Case Report

A Case of Pneumomediastinum Related to COVID-19 Viral Infection

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Abstract

The novel coronavirus, also known as coronavirus disease 2019 (COVID-19) was first identified as a cluster of pneumonia cases in Wuhan, China at the end of 2019. The virus quickly spread, becoming an epidemic and then by February 2020, a global pandemic. The spectrum of severity of symptomatic infection can range from mild disease with or without pneumonia; severe disease, leading to hypoxia, dyspnea, and usually categorized by greater than 50% lung involvement; finally, critical disease which is usually categorized by shock or respiratory failure. The increasing severity of symptoms has been shown to correlate with increasing age, comorbidities including cardiovascular disease, diabetes mellitus, hypertension, chronic lung disease, cancer, chronic kidney disease, obesity, smoking, socioeconomic background, and gender (male predominance). [4] Patients with multiple comorbidities are likely to decompensate. In very rare cases, there are cases of pneumomediastinum seen in patients with COVID-19. We discuss a middle-aged male with multiple comorbidities with COVID-19 that decompensated, received supplemental oxygen, treatment for COVID-19 and developed pneumomediastinum. After extensively searching literature; out of the 49.1 million confirmed cases of COVID-19 in the world to date; there have been less than 68 reported cases of pneumomediastinum as a complication due to COVID-19.

Keywords: *COVID-19, decompensate, pneumomediastinum, subcutaneous emphysema*

Abbreviations:

COVID-19: Coronavirus disease 2019

NC: Nasal Cannula

SPM: Spontaneous Pneumomediastinum

SE: Subcutaneous Emphysema

ARDS: Acute Respiratory Distress Syndrome

PEEP: Positive end-expiratory pressure

BiPAP: Bilevel Positive Airway Pressur

CPAP: Continuous Positive Airway Pressure

ACE-2: Angiotensin-Converting Enzyme 2

SARS: Severe Acute Respiratory Syndrome

CMV: Cytomegalovirus

PCP: Pneumocystis Pneumonia

Introduction:

The novel coronavirus, also known as coronavirus disease 2019 (COVID-19) was first identified as a cluster of pneumonia cases. The severity of symptomatology of COIVD-19 ranges anywhere from mild, severe to critical and has been correlated to a variety of comorbidities. In the reported 370,000 confirmed cases of COVID-19 in the US; the most common initial presentation is associated with the symptoms include fever (85-90%), cough (65-70%), loss of smell or taste (40-50%), fatigue (35-40%), sputum production (30-35%),shortness of breath/dyspnea (15-20%), myalgia/arthralgia (10-15%), headaches (10-36%), sore throat (10-15%), chills (10-12%), diarrhea (3-34%).**[**1**]** As for complications, acute respiratory distress syndrome manifested shortly after dyspnea, and roughly 12-24% of hospitalized patients required mechanical ventilation. Of those patients in the ICU with cardiac or cardiovascular complications, 33% developed cardiomyopathy. Inflammatory complications including persistent fevers, elevated inflammatory markers (ferritin, D-dimer, etc.) as well as elevated proinflammatory cytokines.**[**5**]**

In a comprehensive literature search, pneumomediastinum as a complication of COVID-19 pneumonia is rare. In three separate case reports, it showed that there was a male predominance, had a mean temperature of 37.6°C, mean oxygenation of 89%, cough in 82%, and 91% had shortness of breath. They also had comorbidities including older age, being overweight, asthma, corticosteroids, respiratory irritants, and reported lifelong nonsmokers. [4][8][9][10] In COVID-19 pneumonia, the pathophysiology basis of the air-leak has not yet been well established, however alveolar damage can lead to alveolar rupture leading to emphysema and pneumomediastinum. We present an unusual case about a middle-aged male with multiple comorbidities with COVID-19. He decompensated and pneumomediastinum was found on imaging.

Case Report:

A 52-year-old male with morbid obesity, allergies, hypertension presented with a 7-day history of onset of fever and malaise. 14 days before the onset of symptoms, he had exposure to COVID-19 at his workplace. A COVID-19 test was sent and the SARS-CoV-2 RT-PCR returned positive for viral RNA.

Upon presentation, he had a cough, especially with deep breathing along with dyspnea. His temperature reached a maximum of 101.1°F (38.4°C) but never surpassed that. On pulse oximetry, he was oxygenating at 88% on room air, and 92% with two liters via nasal cannula.

On day two of admission, an Infectious Disease consultation was obtained. Based on the patient's current COVID-19 status, the patient was then started on empiric COVID-19 treatment. This included steroids, 400 mg IL-6 inhibitors, 1 bag of plasma, as well as five doses of Remdesivir.

On day three of admission, the patient began to decompensate and was escalated from 2L to 6L NC but was still saturating in the mid to upper 70s. The Rapid Response Team was called due to worsening hypoxia and he was placed on high flow oxygen, which improved his oxygenation to only mid-80s. He was then placed on non-rebreather and FiO2 100% after which his oxygen saturation remained in the upper 80s to mid-90s. He was on the non-rebreather for a total of 5 days. He was never intubated but instructed to self-prone. After a week on high flow oxygen on nasal cannula, the patient began improving.

Upon completing a physical, it was noted that he had crepitus in his neck and anterior chest. It was also noted that he had bilateral crackles, no neck vein distention. No pleural friction rub, nor pericardial rub were noted. Cough was clear, he was afebrile and not producing any sputum. His WBC was 8.5, and there was no shift. Procalcitonin level was 0.04. As the patient began improving, the lab findings were consistent with good COVID response to therapy the current therapy.

To evaluate further, he had a chest X-ray and CT scan. In **figure 1**, the CT revealed ground-glass opacities and spontaneous pneumomediastinum. The patient in our case developed subcutaneous emphysema and pneumomediastinum 8 days during COVID treatment with steroids and high flow nasal cannula. He was hospitalized for a total of 5 weeks but completely recovered.

Labs:

CRP:	53.7 MG/L	*High*	(08/06/20 07:40:00)
CRP:	102.5 MG/L	*High*	(08/05/20 08:49:00)
CRP:	188.2 MG/L	*High*	(08/04/20 07:15:00)
LD:	286 UNIT/L	*High*	(08/04/20 07:15:00)
LD:	241 UNIT/L	-	(08/03/20 00:20:00)
D-Dimer:	<150 ng/mL DD	-	(08/04/20 07:15:00)
D-Dimer:	<150 ng/mL DD	-	(08/03/20 00:21:00)

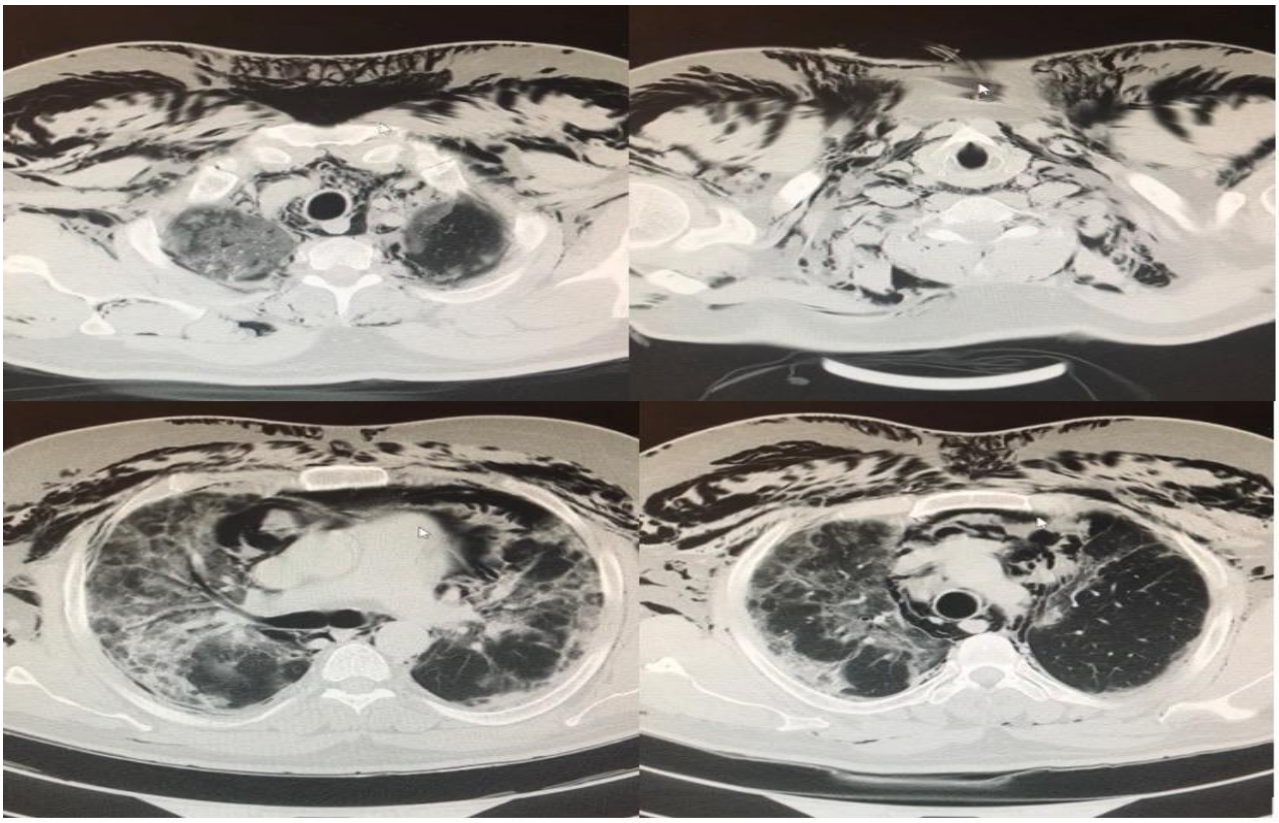

Figure 1: Spontaneous pneumomediastinum

Discussion and Conclusions:

Pneumomediastinum is a rare clinical finding in adults, and in the setting of severe COVID-19 pneumonia infection, it is a cause of concern.**[**9**]** The severe strain that COVID-19 pneumonia due to persistent coughing can be the causative factor leading to a cystic change, that manifest as small air-containing spaces in the lung parenchyma. It remains unclear to what exact pathophysiology leads to the development of these pneumatoceles, but hypothesis supports that this may be explained by damage to the alveolar walls from the COVID-19 virus.**[**6**]** Pneumomediastinum and subcutaneous emphysema may also occur. The patient in our case developed subcutaneous emphysema and pneumomediastinum 8 days during COVID treatment with steroids and high flow nasal cannula.

In 1819 Laënnec, a French Pathologist, first described pneumomediastinum in which there is a communication of gas or free air from the conducting airways or alveolar space to the mediastinum.[3] SPM is the presence of free air in the mediastinal space as well as occasional entry into the pericardium. The Macklin effect is the theory for the pathophysiology for the development of SPM. It proposed that free air was from ruptured alveolus and the perivascular sheaths from overinflation.[8] The air that escapes from the mediastinum can manifest as subcutaneous emphysema if it goes to the head and trunk. [7] The tissue planes of the neck and upper thorax are where free air will track from the mediastinum to the cervical region leading to SE. Since SE predominately tracks superiorly, it can decrease the blood flow to the head and neck and may even constrict the airway.[10]

Pneumomediastinum most commonly results from trauma, surgical procedures, or pulmonary infections. It may also occur secondary to mechanical ventilation because of the increased airway pressures, a rise in intra-thoracic pressure, coughing or vomiting excessively, or secondary to alveolar injury due to disease or infection.[10] They occur more commonly in the pediatric population and it is rarely seen in the adult. Symptomatology can include neck swelling, pain from air dissecting through tissue planes, odynophagia, chest pain, cough, dyspnea voice change, and dysphagia. Also note, that there will be no signs of inflammation, like heat, redness, or erythema.[3]

In a study about spontaneous subcutaneous emphysema and pneumomediastinum in non-intubated patients with COVID-19, 91% of the patients received supplemental oxygen, 36% received non-invasive ventilation from BiPAP or CPAP, 45% received high flow nasal cannula, 10% received either oxygen from non-rebreather or no supplemental oxygen. It also noted that SPM has been previously reported in respiratory infections such as SARS, CMV, PCP, Staphylococcus aureus pneumonia, and influenza bronchiolitis.[4]

In COVID-19 pneumonia, the pathophysiology basis of the air-leak has not yet been established, however, it is acceptable to apply the existing concept of SARS. SARS causes diffuse alveolar damage which can lead to alveolar rupture and may cause pulmonary interstitial emphysema. COVID-19 also enters target cells via the ACE-2 receptor and theoretically the development of SE and pneumomediastinum could be due to the dysregulation of surfactant production. [4]

The definitive diagnostic tool in diagnosing pneumomediastinum is CT, while a standard x-ray can be used to reveal a pneumomediastinum, if a lateral film isn't ordered then it may be missed. Some of the prominent radiographic features include small amounts of gas that appear as linear

or curvilinear lucencies outlining the mediastinal contours. The gas can be anterior to the pericardium (pneumopericardium), around the pulmonary artery and main branches (ring around the artery sign), outlining the major aortic branches (tubular artery sign), outlining the bronchial wall (double bronchial wall sign), can be continuous with the diaphragm, between the parietal pleura and in the pulmonary ligaments.**[**2**]** In regard to SE, a CT will be able to clearly show the extent of the emphysema usually seen a pockets of air which are dark areas of attenuation.**[**10**]**

Examination will reveal crepitus in the neck and anterior chest.**[**3**]** Since the incidence of SE and SPM are 1.2 and 3.0 per 100,000 respectively the rarity warrants further inquiry in prevention and treatment.**[**4**]** Treatment mainstay is supplement oxygen to reduce the partial pressure of nitrogen in the tissues, thereby hastening the elimination of gas pockets, antitussives, and max bronchodilator regimen to decrease intrinsic auto-peep. Decreasing steroids may lead to increase airways resistance and sustain some decrease of intrinsic auto-peep that may have been at play.

According to the current ANZICS CTG PHAR-LAP trials, in patients with ARDS, it is best to titrate the PEEP and lower the targeted airway pressures.**[**7**]** A risk-benefit decision should be had regarding continuing or stopping steroids. Additionally, there a more invasive options to treat the pneumomediastinum which include: (bilateral) chest drain insertion, vacuum-assisted closure dressings to the supra-clavicular space, needle aspiration, or fenestrated subcutaneous catheters.[10] Ultimately, there is a need to follow up with the patient radiologically and clinically. The development of pneumomediastinum in the setting of COVID-19 may be a negative prognostic factor because of the alveolar damage.

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