Case Series

A Case Series of Spontaneous Secondary Pneumothorax in Post Covid Period: A Clinical Insight

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Abstract

Introduction: We are in the era of novel coronavirus pandemic. Each wave is teaching us something new about the disease pathology, long term prognosis. Nonetheless, COVID-19 is giving rise to new challenges to the clinicians every day with its new found complications, long haul Covid symptoms. Secondary spontaneous pneumothorax has been reported to be a rare complication, seen in 1%-2% of COVID-19 patients with a mean time occurrence of 24.3 days from the hospital admission during the early phase of intubation.

Case discussion: We report a case series of four patients in post COVID period presented with sudden onset respiratory distress and hypoxia, diagnosed with spontaneous secondary pneumothorax. Out of three only one patient received NIV support during his past treatment for COVID-19 and others were treated with oxygen. Therefore, barotrauma secondary to positive pressure ventilation and rupture of cystic bulla cannot be a sole cause of this complication. Persistent chronic inflammatory process and ischemic damage of alveoli are other possible etiologies.

Conclusion: We highlight in our case series the importance of clinical examination, especially chest auscultation, which most clinicians circumvent owing to the trepidation of contracting COVID-19. We also proposed large researches to identify causal association with pneumothorax and previous use of steroids to treat COVID-19, persistent inflammation, age, gender, comorbidity etc to prevent it, as it can be debilitating and fatal. A clinician should always keep pneumothorax as a differential in sudden deteriorating breathlessness and hypoxia in post COVID period as it may happen as late as >40 days from primary COVID diagnosis.

Keywords: Secondary spontaneous pneumothorax; Emergency room; Intercostal chest drain

Introduction

Coronavirus disease 2019 (COVID-19) is defined as illness caused by a novel coronavirus called severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2; formerly called 2019-nCoV), which was first identified amid an outbreak of respiratory illness cases in Wuhan City, Hubei Province, China, World Health Organization (WHO) on December 31, 2019. On January 30, 2020, the WHO declared the COVID-19 outbreak a global health emergency [1].

We are in the midst of a pandemic, one could ever experience in lifetime and almost 1 year 8 months now fighting through it, posing a novel challenge to us every day.

As COVID-19 proved to be quite diverse in presentation, from being asymptomatic to mild, moderate, severe disease [2,3], so as it's complications owing to its potential to affect various systems in

human body. COVID-19 causes wide range of pulmonary, vascular, neurological, cardiac, thromboembolic, abdominal complications [4-7]. Secondary spontaneous pneumothorax is a recognized complication in COVID-19, during diagnosis and treatment. It poses significant threat of morbidity and mortality to the patient. Pneumothorax, which is late in onset and in post Covid period or in patients who doesn't have a previous history of invasive or non invasive ventilation requirement, is rare, reported in 1%-2% of COVID-19 patients with mean time occurrence of 24.3 days from the hospital admission during the early phase of intubation [8,9].

We report three cases of Secondary Spontaneous Pneumothorax (SSP) complicating the post Covid period as late as 27, 33 and 40 days from tested COVID positive. None of them had any history of receiving positive pressure ventilation, either invasive or non invasive during the initial diagnosis and treatment period.

Case Presentation 1

Chief complaints

Twenty seven year old male presented in Emergency Room (ER) with complaints of worsening dry cough and breathlessness over past 3 days.

History of present illness

He was tested positive for COVID-19 by RT-PCR test 28 days back. He was hospitalized outside on day 10 secondary to his requirement of O₂ support. His HRCT chest revealed viral pneumonia involving both lungs. He was managed conservatively with O₂ support via Non Re-

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Breathing Mask (NRBM), IV antibiotics, steroids, oral multivitamins etc., never required for invasive/ non-invasive ventilation. He was discharged on day 17 in a stable condition. During his stay he was also diagnosed with Type2 Diabetes Mellitus (T2DM).

Past history

No known co-morbidity/ addiction/ allergy.

On presentation

- BP: 110/50 mmHg, HR: 147/min, SpO₂: 100% on NRBM, RR: 30/min, Temperature (T): 97.8°F, GRBS: 201 mg/dl, GCS: 15/15
- ABG (on 15 Lt/min O₂): pH=7.388, pCO₂=35.7, pO₂=154, HCO₃=21, Lactate=3.35, Na+=137, K+=3.75
- HRCT Chest: Large right sided pneumothorax (PTX) with collapsed lung and left lung showing changes like subpleural thickening, bronchiectasis, fibrosis etc.
- CTPA: within normal limit (WNL)
- ECG-12 leads: sinus tachycardia, Echocardiography was WNL.
- COVID-19 RT-PCR: negative
- His CBC showed HB 16.8 gm/dl with neutrophilic leukocytosis (N/L ratio >5), WBC 27,000/cumm, his inflammatory markers showed raised CRP (245). D-dimer 0.94, LFT & RFT were WNL.

Management

Intercostal Chest Drain (ICD) was done immediately in right 5th intercostal space. Patient was then started on IV broad spectrum antibiotics, SC Low Molecular Weight Heparin (LMWH), IV steroids, antifibrotic drug nintedanib and other supportive treatment and shifted to ICU. His condition gradually improved and he was shifted to ward on day 3, where he developed sudden onset breathlessness, hypoxia. Bedside CXR-AP view showed right sided PTX recurrence for which right sided ICD was placed again. His pleural fluid showed growth of *Klebsiella pneumoniae* for which his antibiotics were upgraded. He responded to the treatment. His ICD got removed later. He was discharged on day 21 of his admission. Retrospectively we realized that neither the junior resident nor any senior auscultated the chest of the patient owing to fear of contracting COVID-19 pneumonia, leading to failure in early clinical diagnosis of a large PTX (Figure 1).

Case Presentation 2

Chief complaints

60 year old male patient referred from outside hospital with deteriorating Shortness of Breath (SOB).

History of present illness

Patient was tested COVID-19 RT-PCR positive 40 days ago. He was hospitalized outside since last 29 days owing to his deteriorating breathing difficulty, for which he was managed with IV broad spectrum antibiotics, remdesivir, SC LMWH, NIV support along with other supportive treatment. His HRCT revealed COVID-19 pneumonia. He was then referred to our hospital on day 40.

Past history

He is a known diabetic on regular medications. No other

Figure 1: HRCT Chest showing right sided pneumothorax with B/L GGOs, right lung partially collapsed, right lower lobe consolidation.

comorbidity. Not a known smoker. No known allergies.

On presentation

- BP=120/80 mmHg, HR=112/min, RR=33/min, SpO₂=70% with O₂ 15L/min via NRBM, Chest=left sided air entry reduced, CBG=264 mg/dl.

Management

Patient was intubated and put on lung protective ventilatory support secondary to respiratory failure and severe hypoxia, to prevent impending respiratory arrest. Left sided intercostal chest drain inserted as HRCT chest & CT pulmonary angiography suggestive of large PTX on left side, bilateral (B/L) consolidations with Ground Glass Opacities (GGO), bronchiectatic changes, and subpleural thickening with evidence of pulmonary thromboembolism. He was started on broad spectrum antibiotics, IV steroids, SC LMWH, antifibrotic drug and other supportive measures. His repeat COVID-19 RT-PCR came negative. Patient's condition deteriorated further and he was started on vesopressor support. His blood report revealed WBC count of 31,400/cumm with raised N/L ratio (>5), deranged Liver Function Test (LFT) with raised SGOT & SGPT, preserved Kidney Function Test (KFT). Echocardiography was WNL. His family discharged him against medical advice on day 8 of admission in our institute (Figure 2).

Case Presentation 3

Chief complaints

63 year male presented to ER with severe SOB.

History of present illness

Patient was tested positive for COVID-19 rapid antigen on 4th May, 2021. He was hospitalized to other centre next day with complaints of fever only and then transferred to another centre with mild dyspnoea and generalized weakness on 21st May 2021 where he was managed with IV antibiotics & steroids, SC LMWH, nebulisations, oral acetylcysteine (mucolite), O₂ support with simple face mask and other

Figure 2: Large PTX on left side, bilateral (B/L) consolidations with Ground Glass Opacities (GGO), bronchiectatic changes, subpleural thickening.

supportive measures, chest physiotherapy, spirometry etc. For better treatment he was brought to us on 12th June 2021. His outside HRCT chest showing patchy multifocal areas of GGOs and consolidations with interlobar septal thickening involving both lung fields suggestive of atypical pneumonia, likely to be sequelae of COVID-19 [CORADS 6, CT severity score 15/25]. Thick walled cavitary lesion with internal “bird nest pattern” in anterior segment of left upper lung likely to be fungal etiology. Bilateral pleural effusion. D-dimer was very high (1580), preserved LFT and KFT (Figure 3).

Past history

He is a known hypertensive on regular medications. No other comorbidity. Not a known smoker. No known allergies.

On presentation

- BP=110/60mmHg, HR=132/min, SpO₂=93% on 15L/min

Figure 3: Patchy multifocal areas of GGOs and consolidations with interlobar septal thickening involving both lung fields suggestive of atypical pneumonia, likely to be sequelae of COVID-19 [CORADS 6, CT severity score 15/25].

O₂ via NRBM, RR=42/min, CBG=98mg/dl, GCS=15/15, Chest Auscultation= reduced AE to left side significantly with trachea shifted to right.

- ECG-12 leads: sinus tachycardia
- ABG on 2L/min O₂: pH=7.43, pO₂=68, pCO₂=35, HCO₃=23.2, Na+=121, K+=4.8, Lactate=2.5
- Chest X-Ray which was done same day from previous hospital was checked and left sided PTX was seen.

Management

Immediately ICD inserted in left 5th intercostal space. He was started on high flow O₂ support, broad spectrum antibiotics, LMWH, antifibrotic, steroids and other supportive measures. Patient improved gradually and was discharged on 18th June 2021 from our hospital facility (Figure 4).

Figure 4: CXR-AP view showing left sided pneumothorax and B/L lung infiltration.

Case Presentation 4

Chief complaints

A 46 years old female patient presented in the ER with severe & deteriorating SOB.

On presentation

- BP=100/60 mmHg, HR=129/min, RR=41/min, SpO₂=70% with O₂ 15L/min via NRBM, Chest=left sided air entry reduced, CBG=318 mg/dl.
- ECG: sinus tachycardia, ABG: type1 respiratory failure with metabolic acidosis.

History of present illness

Patient was diagnosed with COVID-19 pneumonia in the month of April, 2021. She was initially treated at home with medications. Later she needed to get hospitalized for O₂ requirement. Though she did not require any NIV/Invasive Ventilatory support during her stay and managed conservatively with IV antibiotics, steroids, LMWH, multivitamins and other supportive medications. She was readmitted on 2nd June, 2021 with severe SOB. Her HRCT chest on 2nd June, 2021 showed: Diffuse GGOs & fibrotic strands scattered in both lung fields,

CORADS 6 [CT severity score 25/25] with bilateral PTX {right>left} with soft tissue emphysema with minimal pneumomediastinum. She was managed with right sided ICD, high flow O₂ support via NRBM, IV broad spectrum antibiotics, steroids, antifibrotics, steroid & bronchodilator nebulisation etc.

Repeat HRCT chest on 14th June, 2021: Diffuse GGOs & fibrotic strands scattered in both lung fields, CORADS 6 [CT severity score 25/25], a cavitary lesion in left lower lobe. Echocardiography: LVEF~60% and Within Normal Limits (WNL) (Figure 5).

Patient improved clinically, though with persistent need of O₂ support via nasal cannula. Her HRCT chest on 22nd June 2021 returned to WNL. She was discharged home on O₂ support 4 L/min-5 L/min on 24th June, 2021.

Past history

She is a known diabetic on regular medications. No other comorbidity. Not a known smoker. No known allergies (Figure 6).

Figure 5: HRCT chest on 2nd June, 2021 showed: Diffuse GGOs & fibrotic strands scattered in both lung fields, CORADS 6 [CT severity score 25/25] with bilateral PTX {right>left} with soft tissue emphysema with minimal pneumomediastinum. Repeat HRCT chest on 14th June, 2021: Diffuse GGOs & fibrotic strands scattered in both lung fields, CORADS 6 [CT severity score 25/25], a cavitary lesion in left lower lobe.

Management

Immediately ICD inserted in left 5th intercostal space, following which her breathing difficulty improved. She was started on high flow O₂ support, IV co-amoxiclav, LMWH, antifibrotic, insulin, steroids and other supportive medications. She was shifted to ICU where she gradually developed type2 respiratory failure for which she was

out on NIV support. Secondary to development of bronchopleural fistula and persistent leak, pulmonologist planned for pleurodesis. But unfortunately patient succumbed to complication of COVID-19 disease on 5th day of her current hospitalization (Figure 7).

Figure 6: HRCT chest on 22/06/2021 within normal limits.

Figure 7: CXR-AP view showing B/L diffuse lung infiltrations with left sided ICD in situ.

Discussion

We traversed a long way since novel coronavirus outbreak. In India we have been dealing with second wave now that seems to be settling with an impending threat of third wave. The virus not only took many lives so far, but its lingering post COVID complications, about which we are still in a learning process, are often proved to be morbid, debilitating.

Mild or moderate COVID-19 lasts about two weeks for most people. But others experience lingering health problems even when

they have recovered from the acute phase of the illness. In such patients, there is no longer live coronavirus running amok in the body. If tested, the person would test negative for the coronavirus, but they might be severely debilitated nonetheless.

Approximately 1% of patients admitted with COVID-19 disease develop pneumothorax. This can occur without pre-existing lung disease or mechanical ventilation. Pneumothorax does not seem to be an independent marker of poor prognosis and we encourage active treatment to be continued where clinically possible. Two-thirds of patients survive, but age >70 years and acidosis are associated with poor prognosis [8].

Pneumothorax is defined as air in the pleural cavity and can further classify as:

- Traumatic, including iatrogenic cases.
- Spontaneous:
- Primary Spontaneous Pneumothorax (PSP) - no associated lung disease.
- Secondary Spontaneous Pneumothorax (SSP) - occurs in association with pre-existing lung disease.

Pathophysiology of pneumothorax in covid-19 pneumonia:

Though the exact cause is still unknown, many believe that the main cause of pneumothorax is cystic lesions, caused by barotrauma secondary to invasive or non invasive ventilation or severe coughing, leading to increase in intrathoracic pressure and ultimately alveolar damage. Cyst formation does not restricted to patients receiving positive pressure ventilation, thereby indicating that barotrauma is not the sole cause of PTX in COVID-19 or post COVID period [10]. Cyst formation has also been noticed as a late consequence of ARDS due to SARS, as a result of ischemic parenchymal damage and inflammation [1]. In a case series of six patients with SARS and pneumothorax, Sihoe et al. [13] described administration of methylprednisolone was thought to affect lung healing and the presence of a higher peak serum LDH and peripheral leucocyte count was postulated to depict a greater extent of lung injury, raising the risk of a pneumothorax [12,13].

In our case series we reported four cases of PTX in post COVID period, among patients three were male and one female. Three patients reported to have neutrophilic leukocytosis (N/L ratio>5). Two patients had clinical course of recurrent PTX. One patient developed associated PE & pneumomediastinum. Three of them were diabetic and all of them received steroid therapy in past. Inflammatory markers were not evaluated in all, hence association with PTX as a complication of primary disease, COVID-19 could not be inferred. We need large clinical trials to deduce relationship between PTX in post COVID period and persistent/chronic inflammation, age, gender, comorbidity etc to prevent it on the first place from happening, as PTX can be recurrent, morbid and life threatening.

Conclusion

Our case series of four patients is an aide-memoire to the emergency physicians the importance bedside clinical examination and of considering pneumothorax besides pulmonary thromboembolism and pulmonary fibrosis, as a differential in an acutely deteriorating patient and/or persistent hypoxia in post Covid period. Pulmonary thrombo-embolism related infarction can also result in parenchymal cavitation with subsequent pleural rupture leading to a pneumothorax. One of our patients was in fact diagnosed with pulmonary thrombosis associated with pneumothorax. We also highlight the significance of auscultating the chest as many clinicians avoid auscultation given the concern of exposure to the disease. It can delay diagnosing PTX and further complicate the clinical scenario, can even be life threatening.

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