



Case Study of Pneumothorax in a 27-year-old Male as a Possible Post-COVID 19 Infection Complication

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Abstract

Spontaneous Pneumothorax is a potentially life-threatening condition that is brought on when air makes its way into the pleural spaces resulting in decreased negative pressure between the pleural membranes without trauma or injury occurring. Spontaneous pneumothorax was reported as an adverse complication of severe acute respiratory syndrome brought on by SARS-CoV-2. It is thought spontaneous pneumothorax may be a consequence of the breakdown of elastic fibers within the lung tissue. This breakdown is believed to be caused by the body's own immune response in particular by innate lymphoid cells (ILCs) which have been shown to be elevated in response to intracellular pathogens like viruses, but additional research is needed to clearly state a causal relationship between SARS-CoV-2 and the immune-mediated formation of pneumothorax. Limited studies look at the link between the pathophysiology of Pneumothorax and COVID-19, which is why the patient from this case is of interest. We present a unique case of a 27-year-old male presenting to the Emergency Department complaining of difficulty breathing, chest pain, and fatigue who had prior tested positive for COVID-19. A diagnosis of pneumothorax was made by chest x-ray. In this report, we discuss the pathophysiology, imaging, and management related to pneumothorax and its connection to COVID-19.

Keywords

Pneumothorax, COVID-19, Case Report, Innate Lymphoid Cells

Introduction

Pneumothorax occurs when air enters the pleural spaces, resulting in decreased negative pressure between the pleural membranes [1]. It is usually the result of a disease such as a viral infection or an injury in the vicinity of the lungs and can lead to the collapse of the lungs. Pneumothorax that occurs without an injury is known as spontaneous pneumothorax and is divided into primary and secondary spontaneous

pneumothorax based on whether there is underlying lung disease or not [2]. Smokers are at higher risk for pneumothorax, as smoking breaks down type 1 and type 2 alveolar cells which line the alveolar sacs, resulting in the formation of bullae which can burst, thus allowing air to enter the pleural space [3]. This results in various symptoms which are clinically seen, such as chest pain, dyspnea, and absent breathing sounds.

Although pneumothorax can be diagnosed clinically by physical exam, in stable patients, a chest X-ray or ultrasound is usually used to diagnose pneumothorax [4]. Once the diagnosis is made, respiratory support and in most cases, a chest tube are needed to remove the excess air in the pleural space [5]. If the patient is unstable, which is classified by a respiratory rate greater than 24 breaths per minute and an oxygen saturation less than 90%, the patient will require a needle thoracostomy followed by a chest tube placement, most commonly placed between the 4th and 5th intercostal space, between the anterior and midaxillary lines [6,7]. If left untreated, a pneumothorax in an unstable patient may progress to a life-threatening tension pneumothorax.

SARS-CoV-2 is a coronavirus responsible for Covid-19. It is an enveloped, positive-sense ssRNA coronavirus [8]. The primary mode of transmission for the virus is via exposure to respiratory fluids. The incubation period for the virus may vary between 2 and 14 days and could potentially be shorter with newer variants [9,10]. Other symptoms include loss of taste and/or smell, sore throat, rhinitis, and shortness of breath [12]. The severity of these symptoms can also vary as many patients can be asymptomatic while others experience critical symptoms that can be life-threatening [13].

The virus works via the use of its spike protein which binds to the angiotensin-converting enzyme 2 (ACE2) [14]. ACE2 receptors are located on the surface epithelium of the lungs along with other organs such as the heart, kidneys, and nasopharynx [15]. Following this binding, transmembrane protease serine 2 (TMPRSS2), located on the respiratory endothelium, activates the spike protein which then allows for membrane fusion and uncoating of the viral RNA [14,15]. The primary effects of these mechanisms are seen in the alveolar epithelium leading to the breathing issues commonly associated with COVID-19 [16]. Another effect of this pathophysiology is a dysregulated immune response which can lead to inappropriate activation of the immune system via the release of cytokines such as tumor necrosis factor, Interleukin-1 β (IL-1 β), and Interleukin-6 (IL-6) which can go on to cause a cytokine storm in some patients that can lead to organ failure and death [17].

In some patients, spontaneous pneumothorax has been reported as an adverse complication of severe acute respiratory syndrome (SARS), which is a coronavirus variant. Patients with pneumothorax commonly present with symptoms of chest pain, difficulty breathing, elevated carbon dioxide, and lowered oxygen levels [18]. Common chest x-ray findings usually show the accumulation of air inside the pleural space. Studies suggest that the formation of bullae in patients with spontaneous pneumothorax may be a consequence of the breakdown of elastic fibers within the lung tissue [19,20]. This breakdown is thought to be caused by the body's own immune response, in particular by innate lymphoid cells (ILCs), which have been shown to be elevated in response to intracellular pathogens like viruses [21].

A key immunological finding in other SARS strain infections is the viral interference with the immunological response of ILC-1 cells [20]. This is also suspected with the SARS-CoV-2 virus, but more research is needed to clearly state a causal relationship between SARS-CoV-2 and the immune-mediated formation of pneumothorax [22]. Treatment protocols for SARS-CoV-2 infection and underlying lung diseases like pneumothorax are also affected owing to potential interactions such as COPD [23]. The presentation of pneumothorax may be worsened in patients with existing lung disease or a prior smoking history, as well as barotrauma or damage to the lungs, all of which can exacerbate the condition [24].

Case Presentation

A 27-year-old male presented to the emergency department (ED) with difficulty breathing, chest pain, and fatigue. On arrival, vital signs were BP: 101/55 mmHg, P: 68 beats/min, Resp: 22 breaths/min, Temp: 36.3°C. Physical examination showed the patient was alert, oriented, had good strength and sensation, had no pulsatile masses, was walking, and talking. The patient does not report any history of injury, pain, loss of consciousness, nausea, vomiting, headache, dizziness, abdomen pain, numbness, tingling, loss of limb or bowel/bladder function, pain with urination, thoughts of self-harm, or hallucinations. The patient was breathing easily and talking to their mother throughout the course of the physical exam. Past

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history is significant for smoking, marijuana use, and being COVID-19 positive in April 2022 roughly 1 month before the current ED presentation.

The following lab tests were ordered: WBC, Hemoglobin, Potassium, glucose, magnesium, lipase, lactic acid, heart failure test, troponin, B-type natriuretic peptide (BNP), total creatine kinase (total CK), muscle breakdown test, urinalysis, urine toxicology screen, international normalized ration (INR) test, influenza test, and COVID-19 test. Oxygen saturation was low at 85% on room air and repeated with 93% on room air. His oxygen saturation increased to 100% oxygen on 2 liters of oxygen with nasal cannula. Their blood pressure was stable at 101/55 mmHg. Complete blood count showed a WBC of 12 reactive ($N= 3.7-10.6 \times 10^3$ cells/uL), stable hemoglobin of 14.7 g/dL ($N= 11.5-18$ g/dL), and platelets at 245 mCL ($N=140-425 \times 10^3$ platelets/uL). Potassium levels were low at 2.7 mmol/L ($N= 3.4-4.5$ mmol/L).

Glucose was elevated at 192 mg/dL and 162 mg/dL with repeat ($N= 70-139$ mg/dL). The magnesium level was 2.0 mg/dL ($N=1.7-2.2$ mg/dL) and the lipase level was 24 U/L ($N= 11-82$ U/L). The plasma lactic acid test was elevated at 3.0 mmol/L ($N= 0.5-2.2$ mmol/L) and the Troponin and BNP were within normal ranges at <3 pg/mL and 20pg/mL respectively. There was a low risk of blood clot at 286 ng/mL and the heart failure test was normal. Muscle breakdown test, urine test, and urine drug test were pending. The INR test was 1.0

or normal, the influenza test was negative, and the COVID-19 test was negative.

An anterior-posterior chest X-ray revealed left-sided pneumothorax with the AP view of the chest showing total lung collapse of the left lung. No pneumothorax was seen in the right lung which was clear. An electrocardiogram (ECG) was also performed and detected marked sinus bradycardia at 49 beats per minute (**Fig-1**).

Differential Diagnosis

Based on the patient's history and presentation of symptoms the chief differential was COVID-19-related pneumothorax. Symptoms such as chest pain and shortness of breath could also be due to other respiratory conditions such as asthma. Other diagnoses could include heart disease because of the sinus bradycardia seen on ECG. Once the pneumothorax seen on imaging and the patient's prior positive COVID are taken into account COVID-19-related pneumothorax seems most likely among the diagnosis.

Management

Standard care for pneumothorax includes pain management of the symptoms brought on by the pneumothorax and removing the air that is filling the pleural space through the use of a device such as a chest tube. The patient was treated intravenously with fluids, intravenous steroids, magnesium, duonebs (a combination of Ipratropium bromide/Salbutamol), and

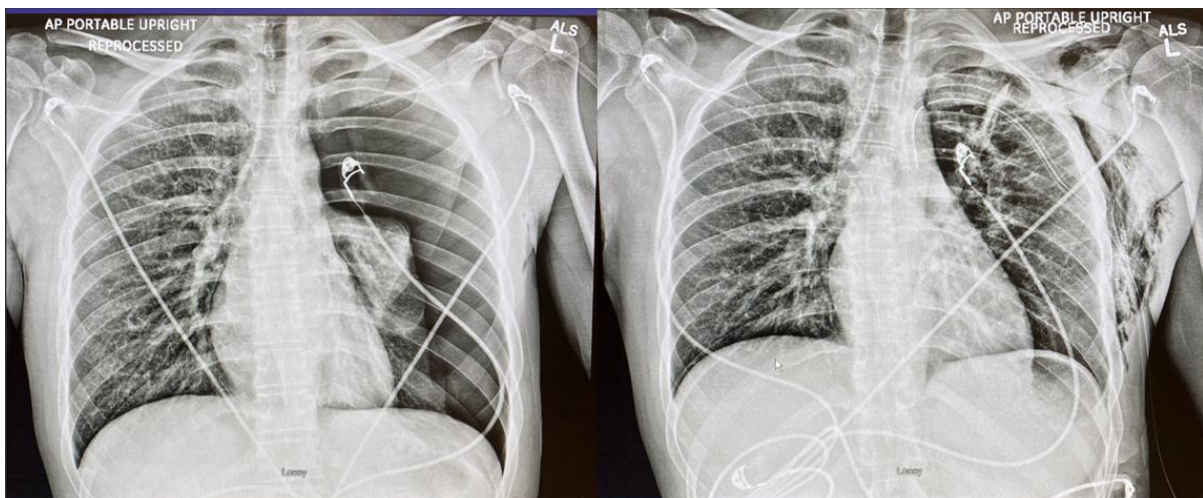


Fig-1: Left Sided Pneumothorax on Chest X-ray Before and after Chest Tube Placement

iv fentanyl to help manage their symptoms. Additionally, a chest tube with local anesthesia was placed in order to create negative pressure in the chest cavity allowing the lungs to expand and also remove air that is filling the pleural space [25]. A repeat Chest X-ray showed the left lung improved as there was less air seen contained within the pleural space once the insertion of the chest tube was performed and chest tube management was done. Additionally, the patient was counseled to stop smoking/using marijuana.

Conclusion

As established before, the patient's prior history of being positive for COVID-19 in conjunction with the imaging which revealed a left-sided pneumothorax helps establish the cause of the patient's symptoms as being due to COVID-related pneumothorax rather than another issue. Destruction of alveolar epithelium and dysregulation of the immune response brought on by the virus contribute to the eventual formation of a spontaneous pneumothorax which develops in severe cases. Our case shows an example of how COVID-19 infection can progress to the formation of spontaneous pneumothorax. Being aware of all the potential complications of an infection is essential when deciding how to treat it. Understanding the link between COVID-19 and pneumothorax is essential to determine effective ways to prevent the progression of COVID so that it does not cause the formation of a spontaneous pneumothorax which can potentially be fatal to the patient.

Conflict of Interest

The authors have read and approved the final version of the manuscript. The authors have no conflicts of interest to declare.

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