorax developed. He was then transfferred to an acute rehabilitation facility for requiring skilled nursing needs with high doses off oxygen.

Discussion

There is some literature regarding ventilator-induced lung injury with invasive ventilation but there is limited data as to whether noninvasive strategies have any risk [8]. Here we discuss the recurrent pneumothorax with the development of tension pneumothorax as well after the use of BIPAP in a COVID-19 patient. Patients with COVID 19 have shown varying presentations that include trivial symptoms to florid respiratory failure and the complications have involved all organ systems [9,10]. The respiratory complications are mostly from the result of Acute Respiratory Distress syndrome that happens as a result of florid pneumonia. There have been reports of spontaneous pneumothoraxes and pneumomediastinum and the pathogenaesis could be alveolar damage and pleural inflammation. Other than the remote history of smoking there was minimal risk factors for fulminant respiratory disease [11]. The COVID-19 infection resulted in significant hypoxia that led to the use of BIPAP. He was maintained on BIPAP when his pneumothoraxes happened and was finally switched to high flow nasal cannula with eventual stability and discharge to subacute rehabilitation institute. We do have some literature review that suggests that Non Invasive ventilation improves outcomes in patients with hypoxemic respiratory failure but limited data as to which modality should be used [12]. The review of studies illustrates some data comparing the BIPAP with high flow in patients with acute hypoxemic failure [13]. These studies suggest the effects of BIPAP devices on the respiratory status of the patient. Besides these risks, we all know that the use of BIPAP in these patients will also predispose the staff to excessive aerosol exposure and the potential spread of pathogens like SARS COV 2. The spread can though be minimized with nasal prongs delivering high flow oxygen with humidified air. We need to have good comparative trials where we can give definitive answers to which mode of Non-Invasive Ventilation is better. If BIPAP is responsible for serious complications that can cause high morbidity and mortality with increased needs of ICU and increased length of hospital stay we need to rethink its use in cases of Covid-19 pneumonia. Pneumothorax in our patient led to a prolonged hospital stay requiring the use of Surgical and Interventional Radiological maneuvers for management. A significant cost and health burden are observed with complications like this. On the first note, this serious life-threatening complication might have been avoided with the use of alternative means for oxygen supplementation. Moreover, once we use BIPAP on patients with extensive lung damage we should always keep complications like pneumothorax and pneumomediastinum high up on the differential in case these patients show deterioration. This also suggests the need to have more trials comparing the different modes of noninvasive ventilation in these patients.

Conclusions

There needs to be a high index of suspicion for life-threatening complications like tension pneumothorax in COVID 19 patients, especially those who are on BIPAP devices so early life-saving measures can

be taken. There also needs to be caution with the modes of NIV strategies so these complications can be avoided in the first place.

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