

Spontaneous Pneumothorax and Pneumomediastinum in COVID 19: Case Series

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Abstract: SARS COV2 infection can produce pneumothorax and spontaneous pneumomediastinum complications, which are associated with a worse prognosis. Here we present a series of cases of patients who presented subcutaneous emphysema caused by pneumothorax or spontaneous pneumomediastinum during care in the intensive care unit for pneumonia and COVID 19. This group of patients showed, in all cases, prolonged mechanical ventilation, refractory hypoxemia and hypercapnia, acute renal failure, bacterial superinfection, need for broad-spectrum antibiotics, and vasopressor support.

Keywords: Subcutaneous Emphysema, Spontaneous Pneumothorax, Spontaneous Pneumomediastinum, COVID 19.

INTRODUCTION

We are currently facing the COVID 19 pandemic, an infection caused by the SARS COV2 virus, which can manifest itself in cases ranging from mild cases (81%) to critically ill patients characterized by the presence of respiratory failure, septic shock, and/or multiorgan dysfunction (5%), with a mortality rate of 49% in the latter (1, 2). Various complications associated with COVID 19 have been reported, including pulmonary complications such as spontaneous pneumothorax and pneumomediastinum, defined as air in the pleural and mediastinal space without an apparent cause (3). Spontaneous pneumothorax occurs in 1% of cases and can occur at any time during the disease (4,5). Below, we present six patients who developed one of these complications secondary to infection by COVID 19.

CLINICAL CASE NO. 1

Sixty-six-year-old male patient with no pathological history, diagnosed with COVID 19. He consulted the emergency department for presenting clinical symptoms of seven days of evolution characterized by cough with purulent expectoration associated with odynophagia, dyspnea, asthenia, and adynamia. On admission, the patient was tachycardic, desaturated, diaphoretic, bibasal sibilance on auscultation, requiring oxygen support by a non-rebreathing mask, and predicted risk of poor prognosis due to positive severity biomarkers (Table 1). Given the imminence of ventilatory failure, he was admitted to the intensive care unit (ICU).

Initial chest X-ray reveals bibasal pulmonary interstitial infiltrates without consolidation (Figure 1(a)). On admission, arterial gases revealed severe oxygenation disorder with PAFI (arterial O₂ pressure/inspired O₂ fraction ratio) but without respiratory work on physical examination. Oxygen therapy with high flow nasal cannula (Optiflow Servo2) was decided, titrating parameters up to FiO₂ 1.0 and

flow of 60 L/min. The patient tolerated this support during the first three days, even with a discrete improvement in oxygenation index.

However, on the fourth day of ICU stay and day 13 since the onset of symptoms, he presented sudden deterioration of oxygenation index associated with an increase in respiratory work and presence of subcutaneous emphysema at the cervical level, requiring orotracheal intubation. A chest X-ray was performed, which revealed the appearance of a right pneumothorax, areas with bilateral subcutaneous emphysema, and extensive pneumomediastinum (Figure 1(b)). Due to the magnitude of the findings, it was necessary to perform an emergency closed thoracostomy by the general surgery service.

A pulmonary protection strategy was established with mechanical ventilatory support and pronation cycles; however, he developed refractory hypoxemia with a torpid evolution due to multiorgan dysfunction with an inflammatory response. The patient received beta-lactam antibiotics to treat a possible pulmonary superinfection, but there was no improvement. A bronchopleural fistula was considered, but his condition did not allow any additional intervention. Without criteria for extracorporeal oxygenation due to the severity and multisystemic involvement, the patient finally died on day 8 of his admission to the ICU.



Figure 1(a): Chest X-ray in anteroposterior projection. Bibasal interstitial infiltrates without consolidation.

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Table 1: Biomarkers of severity of pneumonia COVID 19 at admission.

Biomarkers of severity at admission	Case 1	Case 2	Case 3	Case 4	Case 5
Dimer D (normal value < 500 ng/ml)	11,085	540	1285	1169	640
LDH (normal value < 225 u/L)	578	422	578	958	450
Lymphopenia (normal value: 1.5 - 4 x10 ³ /uL.)	740	1.82	230	600	610
Ferritin (normal value: 30-400 ng/mL.)	4012	869	2012	1940	528

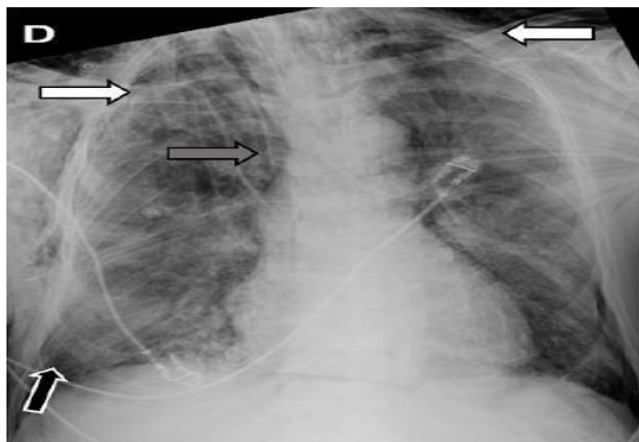


Figure 1(b): Anteroposterior projection chest X-ray. Right pneumothorax, bilateral subcutaneous emphysema, and extensive pneumomediastinum.

CLINICAL CASE NO. 2

Sixty-seven-year-old male patient with a medical history of essential hypertension, insulin-requiring type 2 diabetes mellitus, and grade 2 obesity (BMI 33.61 kg/m²), who was referred to a fourth level institution for presenting clinical symptoms of two days of evolution consisting of quantified febrile peaks, dry cough, and odynophagia. He was tachypneic, tachycardic, normotensive, and afebrile with oxygen saturation of 83% with FiO₂ at 21% on admission. Oxygen therapy with a non-rebreathing mask was decided. On admission, arterial blood gases showed moderate oxygenation disturbance (PaFi of 154 mmHg), chest X-ray with no evidence of consolidations or other alterations (Figure 2(a)). With a prediction of poor prognostic risk by positive severity biomarkers (Table 1).

During the stay, the patient showed increased respiratory work triggered by severe oxygenation disorder (PaFi of 83 mmHg), transfer to ICU was indicated for initiation of noninvasive ventilatory support with high flow nasal cannula (Optiflow Servo2), titrating parameters up to FiO₂ 1.0 and flow of 50 L/min with an adequate response. However, on the second day of stay in the unit, he exhibited a decrease in oxygenation indices (PaFi of 54 mmHg) associated with polypnea, so it was decided to perform orotracheal intubation without complications, insertion of a right subclavian central venous catheter, and initiation of vasopressor support. However, in the following days, he showed a torpid evolution due to a suggestive clinical profile of septic shock, consisting of signs of systemic inflammatory response and an increment in the need for vasopressor support was decided to perform blood culture and start antibiotic treatment with ureidopenicillin.

A chest X-ray was requested in an active search for an infectious focus, showing an extensive right pneumothorax cavity with a total collapse of the pulmonary parenchyma and deviation of the cardiome-diastinum to the left (Figure 2(b)). Therefore, a closed right thoracostomy was performed without complications. During the postoperative period, the patient showed asystole, it was decided to limit therapeutic effort, and he died.



Figure 2(a): Chest X-ray in anteroposterior projection. No consolidations or other alterations are observed.

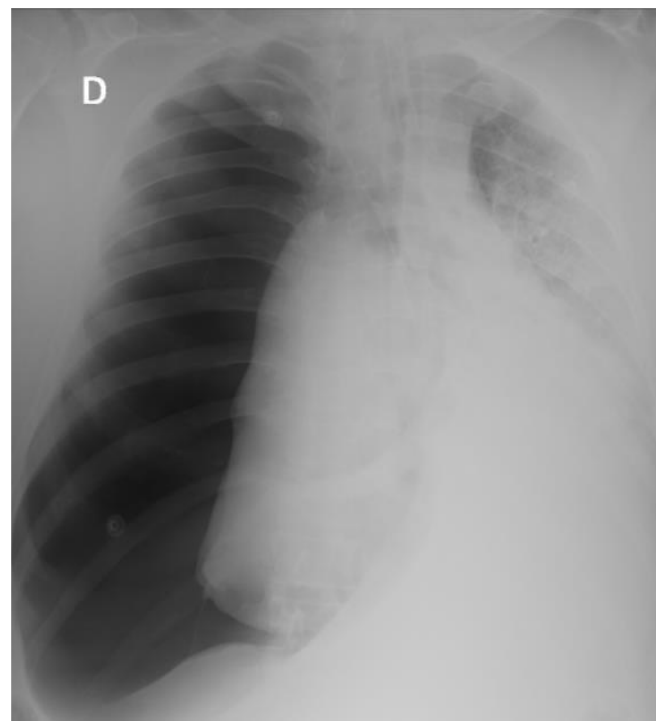


Figure 2(b): Chest X-ray in anteroposterior projection. Extensive right pneumothorax cavity with a total collapse of the pulmonary parenchyma and deviation of the cardiome-diastinum to the left.

CLINICAL CASE NO. 3

Female patient in the ninth decade of life, with multiple pathological antecedents given by: small vessel P-ANCA vasculitis, community-acquired pneumonia with microbiological isolation of pseudomona aeruginosa with carbapenemics resistance pattern, non-insulin-requiring type 2 diabetes mellitus, primary hypothyroidism, essential arterial hypertension, and mixed dyslipidemia.

The patient consulted a fourth-level institution for clinical symptoms of one day of evolution consisting of altered consciousness associated with signs of respiratory distress due to accessory muscles use and polypnea. On admission, the patient was disoriented in the three spheres, with sinus tachycardia (130 bpm), tachypnea (40 rpm), limited mean arterial blood pressure (65 mmHg) and desaturated (85%) with non-rebreathing mask at 15 liters, arterial gases with severe oxygenation disorder (Pafi 89 mmHg) and positive biomarkers of severity (Table 1). Therefore, orotracheal intubation was performed without complications, and mechanical ventilation was started with pulmonary protection parameters, insertion of a right subclavian central venous catheter, and initiation of vasopressor support. After the intervention, a chest X-ray was requested with evidence of orotracheal tube in situ, interstitial process with multifocal consolidations mainly on the left side, and atelectasis in the middle lobe (Figure 3(a)).

Based on the clinical picture described, a diagnosis of shock is defined with an etiology to be established: obstructive due to suspected pulmonary thromboembolism (Wells Scale - Intermediate Risk) versus distributive due to sepsis of viral etiology with bacterial coinfection, for which broad-spectrum antibiotic management was initiated, under suspicion of multidrug-resistant germs with carbapenem plus glycopeptide.

Given the condition of the patient, transfer to ICU was requested. The physical examination on admission showed subcutaneous emphysema at the thoracic level. A chest X-ray was ordered, showing pneumomediastinum and emphysema in the cervical region and the upper portion of the hemithorax, predominantly on the right; pneumothorax was ruled out (Figure 3(b) and 3(c)). On the other hand, chest angiotomography showed a slight pneumomediastinum and no evidence of pulmonary thromboembolism.

Despite the multiple interventions performed, the patient presented a torpid evolution with refractory shock associated with multiorgan dysfunction. After five days, she showed asystole, and cardiopulmonary resuscitation maneuvers were started for approximately 20 minutes; however, she did not return spontaneous circulation and died.



Figure 3(a): Chest X-ray in anteroposterior projection. Interstitial process with multifocal consolidations predominantly on the left side and atelectasis in the middle lobe.



Figure 3(b): Chest X-ray in anteroposterior projection. Pneumomediastinum and emphysema in the cervical region and the upper right hemithorax.



Figure 3(c): Chest X-ray in anteroposterior projection. Pneumomediastinum and emphysema in the cervical region and the upper right hemithorax.

CLINICAL CASE NO. 4

Fifty-three-year-old patient with a pathologic history of gouty arthritis, who consulted the emergency department for clinical symptoms of one week of evolution characterized by adynamia, asthenia, hyporexia, dry cough, chills, quantified fever, and progressive dyspnea up to medium exertion. Since his admission, oxygen saturation was 80% with no respiratory work, so supplementary oxygen was started with a non-rebreathing mask.

The patient with a predicted poor prognostic risk due to positive severity biomarkers (Table 1), arterial blood gases with mild oxygenation impairment, chest X-ray with ground glass multilobar opacities, tends to bibasal consolidation (Figure 4(a)) and RT-PCR for SARS COV 2. In the following twenty-four hours, the patient developed severe acute respiratory distress syndrome (ARDS). High flow nasal cannula (Optiflow Servo2) was started, titrating up to maximum parameters without adequate response, so orotracheal intubation was performed. Orotracheal intubation was performed, followed by invasive mechanical ventilation, insertion of an ultrasound-guided right jugular venous catheter without complications, and initiation of vasopressor support. Nevertheless, the patient had a stationary evolution with evidence on the physical examination of cervical subcutaneous emphysema and the anterior thorax region. A high-resolution chest X-ray and computed tomography were requested because of the findings, which confirmed subcutaneous emphysema and pneumomediastinum, plus radiological worsening by COVID 19 (Figure 4(b) and 4(c)).

The patient developed multiorgan dysfunction due to septic shock of pulmonary origin and suspected bacterial superinfection, respiratory acidemia, and severe refractory hypoxemia with high ventilatory parameters, severe tissue hypoperfusion, minimally invasive hemodynamic monitoring with a permanent hyperdynamic pattern. The following day, the patient showed a non-defibrillable arrest rhythm, and resuscitation was started for 15 minutes without a return to spontaneous circulation, and he died.

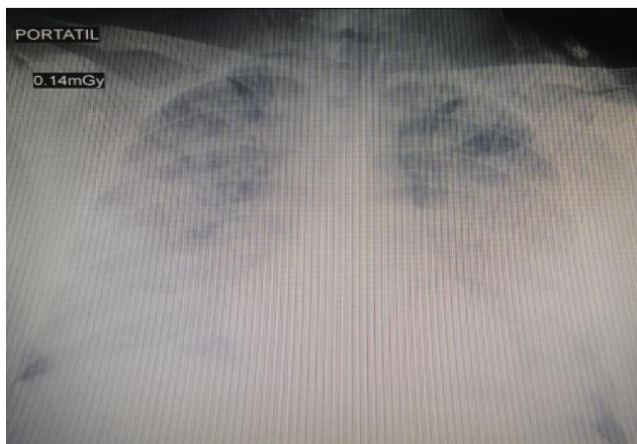


Figure 4(a): Chest X-ray in anteroposterior projection. Ground-glass multilobar opacities with a tendency to bibasal consolidation.

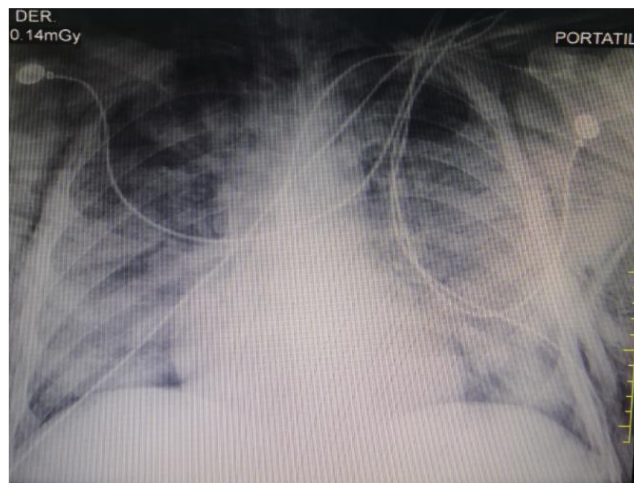


Figure 4(b): Chest X-ray in anteroposterior projection. Subcutaneous emphysema and pneumomediastinum.

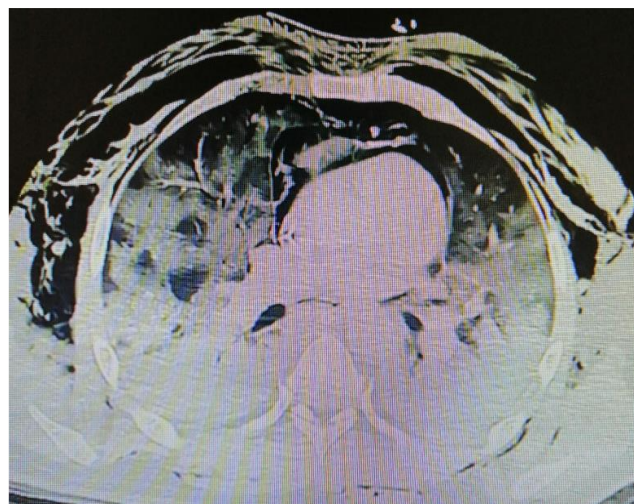


Figure 4(c): High resolution computed tomography of the lung. Subcutaneous emphysema and pneumomediastinum.

CLINICAL CASE NO. 5

Thirty-four-year-old male patient with a history of interstitial lung disease under study who required segmental lobectomy by thoracoscopy for pathological study. Postoperatively, the patient experienced sudden dyspnea, cyanosis, and oxygen desaturation of 56%, with FiO₂ at 21%, for which he consulted a fourth-level institution.

On admission, the patient was tachycardic, desaturated, cyanotic, and polypneic. Pulmonary auscultation showed preserved vesicular murmur without pulmonary aggregates, requiring oxygen support with the conventional nasal cannula at 2 liters with an adequate response. Admission clinical tests showed positive severity biomarkers (Table 1), arterial blood gases with moderate oxygenation disorder (PaFi of 178 mmHg), and chest X-ray showing multiple alveolar consolidations infiltrates, diffusely distributed in both lung fields, with a peripheral predominance of the left hemithorax compatible with SARS VOC 2 infection (Figure 5(a)); confirmed by antigen test. Due to adequate tolerance to nasal cannula oxygen, the patient was discharged with home oxygen.

The patient was readmitted to the institution seven days after hospital discharge due to dyspnea predominantly at night and desaturation despite supplemental oxygen. On admission, the patient had desaturation of 40% with FIO₂ at 28%, generalized cyanosis, intercostal and supraclavicular retractions. A non-rebreathing mask was started at 15 liters which improved oxygen saturation. Chest X-ray was requested, showing no significant changes than the initial one, and severity biomarkers remained positive. During the hospital stay, the patient suffered from respiratory work added to severe oxygenation disorder (PaFi of 72 mmHg), for which invasive mechanical ventilation was started, and the patient was transferred to the ICU. The patient could not adequately adjust to the ventilator due to bronchospasm and was controlled with a salbutamol crisis scheme. In addition, the patient exhibited signs of systemic inflammatory response, so bacterial superinfection was suspected, blood culture was performed, and empirical antibiotic treatment with ureidopenicillin was started.

The patient partially improved his oxygenation, which allowed the decrease of ventilatory parameters. However, the physical examination revealed subcutaneous emphysema in the thoracic region, so a chest X-ray was performed showing alveolar opacities, pneumomediastinum, and extensive subcutaneous emphysema, without pneumothorax. A high-resolution chest computed tomography (CT - RA) was performed with extensive soft tissue emphysema of the thoracic wall, pneumomediastinum, and slight pneumopericardium to enhance the imaging findings, as shown in Figure 5(b).

Patient with improved evolution, with extubation for 8 days of ventilation mechanical and ICU discharge for the hospitalization.

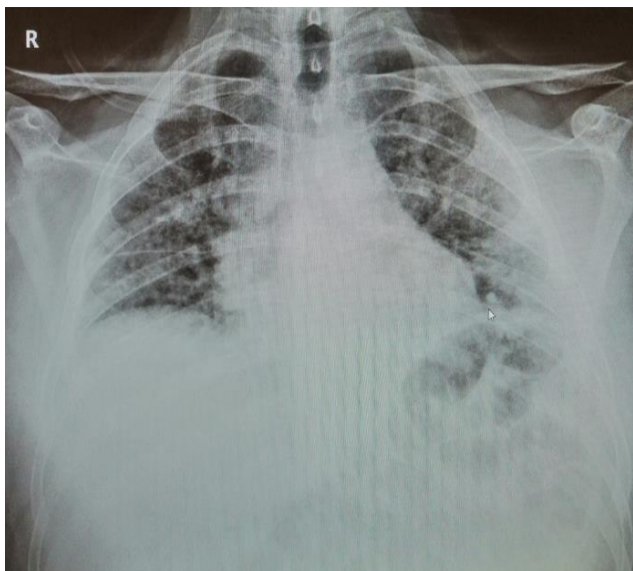


Figure 5(a): Chest X-ray in anteroposterior projection. Multiple alveolar consolidations infiltrate, diffusely distributed in both lung fields, with peripheral predominance in the left hemithorax.

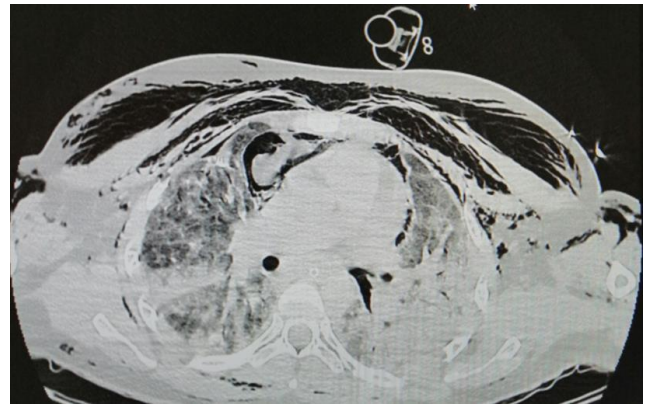


Figure 5(b): High resolution computed tomography of the lung. Extensive soft tissue emphysema of the chest wall, pneumomediastinum, and slight pneumopericardium.

CLINICAL CASE NO. 6

Female patient in the seventh decade of life, with a pathological history of sleep apnea syndrome, essential hypertension, and grade 2 obesity with SARS COV2 infection under home oxygen management who, due to worsening respiratory symptoms, decided to consult the emergency department. On admission, with positive severity biomarkers and severe oxygenation disorder, the patient required support with a high-flow nasal cannula (HFNC), pronation vigil cycles, and admission to the ICU. In the beginning, the patient showed partial improvement in oxygenation, however, she progressed to a severe hypoxemic respiratory failure, and mechanical ventilatory support was started.

Despite the interventions performed, the patient showed clinical deterioration associated with bacterial coinfection by *Klebsiella pneumoniae*, extreme bradycardia that required transcutaneous transvenous pacemaker implantation, and initiation of renal replacement therapy due to signs of fluid overload refractory to treatment. The patient with stationary response to management presented pacemaker dysfunction with subcutaneous emphysema in the neck and anterior thorax. Given the physical examination findings, a chest X-ray was performed showing soft tissue emphysema, and a high-resolution chest CT scan revealed extensive pneumomediastinum, bilateral pneumothorax of no more than 10%. Fibrobronchoscopy was considered to rule out airway injury; however, due to hemodynamic instability, the procedure was deferred. The patient continued with the clinical deterioration that led to cardiorespiratory arrest and death.

DISCUSSION

The distinctive pulmonary involvement in diagnostic images due to SARS VOC 2 infection are bilateral multilobar opacities, ground-glass type, and/or consolidations with peripheral or subpleural distribution (6,7). Our patients debuted mechanical ventilation, with sudden worsening of oxygenation or ventilatory mechanics mainly due to increased airway pressures and poor ventilatory coupling despite adequate

sedation. The above hinders the maintenance of pulmonary protection parameters, regardless of the position adopted (prone/supine), associated with subcutaneous emphysema at cervical or anterior thorax level. Similarly, those who were not intubated required immediate invasive mechanical ventilation.

Although dyspnea and unexpected deterioration of oxygenation are nonspecific signs shared in pneumonia, pneumothorax, and spontaneous pneumomediastinum, our cases are examples for health personnel to suspect these complications in the presence of the following findings: an unexpected increase in dyspnea, abrupt alteration of ventilatory mechanics, dyssynchronies, decreased oxygenation indices or increased airway pressures, despite optimal sedation (6, 7) because initially in our patients these alterations were not considered until the presence of subcutaneous emphysema with control radiological findings.

Approximately 1% of patients diagnosed with COVID 19 may present spontaneous pneumothorax due to this disease (4). In turn, sporadic cases of spontaneous pneumothorax concomitant with pneumomediastinum have been described, even pneumopericardium (Figure 5(b)). It is known that the presence of these entities worsens the clinical outcome and is associated with fatal consequences. Although it has not been corroborated whether these are indicators of severity in the face of infection, the outcome in most our patients was death (8). The pathophysiological mechanism of subcutaneous emphysema, pneumothorax, and spontaneous pneumomediastinum is unknown. However, regarding severe pneumonia associated with acute respiratory distress syndrome, possible etiologies have been described, such as diffuse alveolar damage, spontaneous rupture of a bulla, pneumatocele or subpleural cyst, and barotrauma caused by VILI (ventilation-induced lung injury) (9).

It should be pointed out that the characterization of these lesions employing AR CT is not always available due to availability in the institutions or because the critical conditions of the patient prevent the transfer, which happened in the first two cases of this article. Subcutaneous emphysema, pneumothorax, and pneumomediastinum should be monitored due to the potential risk of respiratory and cardiovascular complications leading to death if they are not detected and intervened in time (10). It is important to mention that these are not recent entities specific to COVID-19 since they have been described as complications of patients subjected to mechanical ventilation or directly due to other severe pulmonary infectious processes. In addition, it seems that these are infrequent complications of viral pneumonias described in SARS pneumonia (severe acute respiratory syndrome), Influenza A (H1N1), and recently SARS COV 2 (6, 7).

Risk factors associated with these conditions include smoking, age, male sex, body mass index <18.5 kg/m², prolonged cough, strenuous exercise, and some diseases such as chronic obstructive pulmonary

disease (11). It has been reported that frequent and intense coughing spells, not necessarily in mechanical ventilation, cause an increase of pressure in the distal or intraalveolar airway, which, when repetitive, generate alveolar rupture, originating pneumothorax, with or without pneumomediastinum due to the leakage of alveolar gas to the mediastinum after dissecting the peribronchovascular interstitium and depending on the magnitude of the air that could pass to the parietal pericardium giving rise to pneumopericardium, a phenomenon called "Macklin Effect," which has also been described in other entities such as closed thoracic trauma, asthmatic crisis and Valsalva maneuvers (12-14). We describe what happened with these patients to inform and alert about this complication, which, although infrequent, is usually fatal when associated with ARDS and refractory hypoxemia in patients with COVID-19 despite the timely diagnosis and medical-surgical management.

CONCLUSION

Spontaneous pneumothorax and/or pneumomediastinum in COVID 19 infection are rare but severe complications, usually generated by increased distal airway pressures. It should be suspected when there is an abrupt deterioration of oxygenation and ventilatory mechanics. It may present in isolation as spontaneous pneumothorax or conjunction with pneumomediastinum, with subcutaneous emphysema being the cardinal alarm sign. It is necessary to report more of these cases to learn more about their pathophysiology and establish specific therapeutic measures or recommendations for these entities.

ACKNOWLEDGMENT

Dedicated to all the patients who suffered COVID 19, especially to my parents Jairo Robayo and Blanca Valbuena, who continue accompanying us from heaven.

REFERENCES

- [1] Accini-Mendoza JL, Beltrán N, Nieto-Estrada VH, Ramos-Bolaños E, Pizarro-Gómez C, Rebolledo CE, et al. Declaration of consensus in critical medicine for multidisciplinary care of the patient with a suspected or confirmed diagnosis of covid-19. *Acta Colombiana de Cuidado Intensivo*. Available online 17 April 2020. <https://doi.org/10.1016/j.acci.2020.04.003>
- [2] Berlin DA, Gulick RM, Martinez FJ. Severe Covid-19. *N Engl J Med*. 2020;383(25):2451-60. <https://doi.org/10.1056/nejmcp2009575>
- [3] Bintcliffe O, Maskell N. Spontaneous pneumothorax. *BMJ*. 2014; 348: g2928. <https://doi.org/10.1136/bmj.g2928>
- [4] Chen N, Zhou M, Dong X, Qu J, Gong F, Han Y, et al. Epidemiological and clinical characteristics of 99 cases of 2019 novel coronavirus pneumonia in Wuhan, China: a

- descriptive study. *Lancet*. 2020;395(10223):507-13.
[https://doi.org/10.1016/s0140-6736\(20\)30211-7](https://doi.org/10.1016/s0140-6736(20)30211-7)
- [5] Brogna B, Bignardi E, Salvatore P, Alberigo M, Brogna C, Megliola A, et al. Unusual presentations of COVID-19 pneumonia on CT scans with spontaneous pneumomediastinum and loculated pneumothorax: A report of two cases and a review of the literature. *Heart Lung*. 2020;49(6):864-8.
<https://doi.org/10.1016/j.hrtlng.2020.06.005>
- [6] Yang Q, Liu Q, Xu H, Lu H, Liu S, Li H. Imaging of coronavirus disease 2019: A Chinese expert consensus statement. *Eur J Radiol*. 2020;127:109008.
<https://doi.org/10.1016/j.ejrad.2020.109008>
- [7] Wang W, Gao R, Zheng Y, Jiang L. COVID-19 with spontaneous pneumothorax, pneumomediastinum and subcutaneous emphysema. *J Travel Med*. 2020;27(5).
<https://doi.org/10.1093/jtm/taaa062>
- [8] López Vega JM, Parra Gordo ML, Díez Tascón A, Ossaba Vélez S. Pneumomediastinum and spontaneous pneumothorax as an extrapulmonary complication of COVID-19 disease. *Emerg Radiol*. 2020;27(6):727-30.
<https://doi.org/10.1007/s10140-020-01806-0>
- [9] Parra Gordo ML, Weiland GB, García MG, Choperena GA. Radiologic aspects of COVID-19 pneumonia: outcomes and thoracic complications. *Radiologia*. 2021;63(1):74-88.
- [10] Zendah I, Bacha S, Daghfous H, Ben M'rad S, Merai S, Tritar F. [Management of spontaneous pneumomediastinum in the adult: 14 cases and a review of the literature]. *Rev Pneumol Clin*. 2010;66(3):163-6.
<https://doi.org/10.1016/j.pneumo.2009.08.003>
- [11] Light RW. Management of spontaneous pneumothorax. *Am Rev Respir Dis*. 1993;148(1):245-8.
<https://doi.org/10.1164/ajrccm/148.1.245>
- [12] Chassagnon G, Favelle O, Derogis V, Cottier JP. Spontaneous pneumomediastinum due to the Macklin effect: less is more. *Intern Emerg Med*. 2015;10(6):759-61.
<https://doi.org/10.1007/s11739-015-1229-1>
- [13] Murayama S, Gibo S. Spontaneous pneumomediastinum and Macklin effect: Overview and appearance on computed tomography. *World J Radiol*. 2014;6(11):850-4.
<https://doi.org/10.4329/wjr.v6.i11.850>
- [14] Barquero Romero J, Izquierdo Hidalgo J, Maciá Botejara E, Arrobas Vacas J, Pérez Miranda M. [Spontaneous pneumopericardium in a patient with community-acquired pneumonia]. *Rev Esp*.
[https://doi.org/10.1016/s1885-5857\(06\)60366-6](https://doi.org/10.1016/s1885-5857(06)60366-6)