Case Report

A rare case of spontaneous combined hydropneumothorax in a case of severe COVID pneumonia

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ABSTRACT

The combined spontaneous hydropneumothorax in a healthy young female of COVID-19 is relatively rare in the absence of ventilator-induced barotrauma and associated with typical COVID-related coagulopathy and inflammation. Here, we present the case of a 32-year-old COVID-19-positive female who developed sudden breathlessness and tachycardia. On clinical evaluation and imaging studies, she was found to have hydropneumothorax along with high D-dimer levels. Although the exact cause for this complication is not known, the COVID-19-related coagulopathy and inflammatory response are the likely cause. Monitoring D-dimer levels and early intervention by putting intercostal drain can be life-saving.

Keywords: COVID-19, Coagulopathy, Hydropneumothorax

During the crisis of COVID-19, many cases were reported to have pleural effusion and spontaneous pneumothorax. Coronavirus is known to cause multiorgan involvement starting from its initial breakdown during 2020 but during the time of the second wave, the particular strains (B.1.617.1, B.1.617.2, and B.1.617.3 because of two spike mutations L452R and E484Q), identified by the International Council of Medical Research (ICMR), were causing a rampage in South India, namely, Maharashtra, Andhra Pradesh, Telangana, Uttar Pradesh, Madhya Pradesh, Tamil Nadu, and Kerala as they have a high predilection to younger patients between 30 and 50 years of age, with a higher rate of complications [1].

Here, we present the case of spontaneous combined hydropneumothorax in a 32-year-old COVID-19-positive female. The combined spontaneous hydropneumothorax in a healthy young female of COVID is relatively rare and due to its unnatural presentation with typical COVID-related coagulopathy and inflammation, the patient was managed in step-wise escalating manner.

CASE REPORT

A 32-year-old female, with no known comorbidities, presented with chief complaints of fever and symptoms of coryza for 3 days and was found positive for COVID-19 by reverse transcription-polymerase chain reaction (RT-PCR) test on April 14, 2021. She was managed at home conservatively with tab. azithromycin 500 mg OD, tab. paracetamol 500 mg BD, and tab. ivermectin 15 mg BD. After 6 days, she presented to the civil hospital with complaints of shortness of breath and generalized weakness. As the patient was not improving clinically in spite of oxygen support, the patient was brought to this hospital (how much oxygen was given and for how long were not mentioned in the discharge summary of the private hospital).

On admission, the patient had a pulse rate of 96 per min, blood pressure of 108/8 mm of Hg, with SPO_2 maintained at 94% on room air. The vitals of the patient from the day of admission till discharge are mentioned in Table 1. The initial X-ray chest of the patient was marked with prominent bronchovascular markings with small peripheral haziness and elevated C-reactive protein (CRP) and D-dimer level (Table 2).

The patient was started on empirical IV antibiotics with inj. piperacillin tazobactam 4.5 gm 8th hourly and inj. Levoflox 500 mg OD, and inj. low-molecular-weight heparin 60 mg SC bd, continued to maintain oxygen saturations till 2 weeks post-admission, with the increased respiratory rate being only clinical sign which was alarming.

After 2 weeks post-admission, the patient was shifted to a non-rebreathing mask as her respiratory rate was high but other vital parameters were normal. The patient was continued with the

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Table 1: Vitals of the patient from day of admission to post-ICD insertion

Day 1	4 days	7 days	9 days	12 days	15 days	18 days	1 month
PR: 96/min	PR: 102/min	PR: 116/min	PR: 120/min	PR: 110/min	PR: 132/min	PR: 96/min	PR: 78/min
BP: 108/84	BP: 108/84	BP: 128/82	BP: 118/80	BP: 132/84	BP: 96/84	BP: 118/82	BP: 108/84
SPO2: 94%	SPO2: 89–94%	SPO2: 88–94%	SPO2: 84–94%	SPO2: 88–94%	SPO2: 80–94%	SPO2: 94%	SPO2: 94%
RR: 18/min	RR: 20/min	RR: 24/min	RR: 22/min	RR: 26/min	RR: 32→18/min	RR: 18/min	RR: 16/min

PR: Pulse rate; BP: Blood pressure; RR: Respiratory rate

Table 2: Investigation reports from admission to discharge

Tests	Admission	Post-admission (4days)	Post-admission (8 days)	Post-admission (14 days)	Post-admission (21 days)	Post-admission (25 days)	Post-admission (29 days)	Post-admission (1 month 4 days)
TLC	16,900 c/cum	9800/cum	10,000 c/cum	12,486/cum	11,362 c/cum	29,100 c/cum	18,200 cells/cum	8000 cell/cumm
NLR	4.09	3.07	1.94	2.22	2.92	7.33	4.00	1.92
CRP	53.5 mg/dl	89.6 mg/dl	53.5 mg/dl	53.5 mg/dl	31.1 mg/dl	68.4 mg/dl	46.2 mg/dl	10 mg/dl
D-dimer	612 ng/ml	612 ng/ml	612 ng/ml	612 ng/m	4191 ng/ml	7848 ng/ml	4000 ng/ml	2200 ng/ml
LDH	620 iu/l	612 iu/l	420 iu/l	360 iu/l	466 iu/l	586 iu/l	230 iu/l	320 iu/l

TLC: Total leukocyte count; NLR: Neutrophil-to-lymphocyte ratio; CRP: C-reactive protein; LDH: Lactate dehydrogenase

above treatment with additional inj. methylprednisolone 62.5 mg IV once daily, and was planned to wean off the oxygen support but in the 3rd week, she developed more dyspnea with existing oxygen support. Therefore, oxygen was increased from the previous 5 L per min to 15 L per min. The chest X-ray suggested the development of combined hydropneumothorax on the right side, for which intercostal drain (ICD) was placed on the right side (Fig. 1). This was followed by repositioning of intercostal drain from posteroapical space to posteroinferior space due to prolonged non-resolution and blunting of cardiophrenic angle and daily output from the drain (Fig. 2). A thorough evaluation of pleural fluid was done to rule out any previously existing pulmonary disease.

Patients pleural fluid analysis showed following biochemical parameters: Volume – 30 ml, color – hemorrhagic, protein – 3.5 gm/dl, glucose – 64 mg/dl, LDH – 256 iu/L, total cell count – 3060/cu.mm, and predominant cells – neutrophils. No acid-fast bacilli (AFB) were seen on Ziehl–Neelsen staining. Gram staining showed no microorganisms. Cytological smears showed predominantly neutrophils and lymphocytes in a hemorrhagic background. No atypical cells were seen.

The patient was followed up after 3 months with the continuation of tab. rivaroxaban 10 mg OD for 3 months, from the date of discharge with monthly CRP and D-dimer values. At the end of the 3 months, an X-ray chest was taken and shifted to tab. Ecosprin 75 mg OD for the next 3 months (Fig. 3).

DISCUSSION

COVID-19 infection is associated with a severe pro-inflammatory state leading to coagulopathy caused by the elevation of fibrinogen and D-dimer degradation products. Secondary complications such as venous and arterial thromboembolic events and microvascular events in pulmonary vascular beds are documented. COVID-19 virus causes lung inflammation progressing to cytokine storm in severe cases. Studies have quoted that the COVID-19 virus does not appear to have intrinsic procoagulant effects itself rather coagulopathy is most likely due to profound inflammatory response and endothelial activation/damage seen in SARS-CoV-2 infection [1].

The hallmark findings of COVID-19 include bilateral patchy ground-glass opacities in peripheral distribution. The most common pleural change in COVID-19 patients is pleural thickening while pleural effusion is extremely uncommon. Wong *et al*. in their study in 2019 found that only two patients had pleural effusion out of 64 patients who had COVID pneumonia, while it is of variable frequency in the study of Ye *et al*. Until this time, there was no clear evidence of what causes pleural effusion, but it carries adverse prognostic sign that may indicate a bacterial superinfection in COVID-19 pneumonia. Elevated pleural fluid LDH levels (greater than 1000 IU/L) suggest empyema, malignant effusion, rheumatoid effusion, or pleural paragonimiasis [2,3]. However, the relevant tests in our case do not support any of the above differential diagnoses.

Primary spontaneous pneumothorax (PSP), however, by definition, occurs in patients with no associated lung disease. Indeed, a finding of abnormal pleura, however, is very common even in PSP patients if looked for carefully and includes blebs and bullae, which are otherwise known as emphysema-like changes. While causality cannot be established exactly what causes pneumothorax in COVID-19 patients who are presently affected and post-recovery within 3 months is difficult, there are case series published supporting the evidence between COVID-19 and pneumothorax, adding the weight to our case report to support the association of pneumothorax in severe COVID-19 rather than being merely coincidental. Explaining the relationship between pneumothorax and COVID-19 is challenging with multiple possible mechanisms [4-6].

Existing pneumatoceles and blebs in a patient who has been on positive pressure ventilation might develop secondary

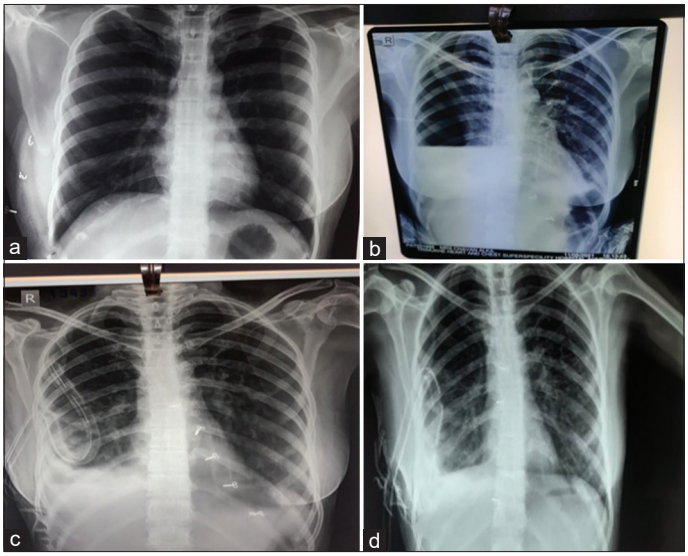

Figure 1: X-ray (a) 1 week before admission; (b) day 15; (c) day 16–25; (d) day 26–40 of admission

Figure 2: One hundred and fifty milliliters on the day of insertion, rest 400 ml in the next 28 days

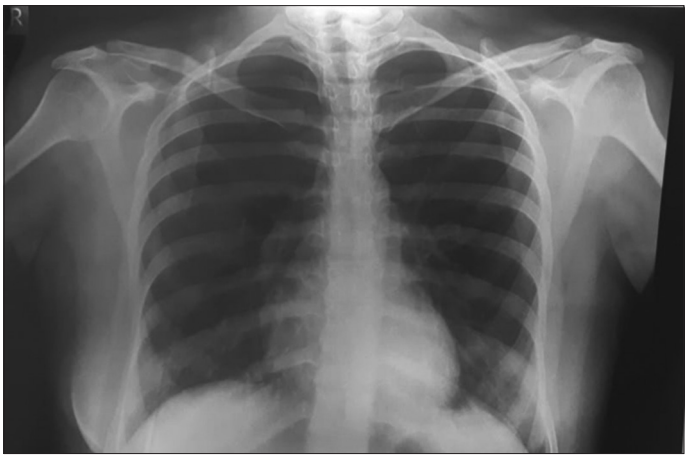

Figure 3: X-ray at the end of 3 months

pneumothorax due to rupture of bullae or cyst. Other possible causative factors for pneumothorax in COVID-19 may be persistent coughing resulting in increased intrathoracic pressure in the presence of underlying pleural abnormalities or alveolar damage from COVID-19 pneumonia-related inflammation or ischemic parenchymal damage [7-9]. Pulmonary embolism-related infarction can also result in parenchymal cavitation with subsequent pleural rupture leading to a pneumothorax, therefore, computed tomography (CT) pulmonary angiography was also done to rule out existing pulmonary thromboembolism.

Monitoring of D-dimer levels as a marker of hypercoagulability is considered standard for treatment of COVID-19 as D-dimer levels are first to increase followed by an increase in prothrombin time and international normalized ratio (PT/INR) and thrombocytopenia progressing toward disseminated intravascular coagulation (DIC) (10). In this case, the patient initially had mildly elevated D-dimer which suddenly increased to 8 times after 2 weeks and 16 times during the 3rd week with the start of symptoms, during which the patient developed this rare complication of hydropneumothorax on the right side. High D-dimer levels persisted for the next 6–8 weeks. The patient was managed with intercostal seal drainage which was removed after 4 weeks when the patient had minimal fluid in pleural space and considerable lung expansion. This was also correlating with decreasing levels of D-dimer levels.

CONCLUSION

Hydropneumothorax in a young COVID-19 female patient is relatively rare, especially in the absence of invasive or non-invasive ventilation. In the absence of comorbidities and previously documented lung pathology, combined hydropneumothorax probably developed due to systemic inflammatory response along with coagulopathy causing damage to the pleural membrane leading to air leak associated with exudative collection and development of combined hydropneumothorax, which cannot be refuted. Management of such patients as per standard guidelines, timely placement of ICD, and monitoring D-dimer levels can prove to be life-saving in these cases.

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