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# A rare case series of bilateral pneumothorax among adult COVID-19 patients reported at a tertiary care hospital in Mizoram, India

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## ABSTRACT

Apart from routine symptoms such as fever, cough, sore throat, myalgia, and dyspnea in severe form of Coronavirus Disease-2019 (COVID-19) infection, very rarely patients can develop worsening of dyspnea due to bilateral pneumothorax. The present case series is about five adult patients, of age ranging from 39 to 57 years, who developed bilateral pneumothorax during their stay in the hospital. All the cases were reported between May 2021 and October 2021 and were tested positive for COVID-19 by reverse transcriptase polymerase chain reaction. Out of five adults, three patients were males and were two females. All the patients were assessed with quick sequential organ failure assessment (q SOFA) score on admission and then monitored by SOFA Score. On admission, baseline contrast enhanced computer tomography chest was done for three patients, and chest radiography for one patient all showing features of moderate to severe COVID-19 pneumoniae. One patient with q SOFA Score of 3 on admission required immediate invasive mechanical ventilatory support with ultrasonogram chest immediately performed showing bilateral pneumothorax. Patients were started on remdesivir, dexamethasone, low molecular weight heparin or unfractionated heparin, tocilizumab, and antibiotics. Subsequently, during the course of stay in the hospital, rest of the four patients developed symptoms of pneumothorax and emergency bedside chest ultrasonography showed the typical barcode or stratosphere sign confirming bilateral pneumothorax. All the patients were managed with bilateral chest intercostal water seal drainage intercostal drain tube and invasive mechanical ventilation. Fraction of Inspired Oxygen ($FIO_2$), and other ventilatory settings were adjusted depending on daily arterial blood gas findings. Attempts to wean off from ventilatory support and extubation were successful for two patients, whereas three patients did not survive. In this case series, we will be presenting about those five cases of bilateral pneumothorax in COVID-19 patients reported at a tertiary care hospital in Mizoram, India.

**Key words:** Pneumothoraces; SARS Co-V2; Atypical Presentations; q SOFA Score; Ultrasonography

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## INTRODUCTION

A pneumothorax occurs when air leaks into the space between the lung and chest wall. This air pushes on the outside of the lung and makes it collapse. A pneumothorax can be a complete lung collapse or a collapse of only a portion of the lung.[1] There are different types of Pneumothorax such as Primary Spontaneous Pneumothorax, Secondary Spontaneous Pneumothorax, Iatrogenic, and Traumatic Pneumothorax. Sometimes no reasons can be found for the occurrence of Pneumothorax. In general smoking, being thin, tall and male are at risk of developing pneumothorax.[2] Other risk factors for developing pneumothorax are pre-existing lung diseases

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such as chronic obstructive pulmonary disease (COPD), cystic fibrosis, lung cancer, lung tuberculosis, pneumoniae, and HIV associated pneumocystis pneumoniae. Chest trauma can also result in pneumothorax. Small air blisters (blebs) can develop on the top of the lungs. When these blebs burst, it can cause pneumothorax. Mechanical ventilation itself may cause pneumothorax because of the ventilator creating an imbalance of air pressure within the chest and the lung may collapse eventually.[1,3-6] The Pneumothorax in relation to COVID19 disease in India is very less reported. Here, in this case series, we will be describing five cases of bilateral pneumothorax that occurred in an adult population diagnosed to have COVID 19 pneumoniae by ultrasonography (US) of the chest.

## CASE PRESENTATIONS

### Case 1

Case 1 was a 39-year-old male, SARS- CoV-2 positive patient referred from a COVID care center (CCC) on May 5, 2021. Patient had no comorbidity, non-smoker, non-obese and not a known case of pre-existing lung disease. He was admitted directly to intensive care unit (ICU) after being clinically diagnosed as Severe COVID -19, with admission vitals of heart rate (HR) 115/min, blood pressure (BP) 114/82 mm of Hg, respiratory rate (RR) 28–30/min, and $SpO_2$ 73% on room air (RA) with quick sequential organ failure assessment (q SOFA) score of 1. Significant baseline investigations were elevated C reactive proteins (CRP) and D-Dimer of 1:16 and 921 ng/mL, respectively. Except for mild respiratory alkalosis with $PaO_2$ of 70 mm of Hg and Ph of 4.746, other baseline arterial blood gas (ABG) reading was relatively normal. Baseline contrast enhanced computer tomography (CECT) thorax showed features of Severe COVID -19 pneumonia. He was given oxygen support with Non-Rebreather (NRB) mask at 15 L/min immediately, but due to non-compliance by the patient, $O_2$ via high flow nasal cannula (HFNC) starting at 30 L/min and later flow increased up to 60 L/min was attempted. COVID -19 protocol for severe COVID-19 was started which included remdesivir, low molecular weight heparin (LMWH), and dexamethasone. Antibiotic coverage was given with intravenous (IV) broad spectrum antibiotic. Other supportive treatments as required were given. Since his condition showed no signs of improvement following 3 days of intermittent oxygen support with NRB mask and HFNC, the patient was put-on non-invasive ventilation (NIV) with $FiO_2$ of 1. With no signs of improvement clinically and worsening hypoxemia on ABG with P/F ratio remaining at <70, the patient was electively intubated on the 7$^{th}$ day of NIV support. Ventilatory setting was at first attempted with volume control (VC) mode with tidal volume of not more than 6 ml/kg, but due to persistent hypoxia even with $FiO_2$ of 1, mode was changed to pressure control mode with a baseline positive end expiratory pressure (PEEP) of 7 cm of water. The following day, patient developed subcutaneous emphysema on the neck, upper part of chest, and upper limbs. Although no change was observed in $SpO_2$, auscultation of chest showed bilateral decreased air entry with bilateral crackles. Bedside chest ultrasonogram (USG) was immediately done which showed bilateral pneumothorax, and bilateral intercostal drain (ICD) with water seal was inserted under local anesthesia (LA). $FiO_2$ and other ventilatory settings were adjusted depending on daily ABG findings, and the patient was weaned off from ventilatory support and successfully extubated on the 9$^{th}$ day following intubation. Chest drain was removed 4 days after extubation and patient was shifted to ward with $O_2$ requirement of 2 L/min by nasal cannula the following day. Patient was discharged following negative reverse transcriptase polymerase chain reaction (RT-PCR) test from the hospital on June 4, 2021. The Hemogram, D- Dimer, CRP values, coagulation profile, and ABG reports of case 1 are shown in Table 1.

### Case 2

Case-2 was a 48 year old, SARS-CoV-2 positive female patient referred to our hospital on July 7, 2021, from CCC. She was referred to Zoram Medical College due to fall in $SpO_2$ below 80% on RA. She was a known case of Type 2 Diabetes Mellitus (DM) on insulin and hypertension on telmisartan (40 mg once daily) with a body mass index (BMI) of 32. She was directly admitted to ICU with baseline hemodynamics of BP-169/100 mm of Hg, HR-115/min, RR of up to 30/min, and $SpO_2$ 76% on RA with a qSOFA score of 1. Oxygen supplementation was started with a NRB mask with oxygen ($O_2$) at 15 L/min, but due to persistent low $SpO_2$ below 90% she was put on NIV support with $FiO_2$ of 1. Significant results on baseline investigation were blood sugar: 342 mg/dl and D-dimer 4997 ng/mL. ABG was relatively normal on admission. Baseline CECT on the day of admission showed features of severe COVID-19 pneumonia. Supportive measures for control of blood sugar, hypertension, broad spectrum antibiotic, and protocol for severe COVID-19 were started which included remdesivir, LMWH, and steroid. Tocilizumab 400 mg was also given IV. Due to worsening hypoxemia with P/F ratio of <60 as indicated by ABG analysis, she was placed on IMV support on the 4$^{th}$ day of NIV at PC mode with baseline PEEP of 7. The next day after invasive mechanical ventilation, the patient suddenly deteriorated with increase in HR from <110 to more than 140/min with acute drop in $SpO_2$ below 50%. On chest auscultation, there were decreased breath sounds bilaterally. Bedside chest USG was immediately done which showed bilateral pneumothorax. Bilateral ICD with water seal was inserted under LA. Regrettably, the patient's condition

declined during the course of treatment and she expired on July 30, 2021. The hemogram, D- Dimer, CRP values, coagulation profile, and ABG reports of case 2 are shown in Table 2.

### Case 3

Case 3 was a male, aged 57 years who was tested positive for COVID-19 on July 4, 2021. He presented with signs of moderate COVID-19 pneumonia when admitted on

**Table 1: The hemogram, D-dimer, CRP values, coagulation profile, and arterial blood gas of case 1**

| Hemogram | Result | | |
|---|---|---|---|
| | Baseline (on ICU admission) | Last recorded values | Reference range |
| Hemoglobin | 10.5 g/dL | 9.8 g/dL | 12–16 g/dl g/dL |
| Total WBC count | 12,000/cumm | 29,000/cumm | 4–11,000/cumm |
| DLC | | | |
| Polymorphs | 86% | 93% | 40–80% |
| Lymphocytes | 11% | 2% | 20–40% |
| Eosinophils | 01% | 2% | 1–6% |
| Monocytes | 02% | 3% | 2–10% |
| Platelet count | 2.1 lacs/cumm | 2.2 lacs/cumm | 1.5–4.05 lacs/cumm |
| Erythrocyte sedimentation rate | 45 mm/1 h | 50 mm/1 h | <20 mm/1 h |
| D-dimer | 921 ng/mL | 713 ng/mL | <500 ng/mL |
| CRP | 1:16 | 1:2 | |
| Coagulation profile | | | |
| PT | | | |
| Control | 12.4 s | 13.8 s | H: 10–12 s |
| Test | 13.8 s | 15.2 s | R: 11–16 s |
| INR | 1.0 | 0.8 | 0.89 |
| Arterial blood gas analysis report | | | |
| pH | 7.476 | 7.495 | 7.35–7.45 |
| $PaCO_2$ | 34.4 mmHg | 23.9 mmHg | 35–45 mmHg |
| $PaO_2$ | 70 mmHg | 63.6 mmHg | 80–100 mmHg |
| $HCO_3$ | 25.3 mmol/L | 18.5 mmol/L | 22–26 mmol/L |
| $SaO_2$ | 95.1% | 83.9% | 95–100% |

WBC: White blood cell, PT: Prothrombin time, CRP: C- reactive protein, pH: potential of hydrogen, $PaCO_2$: Partial pressure of carbon dioxide, $PaO_2$: Partial pressure of oxygen, $HCO_3$: Bicarbonates, $SaO_2$: Oxygen saturation, DLC: Differential leukocyte count

**Table 2: Hemogram, d-dimer, CRP values, coagulation profile, and arterial blood gas of case 2**

| Hemogram | Result | | |
|---|---|---|---|
| | Baseline (on icu admission) | Last recorded values | Reference range |
| Hemoglobin | 13.3 g/dl | 11.7 g/dl | 12–16 g/dl g/dl |
| Total WBC count | 12,300/cumm | 15,700/cumm | 4–11,000/cumm |
| DLC | | | |
| Polymorphs | 89% | 88% | 40–80% |
| Lymphocytes | 08% | 10% | 20–40% |
| Eosinophils | 01% | 02% | 1–6% |
| Monocytes | 02% | 01% | 2–10% |
| Platelet count | 3.2 lacs/cumm | 5.2 lacs/cumm | 1.5-4.05 lacs/cumm |
| ESR | 30 mm/1h | 46 mm/1 h | <20 mm/1 h |
| Abo and RH typing | "A" positive | | |
| D-dimer | 4997 ng/ml | 3419.9 ng/ml | <500 ng/ml |
| CRP | Negative | 1:8 | |
| Coagulation profile | | | |
| PT | | | |
| Control | 12.5 s | 15.1 | H: 10–12 s |
| Test | 14.5 s | 13.9 | R: 11–16 s |
| INR | 1.06 | 1.11 | 0.89 |
| Arterial blood gas analysis report | | | |
| PH | 7.404 | 7.385 | 7.35–7.45 |
| $PaCO_2$ | 35.4 mmhg | 50.6 mmhg | 35–45 mmhg |
| $PaO_2$ | 93.6 mmhg | 58.2 mmhg | 80–100 mmhg |
| $HCO_3$ | 22.2 mmol/l | 29.1 mmol/l | 22–26 mmol/l |
| $SaO_2$ | 97.4% | 80.% | 95–100% |

WBC: White blood cell, PT: Prothrombin time, ESR: Erythrocyte sedimentation rate, CRP: C- reactive protein, pH: Potential of hydrogen, $PaCO_2$: Partial pressure of carbon dioxide, $PaO_2$: Partial pressure of oxygen, $HCO_3$: Bicarbonates, $SaO_2$: Oxygen saturation, DLC: Differential leukocyte count

July 25, 2021 at our hospital. He was a non-smoker with no other co-morbidity except for obesity with a BMI of 35. Quick SOFA score on admission was 1. Baseline CECT chest on admission showed ground glass opacities graded as moderate COVID-19 pneumonia. He was first admitted in the ward, but due to worsening dyspnea with persistent low $SpO_2$ even with $O_2$ supplement at 15/min by NRB, patient was transferred to ICU on the 3rd day of admission. Chest radiography (CXR) was done on the day of transfer which now showed features of severe COVID-19 pneumonia. The patient was put on NIV support with $FiO_2$ of 1. All investigations were repeated on admission into ICU indicating severe sepsis. Treatment including broad spectrum antibiotic coverage, antiviral with remdesivir, steroid, and LMWH which were already initiated in the ward was continued. The patient had persistent high-grade fever with temperature of up to 103°F not controlled with antipyretics indicating severe inflammatory storm which was supported by rise in CRP of 1:18 and D-dimer value of 4112 ng/mL by 2nd day of ICU admission; hence, Tocilizumab 400 mg was also included in the treatment. Regular monitoring and control of blood sugar was required since it was on the higher side from the day of admission. By day 5 of NIV, there was no worsening of condition clinically but daily ABG analysis showed no improvement with $FiO_2$ requirement remaining at 1. Counseling was given by ICU team for the need of elective IMV by day 5 of NIV. But intubation and invasive ventilation was delayed due to refusal by the patient and other family members. On day 12 of NIV support, the patient suddenly deteriorated with worsening of dyspnea, tachypnea, and tachycardia with HR more than 150/min and fall in $SpO_2$ below 65% with bilateral decreased air entry on chest auscultation. The patient was immediately intubated and put on mechanical ventilatory support. Emergency bedside US confirmed bilateral pneumothorax and bilateral ICD with water seal was inserted. There was intermittent drop in $SpO_2$ in spite of these measures, with fall in BP even with ionotropic support. 6 h after IMV support and ICD insertion, the patient suffered a cardiac arrest, resuscitative attempts failed and the patient expired on August 5, 2021. The hemogram, D- Dimer, CRP values, coagulation profile, and ABG reports of case 3 are shown in Table 3.

## Case 4

Case 4 was a 57-year-old female patient who was referred from a private hospital on July 20, 2021, with RT-PCR positive test for SARS-CoV-2. She was a known case of type 2 DM, with peripheral neuropathy and gout. Initial presentation on admission was altered sensorium, tachypnea with RR of up to 45/min, $SpO_2$ of 65% on RA, rapid feeble pulse of 138/min and hypotension with BP of 80/56 mm of Hg with a qSOFA score of 3. She was directly transferred to ICU where she was immediately placed on IMV support. Chest USG was urgently done which

**Table 3: Hemogram, d-dimer, CRP values, coagulation profile, and arterial blood gas of case 3**

| Hemogram | Result | | | |
|---|---|---|---|---|
| | Baseline (on admission in ward) | 1st day of ICU admission | 2nd day of ICU admission | Reference range |
| Hemoglobin | 12.1 g/dl | 14.2 g/dl | 14.0 g/dl | 12–16 g/dl g/dl |
| Total WBC count | 12,100/cumm | 43400/cumm | 48000/cumm | 4–11,000/cumm |
| DLC | | | | |
| Polymorphs | 91% | 92% | 92 | 40–80% |
| Lymphocytes | 04% | 01% | 02% | 20–40% |
| Eosinophils | 02% | 02% | 02 | 1–6% |
| Monocytes | 03% | 05% | 05 | 2–10% |
| Platelet count | 2.5 lacs/cumm | 2.9 lacs/cumm | 3.0 lacs/cumm | 1.5–4.05 lacs/cumm |
| ESR | 30 mm/1h | 50 mm/1 h | 65 mm/h | <20 mm/1 h |
| ABO and RH typing | "O" positive | | | |
| D-dimer | 956.7 ng/ml | 1182 ng/ml | 4112 ng/ml | <500 ng/ml |
| CRP | Positive (1:16) | 1:16 | 1:18 | |
| Coagulation profile | | | | |
| PT | | | | |
| Control | 12.5 s | 12.8 | 12.58s | H: 10–12 s |
| Test | 13.5 s | 15.0 | 15.0 s | R: 11–16 s |
| INR | 0.98 | 1.0 | 1.0 | 0.89 |
| Arterial blood gas analysis | | | | |
| PH | 7.4 | 7.44 | 7.35-7.45 | |
| $PaCO_2$ | 38.4 mmhg | 34.7 mmhg | 35–45 mmhg | |
| $PaO_2$ | 40.2 mmhg | 62.4 mmhg | 80–100 mmhg | |
| $HCO_3$ | 24.4 mmol/l | 23.8 mmol/l | 22–26 mmol/l | |
| $SaO_2$ | 76.2% | 92% | 95–100% | |

PT: Prothrombin time, ESR: Erythrocyte sedimentation rate, CRP: C- reactive protein, pH: potential of hydrogen, $PaCO_2$: Partial pressure of carbon dioxide, $PaO_2$: Partial pressure of oxygen, $HCO_3$: Bicarbonates, $SaO_2$: Oxygen saturation, DLC: Differential leukocyte count

revealed bilateral pneumothorax and immediate placement of bilateral ICD done. ABG showed severe metabolic acidosis correction of which was also done. Hemodynamic improvement was seen following ICD insertion without the need of vasopressors. Baseline investigation also showed hyperglycemia, AKI and dyselectrolytemia with hyponatremia (120 meq/L) and hyperkalemia (6 meq/L), elevated D-dimer and CRP of 1412 ng/mL and 1:16, respectively. Supportive measures were given with broad spectrum antibiotics, unfractionated heparin, steroid, PPI, and control of blood sugar done with insulin. However, the patient deteriorated gradually irrespective of all supportive monitoring and management, and unfortunately expired on August 3, 2021. The hemogram, D- Dimer, CRP values, coagulation profile, and ABG reports of case 4 are shown in Table 4.

### Case 5

Case 5 was a 43-year-old male referred on October 6, 2021, who was RAGT positive for SARS-CoV2 on the day of referral with complaint of shortness of breath and cough for 1 day, fever for 2 days and abdominal distension for 3 days. He was a non-smoker and gave no history of co-morbidity. His BP was 134/94 mm Hg, RR 42/min, and HR 112/min on the day of admission in ICU. He was directly admitted into ICU with qSOFA score of 1. Baseline CXR showed features of severe Covid-19 pneumonia. X-ray erect abdomen was not significant except for distended bowel loops. Since hypoxia was not corrected with 15 L/min of $O_2$ supplement by NRB, $O_2$ support was continued with NIV. Other baseline investigations were grossly normal except for elevated CRP of 1:6 and serum creatinine of 1.5 mg/dL. Abdominal distension improved with conservative management by nasogastric tube. However, there was acute worsening of dyspnea and drop in $SpO_2$ below 60% on the 3rd day of NIV. Chest USG confirmed bilateral pneumothorax and ICD with water seal was immediately inserted. There was immediate raise in $SpO_2$ of up to 82% following placement of chest drain. However, the patient could not be weaned off from NIV, and there was no improvement in $SpO_2$ beyond 86%. Daily ABG also showed worsening P/F ratio and ultimately after obtaining consent, endotracheal intubation was performed on the 3rd day of chest tube placement and put on IMV support with VC mode of tidal volume not more than 6 ml/kg. The patient showed daily improvement clinically and with no deterioration in laboratory investigations, successfully weaned off from IMV by the 10th day of endotracheal intubation. ICD was removed with no further complication and patient was discharged from hospital on October 29, 2021. The hemogram, D- Dimer, CRP values, coagulation profile, and ABG reports of case 5 are shown in Table 5.

Table 6 shows the q SOFA Score of all the five patients on admission in ICU.

Table 7 shows the sequential organ failure assessment scores of all the patients.

**Table 4: Hemogram, d-dimer, CRP values, coagulation profile, and ABG for case 4**

| Hemogram | Result | | |
|---|---|---|---|
| | Baseline (on ICU admission) | Last recorded values | Reference range |
| Hemoglobin | 10.8 g/dl | 9.5 g/dl | 12–16 g/dl g/dl |
| Total WBC count | 12,000/cumm | 18,000/cumm | 4–11,000/cumm |
| DLC | | | |
| Polymorphs | 91% | 92% | 40–80% |
| Lymphocytes | 07% | 05% | 20–40% |
| Eosinophils | 01% | 01% | 1–6% |
| Monocytes | 01% | 02% | 2–10% |
| Platelet count | 1.0 lacs/cumm | lacs/cumm | 1.5–4.05 lacs/cumm |
| ESR | 30 mm/1hr | 130 mm/1hr | <20 mm/1 h |
| Abo and RH typing | "A" positive | | |
| D-dimer | 1412 ng/ml | 1681 ng/ml | <500 ng/ml |
| CRP | Positive1:16 | 1:4 | |
| Coagulation profile | | | |
| PT | | | |
| Control | 12.3 s | 12.0 s | H: 10–12 s |
| Test | 13.0 s | 15.0 s | R: 11–16 s |
| INR | 0.93 | 1.10 | 0.89 |
| PH | 7.108 | 7.604 | 7.35–7.45 |
| $PaCO_2$ | 28.1 mmhg | 59.1 mmhg | 35–45 mmhg |
| $PaO_2$ | 45.2 mmhg | 48.2 mmhg | 80–100 mmhg |
| $HCO_3$ | 14.2 mmol/l | 30.1 mmol/l | 22–26 mmol/l |
| $SaO_2$ | 63% | 72% | 95–100% |

pH: Potential of hydrogen, $PaCO_2$: Partial pressure of carbon dioxide, $PaO_2$: Partial pressure of oxygen, $HCO_3$: Bicarbonates, $SaO_2$: Oxygen saturation, CRP: C- reactive protein, WBC: White blood cell, DLC: Differential leukocyte count

**Table 5: Hemogram, d-dimer, CRP values, coagulation profile, and arterial blood gas analysis of case 5**

| Hemogram | Result | | |
|---|---|---|---|
| | Baseline (on ICU admission) | Last recorded values | Reference range |
| Hemoglobin | 12.7g/dl | 14.7g/dl | 12–16 g/dl g/dl |
| Total WBC count | 9600/cumm | 14,600/cumm | 4–11,000/cumm |
| DLC | | | |
| Polymorphs | 77% | 92% | 40–80% |
| Lymphocytes | 19% | 09% | 20–40% |
| Eosinophils | 01% | 01% | 1–6% |
| Monocytes | 03% | 02% | 2–10% |
| Platelet count | 3.1 lacs/cumm | 2.3 lacs/cumm | 1.5–4.05 lacs/cumm |
| ESR | 5 mm/1hr | 15 mm/1 h | <20 mm/1 h |
| Abo and RH typing | "O" positive | | |
| D-dimer | 232.6 ng/ml | 355.5 ng/ml | <500 ng/ml |
| CRP | 1:8 | 1:4 | |
| Coagulation profile | | | |
| PT | | | |
| Control | 13.2 s | 13.4 s | H: 10–12 s |
| Test | 17.7 s | 15.9 s | R: 11–16 s |
| INR | 1.0 | 1.32 | |
| PH | 7.440 | 7.501 | 7.35–7.45 |
| $PaCO_2$ | 35.9 mmhg | 28.8 mmhg | 35–45 mmhg |
| $PaO_2$ | 58.9 mmhg | 78.6 mmhg | 80–100 mmhg |
| $HCO_3$ | 24.4 mmol/l | 22.8 mmol/l | 22–26 mmol/l |
| $SaO_2$ | 91.3% | 90.1% | 95–100% |

PT: Prothrombin time, ESR: Erythrocyte sedimentation rate, CRP: C- reactive protein, pH: potential of hydrogen, $PaCO_2$: Partial pressure of carbon dioxide, $PaO_2$: Partial pressure of oxygen, $HCO_3$: Bicarbonates, $SaO_2$: Oxygen saturation, DLC: Differential leukocyte count

**Table 6: Quick SOFA (q SOFA) score of five patients on ICU admission**

| Case number | Respiratory rate ≥22/min | SBP ≤100 mm of Hg | Altered mental status | Total score |
|---|---|---|---|---|
| Case 1 | −1 | 0 | 0 | −1 |
| Case 2 | −1 | 0 | 0 | −1 |
| Case 3 | −1 | 0 | 0 | −1 |
| Case 4 | −1 | 1 | 1 | −3 |
| Case 5 | −1 | 0 | 0 | −1 |

SOFA: Sequential organ failure assessment, SBP: Systolic blood pressure, =ICU: Intensive care unit

**Table 7: SOFA score**

| Hours/days of ICU admission | Case 1 | Case 2 | Case 3 | Case 4 | Case 5 |
|---|---|---|---|---|---|
| 24 h/1st day | 4 | 4 | 9 | 10 | 4 |
| 48 h/2nd day | 4 | 7 | 7 | 10 | 4 |
| 72 h/3 day | 3 | 4 | 6 | 12 | 5 |
| 120 h/5th day | 2 | 4 | 8 | 12 | 4 |
| 168 h/7th day | 4 | 4 | 8 | 14 | 4 |
| 216 h/9th day | 2 | 7 | 12 | 13 | 3 |
| 264 h/11th day | 0 | 8 | - | 16 | 2 |
| 312 h/13th day | 0 | 8 | - | 15 | 0 |
| 360 h/15th day | 0 | 12 | - | 15 | 0 |
| 408 h/17th day | - | 16 | - | 18 | - |
| 456 h/19th day | - | 14 | - | 18 | - |
| 504 h/21st day | - | 16 | - | | - |

SOFA: Sequential organ failure assessment, ICU: Intensive care unit

Baseline CT chest showed subpleural and peribronchial ground glass opacity (Figure 1) and crazy-paving pattern (Figure 2) or a mixture of these parenchymal lesions with involvement of all lobes in all the case series where Chest CT was performed [Figures 3 and 4].

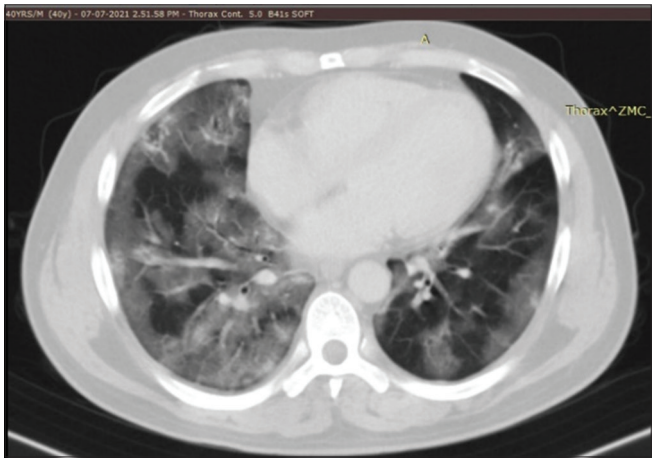


**Figure 1:** Baseline CT chest showed subpleural and peribronchial Ground glass opacity in all the case series where Chest CT was performed

## DISCUSSION

The present case series was a retrospective type of observational study. This was comparable with other studies reporting pneumothoraces among COVID-19, namely, Cate et al.,[7] Ding et al.,[8] Ekanem et al.,[9] Guo et al.,[10] Martinelli et al.,[11] McGuinness et al.,[12] Wang et al.,[13] Zantah et al.,[14] and Miró et al.[15] All the above-mentioned COVID19 and pneumothorax related studies were

retrospective in nature, but among them few studies were case control in nature and while few others were retrospective cohort in nature.

The age range in the present case series were from 39 to 57 years. The mean age was around 48.8 years in the present case series. The age was comparatively less in the present case series when compared to a systematic review done by Chong et al.,[16] which showed the mean and medium age of COVID-19 patients fell between fifth to seventh decade of life. Majority of our case series were males (60%). This was comparable with the systematic review done by Chong et al.,[16] and also by other studies[9,11,13] shown in Table 8, which shows majority were males. From the below

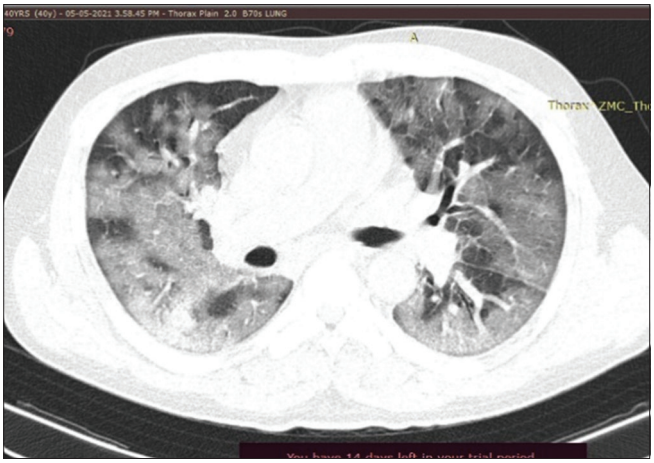

**Figure 2:** Baseline CT Chest showed crazy - paving pattern or a mixture of parenchymal lesions with involvement of all lobes in all the case series where Chest CT was performed

Table 8, which shows a comparison with other studies of pneumothorax among COVID-19 patients, we can see that the mean and median age was higher when compared with the present study.[9,11,13] In the present case series, the mortality was around 60% this was comparable with a study done by Wang et al.,[13] which showed a mortality of 80%, while other studies showed less mortality[9,11] (Table 8).

In the present case series, COVID -19 patients were stable enough to be shifted to Radiodiagnosis Department underwent CT examination using Siemens Somatom Scope 16 slice CT scanner or Fuji Digital X-ray machine according to availability at the time of admission as a part of baseline investigation. CT and CXR Severity Index scores were obtained for all patients. Although portable plain CXR is less cumbersome than CT for the evaluation of pneumothorax, it has low sensitivity in detecting intrapleural air especially in supine position.[17,18] CT has long been recognized as the gold standard for diagnosis of pneumothorax but it has limitations in unstable patients who cannot be transported outside the ICU. Chest US has become more popular over the years in the setting of acute respiratory emergencies including pneumothorax. Diagnosis of pneumothorax with US was first reported in 1986 by Rantanen.[19] Recently numerous clinical studies have shown that pneumothorax and pneumomediastinum can be diagnosed by US with high sensitivity and specificity.[20-26] In the present case series, all the five patients were found to have bilateral pneumothorax as confirmed by US of the chest. Another advantage of US is that while performing the US, the site marking for insertion of the ICD can be done simultaneously. In our center, we used GE Loqiq E US machine for those patients

**Table 8: Comparison of present case series with similar studies related to pneumothorax in COVID-19 patients[9,11,13]**

| Study done by | Study design location | Sample size | Age and gender | Co morbidity and other risk factors | Diagnosed by | Mortality (%) |
|---|---|---|---|---|---|---|
| Ekanem et al. | Retrospective Cohort USA | 22 cases of pneumothorax | Median Age - 60 Males- 82% | 52%- Hypertension 32%- Diabetes 14%- Smokers | Chest X-ray | 36 |
| Martinelli et al. | Retrospective case series United Kingdom | 60 patients with pneumothoraces. 6 patient had pneumomediastinum in addition to pneumothorax. 11 patients had pneumomediastinum alone. | Males- 77% patients aged≥70 years had a significantly lower 28-day survival than younger individuals. | Pre-Existing Lung Disease- 60% Hypertension- 32% Diabetes- 17% Chronic Kidney Disease- 7% | All patient by Chest Radiography. 37 patients underwent CT Scan also. | 48 |
| Wang et al. | Retrospective Case series China | 5 patients | Males- 100% Mean Age- 64.2 | No history of any pre-existing lung disease. Smokers- 0% | Chest-Ray and CT Scan chest | 80 |
| Present case series | Retrospective case series Mizoram, India | 5 Cases of Bilateral Pneumothorax | Mean Age – 48.8 60%- Males | Diabetes-40% Hypertension- 20% No history of any pre-existing lung disease | Chest ultrasonography | 60 |

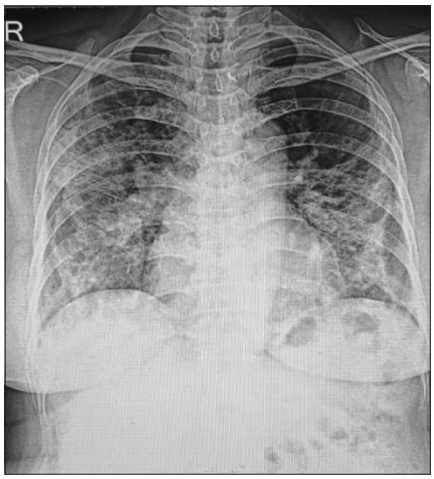


**Figure 3:** Chest radiograph of case 5 showing Severe COVID pneumonia with relative sparing of left upper zone

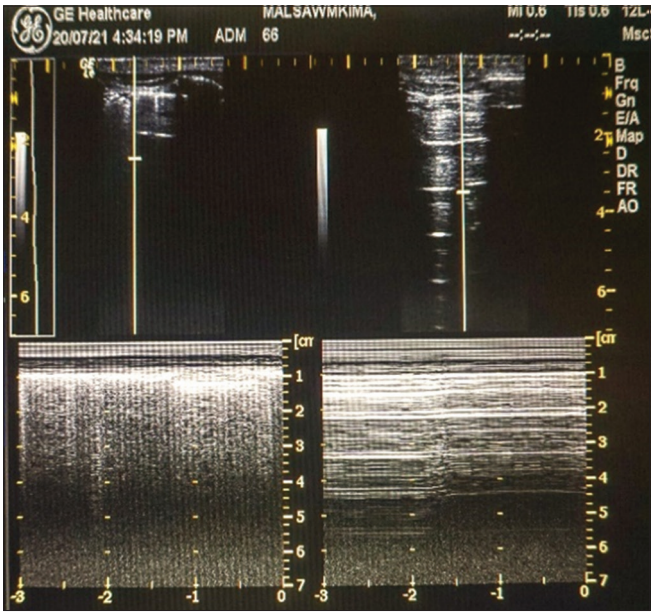


**Figure 4:** (a) Ultrasound of normal chest showing the seashore sign in Motion mode. (b) The typical barcode or stratosphere sign in pneumothorax which was observed in all our cases in the study

suspected to have pneumothorax using linear probe and motion mode during examination.

Smoking has been found a risk factor for Pneumothorax. Studies done by Tsuboshima et al.,[27] and Akinci et al.,[28] showed that majority were smokers who developed pneumothorax. In the present case series of 5, we did not have any smoker, all the patients were reported to be non-smokers. Even in a retrospective case series of 5 done by Wang et al.,[13] reported that all patients were non-smokers and also without any pre-existing lung disease which was very much comparable with the present study. Except for two cases with comorbidity of DM, and a case of hypertension all cases were free of risk factors for secondary spontaneous pneumothorax such as underlying pulmonary diseases, namely, COPD, cystic fibrosis, tuberculosis, lung cancer, HIV associated pneumocystis jiroveci pneumonia, and pulmonary cystic lung diseases.[1-3] Mechanical ventilation itself can be a risk factor for developing pneumothorax. Although an uncommon presentation of COVID-19, the occurrence of subcutaneous emphysema, pneumothorax, and pneumomediastinum can all occur in COVID-19 pneumonia in the presence or absence of mechanical ventilation, and these can contribute to profound hypoxemia seen in these patients.[29]

Although imaging of the cardia and pericardium can be done with the echocardiography (ECHO) probe to demonstrate pneumomediastinum, which may show diffuse A lines in the parasternal long and short views and apical views suggesting air artifacts, ECHO was not performed in view of rapid decline of our patients and immediate insertion of chest tube with water seal was considered to be more beneficial for the patients in such acute settings. In the present case series, two patients appeared to have developed pneumothorax before initiating invasive mode of mechanical ventilation, and one of these two patients had developed subcutaneous emphysema with bilateral pneumothorax. In the absence of predisposing factors for pneumothorax in the five cases reported here, COVID-19 pneumonia seems to be the primary cause of bilateral pneumothorax.

## CONCLUSION

The present case series showed that none of the cases were smokers, with no previous history of lung diseases and were all below 60 years indicating that except for COVID-19 pneumonia, there were no risk factors for respiratory compromise or dysfunction. Hence, we can conclude that COVID-19 patients may be prone to develop pneumothorax irrespective of absence of prior underlying lung disease or respiratory compromise. Likewise we can also conclude bilateral pneumothorax can be quite common among COVID-19 patients because of COVID-19 pneumoniae causing damage to the lungs. Our case series also showed that chest US can be an effective tool in diagnosis of pneumothorax and also has the advantage to site mark the place for insertion of ICD along with diagnosis. Finally, we can conclude that the presentation of pneumothorax in COVID-19 patients can be atypical at times and may not follow the routine pattern.

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