

Barotrauma Secondary to COVID-19: A Case Series and Literature Review

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Received: January 15, 2024; **Accepted:** February 09, 2024; **Published:** February 17, 2024

Abstract

A frequent consequence in critically ill patients with ARDS resulting from various etiologies is barotrauma, which is estimated to occur in approximately 10% of cases. It is seen in patients with COVID-19 as well, but the underlying mechanism of the barotrauma is different as in other viral pneumonias. We here in present a case series of six patients who had barotrauma lung injury and despite optimal management they didn't survive. The detection of Sars-CoV-2 is via throat/nasal swab PCR testing.

1. Abbreviations

ARDS: Acute respiratory distress syndrome; Bi-Pap: Bilevel positive airway pressure; CORADS: The coronavirus disease 2019 (COVID-19) Reporting and Data System; CT scan: Computed tomography scan; COVID-19: The coronavirus disease 2019; CXR: Chest X-ray; HRCT: High-resolution contrast enhanced CT; IL-1B: Interleukin-1B; NRB: Non rebreather mask; RNA: Ribonucleic acid

2. Introduction

The novel Sars-CoV-2 virus is a positive stranded RNA virus [1] Droplet transmission being the most common source of viral passage from human to human [2]. The incubation period is from 2 to 14 days. The spectrum of symptoms include fever, dry cough, dyspnea, vomiting, diarrhea, myalgia, anosmia & headache. The majority of the patients present with milder symptoms, while few of them progress to pneumonia, ARDS, sepsis with multi organ dysfunction, ARDS & pneumothorax [3-6].

Citation: Jabeen R, Maktoob M, Arif A, et al. Barotrauma Secondary to COVID-19: A Case Series and Literature Review. Clin Case Rep Open Access. 2024;7(1):286.

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The route of entrance of virus into the cells is via Angiotensin Converting Enzyme - (ACE)-Receptor, which is extensively expressed over alveolar cells of lungs. The virus then causes immense host response & tissue damage mainly via interleukin - 6 (IL-6). Another culprit is IL-1B, responsible for inflammation & fibrosis of lungs [7-8]. A frequent consequence in critically ill patients with ARDS resulting from various etiologies is barotrauma, which is estimated to occur in approximately 10% of cases. Generally speaking, it is linked to increased airway pressures [9].

Damage to body tissue resulting from pressure differences in confined cavities within the body is known as barotrauma [10]. Extra alveolar air in places where it should not be present under normal circumstances is known as pulmonary barotrauma. Alveolar rupture, which causes an accumulation of air in extra-alveolar regions, is the most prevalent cause of barotrauma. The consequences of excess alveolar air could then include subcutaneous emphysema, pneumothorax, and pneumomediastinum [11].

Invasive and non-invasive mechanical ventilation techniques, such as bilevel positive airway pressure (BiPap), are examples of mechanical ventilation modalities. Compared to patients getting invasive mechanical ventilation, the incidence of barotrauma in patients undergoing non-invasive mechanical ventilation is significantly lower. Patients with predisposing lung pathologies such as asthma, interstitial lung disease (ILD), chronic obstructive pulmonary disease (COPD), pneumocystis jirovecii pneumonia, and acute respiratory distress syndrome (ARDS) are at high risk of developing barotrauma from mechanical ventilation [12].

3. Case 1

A 60-year-old male known case of hypertension, presented with a 10-day history of fever, and cough, with worsening shortness of breath for 2 days and alteration of mentation for 1 day. The patient never smoked and had no underlying lung disease. HRCT findings were suggestive of CORADS-4. He was requiring high flow Oxygen and maintaining saturation on NRB. Treated for COVID-19 pneumonia severe disease and was given steroids and anticoagulation. Symptoms of the patient worsen on the 15th day of illness, and CXR findings suggest left-sided pneumothorax (FIG. 1). Chest tube placement is done via a thoracotomy approach. His pneumothorax resolved soon after chest tube placement. Unfortunately, following days his symptoms worsened despite optimal treatment and care, and the patient expired.



FIG. 1

4. Case 2

A 45-year-old male, with prior no known co-morbidities presented with 5 days history of fever, cough, and progressive shortness of breath. At presentation, his saturation of oxygen on room air is 78%. He was kept on NRB and maintained saturation on high-flow oxygen. HRCT findings were suggestive of CORADS-6. He started on corticosteroids, remdesivir, and tocilizumab. Despite this, his symptoms got worse on day 9th of illness as he developed subcutaneous emphysema involving the face, neck, and upper chest and later developed bilateral pneumothorax. The patient developed multi-organ failure and died despite providing resuscitative measures.

5. CASE 3

A 40-year-old female, with a known case of diabetes, presented with fever and dry cough for 6 days. The saturation of Oxygen in room air was 85% at the time of presentation. She kept on NRB and maintained saturation on it. HRCT shows CORADS-4. Treated with steroids, remdesivir, and anti-coagulation with enoxaparin. She improved on this management. On the 10th day of illness, her shortness of breath worsens suddenly with an increase in the demand of oxygen. A CXR was done, and findings suggestive of bilateral pneumothorax (FIG. 2). Chest tube placed via a thoracotomy approach. Unfortunately, despite all the possible measures being taken patient died the following day.

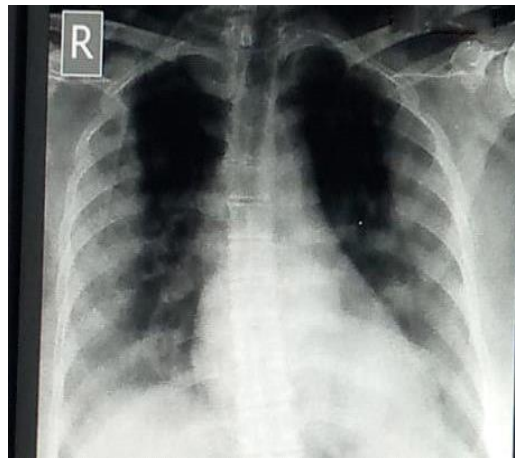


FIG. 2.

6. Case 4

A 42-year-old female, known case of diabetes presented with a 4-day history of fever, dry cough and shortness of breath. HRCT suggestive of CORADS-6. She required High flow Oxygen and kept on NRB. Her treatment includes steroids, remdesivir, tocilizumab and anti-coagulation with low molecular weight heparin. Her symptoms as well as lab parameters worsen along with an increase in oxygen demand. Trials of Bi-Pap given by day 4 of admission. Her shortness of breath worsens by the 9th day of illness, as she develops bilateral pneumothorax evident by CXR (FIG. 3). The chest tube placement done but patient did not survive.

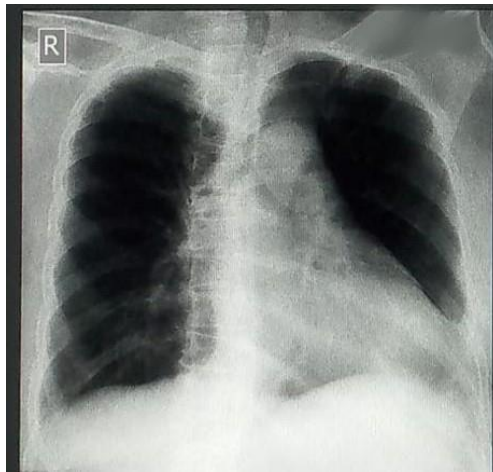


FIG. 3.

7. Case 5.

A 72-year-old gentleman, known case of diabetes presented with severe abdominal pain and mild shortness of breath for 2 days. He is maintaining saturation on low flow oxygen. HRCT findings suggestive of CORADS-6. The treatment included steroids, remdesivir for 5 days and anti-coagulation with subcutaneous low molecular weight heparin. The patient improved on this treatment and was discharged to home after 6 days hospital stay on low flow (2 litres/min) oxygen supply via nasal prongs. After 4 days of discharge, the 10th day of illness, the patient arrived again with worsening shortness of breath and swelling over face and neck.

He had developed sub cutaneous emphysema with bilateral pneumothorax, being favoured by CXR findings. Bilateral chest tube placement done via thoracotomy approach and patient's shortness of breath improved. Unfortunately, the patient collapsed after 3 days of chest tube placement despite possible resuscitative measures.

8. Case 6

A 54-year-old male, with prior no known co-morbidities presented with severe abdominal pain after 15 days history of fever and dry cough. He never smokes in his life and had no underlying lung pathology. His saturation of oxygen at the time of presentation was 89% on room air and maintaining saturation on 2-3 litres of oxygen via face mask. HRCT shows CORADS 4. He was treated with steroids and anti-coagulation with low molecular weight heparin.

On the 19th day of illness, he developed swelling over neck and upper chest with worsening shortness of breath and increase in demand for oxygen. He shifted to NRB. A CXR done which is consistent of bilateral pneumothorax (FIG. 4). Chest tube placement done but patient did not survive and died after 2 days.

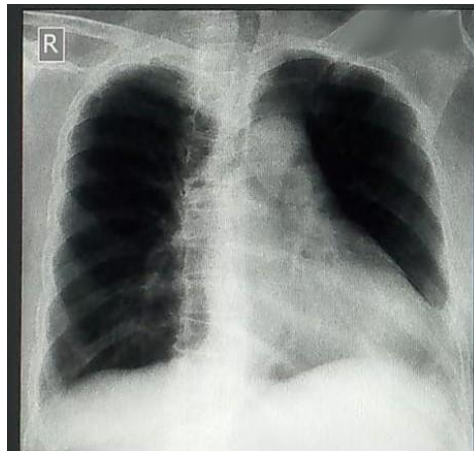


FIG. 4.

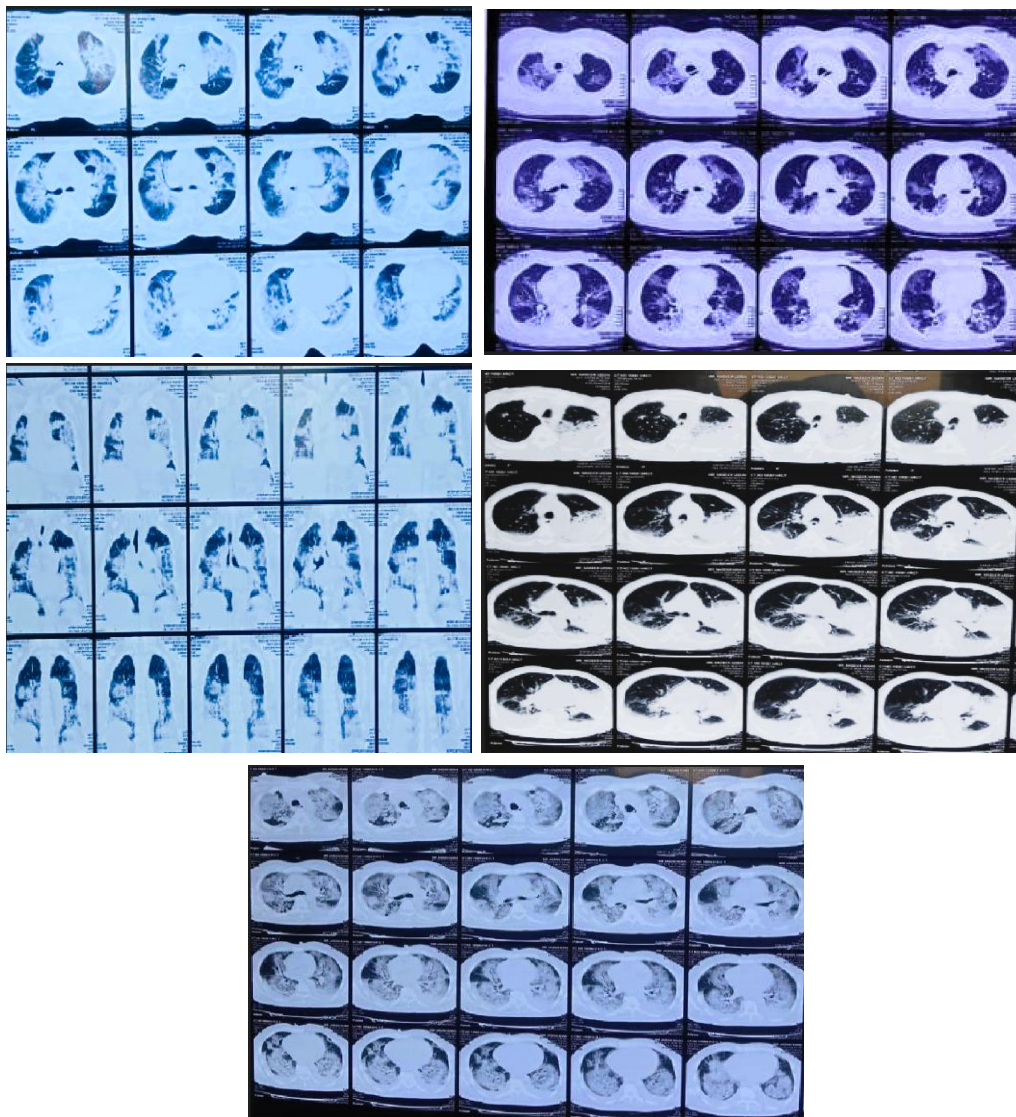


FIG. 5. HRCT- images showing bilateral pneumothorax.

TABLE 1. pneumothorax* gender Crosstabulation.

Count		gender		Total
		Male	Female	
pneumothorax	left sided	1	0	1
	bilateral	3	2	5
Total		4	2	6

TABLE 2. Case Processing Summary.

		Cases					
		Valid		Missing		Total	
		N	Percent	N	Percent	N	Percent
pneumothorax * HRCT		6	100.0%	0	0.0%	6	100.0%

pneumothorax * HRCT Crosstabulation

Count		HRCT		Total
		CORADS4	CORADS6	
pneumothorax	left sided	1	0	1
	bilateral	2	3	5
Total		3	3	6

TABLE 3. **Investigations.**

Investigations	Unit	Normal Range	Case-1	Case-2	Case-3	Case-4	Case-5	Case-6
Hb	mg/dl	(13.5-16.3♂) (11.5-14.5♀)	14.1	15	13.3	11.6	15.3	14.4
TLC	x10	4-11	18.7	5.5	17.1	4.6	10.3	13.8
Platelets	X10 ⁹	150-400	265	150	284	150	571	406
CRP	mg/dl	<5	197.1	111.9	48.7	68.8	86.4	67.2
Ferritin	mg/dl	(12-300♂) (12-150♀)	860	1234	1339	1229	1007	1286
LDH	IU/L	140-280	1224	1336	1089	1557	1292	1297
Pro-BNP	pg/ml	<125	785	1580	1174	941	832	621
D-dimer	mg/l	< 0.50	3.6	2.7	5.12	0.86	0.99	2.11
ALT	IU/L	(29-33♂) (19-25♀)	21	94	44	15	56	63
PT	sec	11-13.5	10.8	14.5	11.7	13.5	8.9	9.2
aPPT	sec	21-35	18	36.5	21.6	31.2	23.6	22.4
Procalcitonin	ng/mL	< 0.1	0.005	3.24	0.025	0.005	0.086	0.024
Trop-I	ng/mL	0 - 0.04	0.1	0.03	0.1	0.001	0.1	0.1

Hb: Hemoglobin, TLC: Total Leucocyte count, CRP: C-Reactive Protein, LDH: Lactate DeHydrogenase, Pro-BNP: Pro B Type Natriuretic Peptide, D-dimer: dimer of fibrin degradation product, ALT: alanine transaminase, PT: Prothrombin time, aPPT: Activated Partial Thromboplastin Time, Trop-I: Troponin I

9. Discussion

The definitions of pneumothorax and pneumomediastinum, respectively, are the existence of free air in the pleural and mediastinal cavities. When air enters the tissues beneath the skin, it causes subcutaneous emphysema. Pneumothorax and pneumomediastinum are recognized adverse events associated with intubation-related mechanical ventilation. Pneumothorax or pneumomediastinum can occur in COVID-19 patients even in the absence of barotrauma [13].

On the other hand, pneumothorax and pneumomediastinum appear to be more prevalent and differ in their clinical features in COVID-19 individuals. Six individuals who experienced pneumothorax and/or pneumomediastinum while residing in the intensive care unit are reported here.

The radiographic changes, consistent with reticular alterations in initial stages which further advances to ground glass opacities. They spread peripherally with middle /lower zones ascendancy. With the passage of time consolidation occurs [14].

As per previous data/studies shows that dyspnea manifest after 5-8 days of symptoms onset our study comprises of six cases, shows that the time duration required from symptom onset to the development of complications like ARDS, pneumothorax & requirement for intubation is 10th day [2-4].

The reported, median time period for the development of the ARDS is eighth day [3]. Characteristic CT- Scan finding suggestive of COVID-19, appeared around this time period as per Pan et al [15].

The remarkable feature we learn through this series of case discussion is that unlike other viral pneumonias, patients suffering from COVID-19 develop pneumothorax soon after initial presentation.

Miro et al conducted a case control study which showed that there is a 3.85-fold chance of COVID-19 associated pneumothorax to be occurred on right side with a frequency of (81.1% vs 52.7%; $P < 0.001$) as compared to non-COVID associated pneumothorax. This same study showed no gender prediction (72.5% vs 51.3%; $P > 0.05$) in development of COVID-19 associated pneumothorax. It is contrary to our observation which shows a male predominance [16].

The culprit behind the development of this barotrauma (pneumothorax, pneumomediastinum) found to be related to imbalance between patient's immune response and marked inflammatory changes cause by COVID-19 infection & unrelated to ventilator induced barotrauma [17-19].

The pathophysiology behind the development of this air injury/barotrauma is diffuse alveolar damage causes weakening of alveolar wall that will lead to dilated cystic & bullous air spaces (pneumatocele) formation within the lung parenchyma.

Scenarios such as coughing lead to sudden rise in intrathoracic pressure, causes rupture of these air pockets, and eventually pneumothorax development. Other mechanism is the escape of air during positive pressure ventilation into the pleural cavity or as per Macklin effect, sailing of air along Broncho vascular sheaths and spreading of this interstitial emphysema into the mediastinum causing pneumomediastinum [20-25].

Pneumomediastinum/subcutaneous emphysema did not correlate with the classic barotrauma mechanism, even with low tidal volume ventilation. Consequently, when barotrauma is ruled out as the cause, COVID-19 appears to be the cause of these manifestations due to lung frailty and the Macklin effect [26].

10. Conclusion

In a patient suffering from Sars-Co-V 2, and during the illness developed an acute decline in the oxygen desaturation or palpation of crepitation over the thorax and neck should trigger the physician for a search for a pneumothorax or pneumomediastinum. Conservative care could be an option if the patient is stable and there is no evidence of progression.

11. Study Limitations

None of the patient kept on invasive ventilation. Autopsy not performed in any of the case.

12. Competing Interest

The authors declared no competing interest.

13. Consent from Patient

Written informed consent for patient information and images to be published was provided by the patient.

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