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Pneumothorax, Pneumomediastinum and Subcutaneous Emphysema Associated with Covid-19 Pneumonia

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ABSTRACT

Since Coronavirus 2019 (COVID-19) was considered a pandemic, research has helped us learn more about the disease and its complications. Spontaneous pneumothorax, pneumomediastinum and subcutaneous emphysema are rare complications of COVİD-19 and may develop in cases of underlying chronic diseases, infection or mechanical ventilation. Although they are not very common in viral pneumonia, we have compiled in our study that these pathologies can be encountered in patients followed up with Covid-19 pneumonia, even if mechanical ventilation is not applied.

Materials and Methods: In our hospital, 133 patients who were admitted to Chest Diseases / Covid services with the diagnosis of Covid pneumonia between June 2020 and January 2022 and developed spontaneous pneumothorax, pneumomediastinum and subcutaneous emphysema during follow-up were examined according to age, gender, comorbidities, laboratory and radiological findings, severity of accompanying pneumonia, and treatment method. and we evaluated it retrospectively according to its clinical results.

Results: We have evaluated 133 COVID-19 patients. The mean(±SD) age was 59.63(±15.24) (23-93). Women accounted for 35% (n=47) of the study population. Hypertension was the leading comorbidity (40%); 27 cases (20%) had Diabetes Mellitus, and 20 cases (15%) had a chronic lung disease, namely asthma or COPD. 130 cases had pneumothorax, 42 were left-sided (32.3%), 60 were right-sided (46.2%), and 28 were bilateral (21.5%). Pneumomediastinum was observed in 20 cases (15%) and 53 (39.8%) had subcutaneous emphysema. Bullae were detected in only 8 (6%) of the cases. It was observed that pneumothorax developed approximately 16.5 (1-49) days after PCR positivity. Mechanical ventilation (MV) was applied to 117 (88%) of 125 (94%) patients who were followed up in the ICU, and the mean duration was 6.9 (0-43) days in those who developed pneumothorax under MV. The mean duration of tube drainage follow-up was 8.5 (1-57) days, and the mean survival time after pneumothorax was 9.9 (0-122) days.116 (87.2%) of the cases died. The duration of hospital stay was 25.07±19.49(2-129) days; the duration of ICU admission was 19.22±16.61(1-95) days. 16 of the cases were followed up without mechanical ventilation. Pneumothorax was detected in 7 of them when they applied to the emergency service, 6 of them in the ICU, and 3 of them while receiving oxygen support in the service. When the laboratory parameters of the cases when pneumothorax developed compared to the time they applied to hospital, it was observed that there was a significant ($p<0.001$) increase in the leukocyte and platelet counts and D-dimer levels, while no significant change was found in lymphocyte count, CRP and Ferritin values.

Conclusion: An uncommon presentation of COVID-19 is the occurrence of SPM, PNX, and SCE. These unwanted clinical conditios may also develop during follow-up in the hospital or in the ICU. Mechanical ventilation is a major risk factor. As seen in the literature and our study, this is a wide spectrum, from a self-limiting condition that can be controlled with conservative treatments, to serious respiratory and circulatory pathologies and even death. For this reason, it is very important to question the symptoms, repeat the examination, laboratory and radiological imaging and closely monitor the patient who develops sudden deterioration, shortness of breath and a sudden increase in oxygen demand during clinical follow-up.

Abbreviations: SPM: Spontaneous Pneumomediastinum; PNX: Pneumothorax; SCE: Subcutaneous Emphysema; CT: Computerized Tomography; MV: Mechanical Ventilation

Introduction

All the research since the acceptance of Coronavirus 2019 (COVID-19) as a pandemic allows us to learn more about the disease and its complications. Spontaneous pneumomediastinum (SPM), pneumothorax (PNX) and subcutaneous emphysema (SCE) are rare complications of COVID-19. [1] SPM is a rare complication that may develop in patients with underlying chronic pulmonary disease, infection and mechanical ventilation. [2] Although it is not very common in viral pneumonia, it can be encountered in patients followed up with COVID-19 pneumonia, even if mechanical ventilation is not applied. Although the pathophysiological mechanism is not known exactly, it is thought that the increase in alveolar pressure, which may occur with coughing, together with diffuse alveolar damage in COVID-19 pneumonia, may cause alveolar rupture and thus air leakage extending to the mediastinum along the bronchoalveolar sheath. [3] Although this is a self-limiting condition that can be controlled with conservative treatments in many cases, it can lead to serious respiratory and circulatory pathologies in some cases, and even result in death.

For this reason, it is very important to question the symptoms, repeat the examination and radiological imaging, and closely monitor the patient, in patients whose clinical condition deteriorates rapidly with shortness of breath and sudden increase in oxygen demand in clinical follow-up. In this study, we aimed to present the demographic, laboratory, radiological and clinical findings of the patients admitted to dedicated COVID-19 wards and who were found to have pneumomediastinum, pneumothorax and subcutaneous emphysema in the clinical follow-up.

Materials and Methods

Study Design

We conducted this retrospective observational study on patients admitted to our hospital with the diagnosis of COVID-19 between 2020, June, and 2022, January. The study was approved by the Turkish Republic Ministry of Health and the local ethics committee. (Approval number: E-46059653-020).

Patients

The data of the consecutive patients admitted to dedicated COVID-19 wards in our hospital between 2020 June and 2022 January were collected. SARS-CoV-2 was confirmed by a real-time RT-PCR (reverse transcriptase polymerase chain reaction) test. Throat-swab specimens from the oropharynx and nasopharynx were obtained from all patients at admission and were put in a viral-transport medium. All the COVID-19 diagnoses were in accordance with the guidelines provided by the Ministry of Health, COVID-19 Turkey National Health Commission. Inclusion criteria in the study group were as follows: Clinical, laboratory, and/or radiological findings consistent with COVID-19, need for hospital admission, and having been diagnosed with pneumothorax, pneumomediastinum and subcutaneous emphysema. The subjects who had no confirmed SARS-CoV-2 infection, COVID-19 patients who did not have inpatient admission, patients aged under 18, and patients with insufficient data required for the study protocol were excluded from data collection.

Data Collection

The demographic data including age and gender, and comorbidities, namely hypertension, cardiovascular disease, and chronic respiratory disease of all patients were recorded. All subjects had been computerized tomography (CT) scanned on admission and radiological findings were recorded. The laboratory test results on the date of admission included complete blood count, D-dimer, Ferritin, CRP (C-reactive protein) Length of hospital stay, need for admission to intensive care unit, the course, and outcomes during hospitalization were recorded. Treatment protocols were applied according to the COVID-19 guide of the Turkish Ministry of Health [4].

Statistical Analysis

Categorical variables were described as numbers (%). Continuous variables were analyzed parametrically by means and standard deviations, median, minimum and maximum. Differences between categorical variables were calculated using the chi-square method. Differences between continuous variables were calculated using the T-test and ANOVA test. Pearson analysis was used for correlation tests. A P-value less than 0.05 was considered statistically significant. Statistical analyses were performed using SPSS software (version 21.0).

Results

We have evaluated 133 COVID-19 patients. The flowchart of the study population is demonstrated in Table 1. The mean(±SD) ages was 59.63(±15.24) (23-93). Women accounted for 35% (n=47) of the study population. Hypertension was the leading comorbidity (40%); 27 cases (20%) had Diabetes Mellitus, and 20 cases (15%) had a chronic lung disease, namely asthma or COPD. 130 cases had pneumothorax, 42 were left-sided (32.3%), 60 were right-sided (46.2%), and 28 were bilateral (21.5%). Pneumomediastinum was observed in 20 cases (15%) and 53 (39.8%) had subcutaneous emphysema. Radiological findings are presented in Figure 1. Bullae were detected inonly8 (6%) of the cases. It was observed that pneumothorax developed approximately 16.5 (1-49) days after PCR positivity. Mechanical ventilation (MV) was applied to 117 (88%) of 125 (94%) patients who were followed up in the ICU, and the mean duration was 6.9 (0-43) days in those who developed pneumothorax under MV. The mean duration of tube drainage follow-up was 8.5 (1-57) days, and the mean survival time after pneumothorax was 9.9 (0-122) days.116 (87.2%) of the cases died. The duration of hospital stay was 25.07±19.49(2-129) days; the duration of ICU admission was 19.22±16.61(1-95) days. 16 of the cases were followed up without mechanical ventilation.

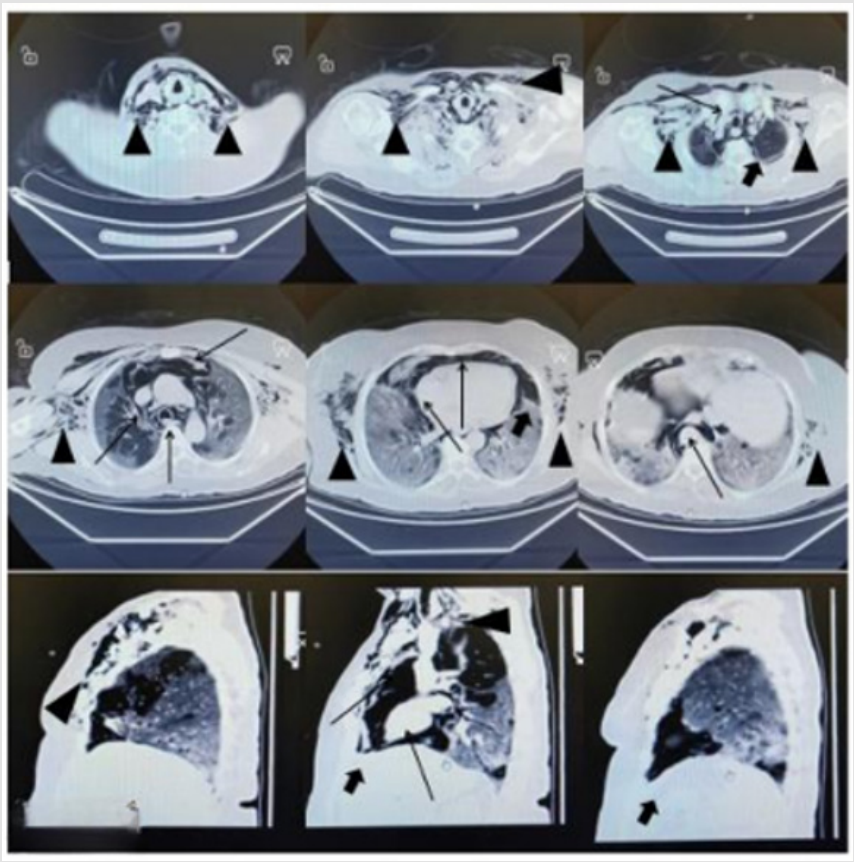

Figure 1: Axial and sagittal Chest CT scans of a patient 8 days after admisson showing groung glass opacities in both lungs. Subcutaneous emphysema starting from the neck, continuing to anterior and lateral walls of the chest and extending to both axillary regions.
(black triangle), pneumomediastinum in the pericardial area and surrounding all mediastinal structures (long arrow), as well as a pneumothorax on the left side (short arrow).

Table 1: Demographic and clinical details for pneumothorax, pneumomediastinum and subcutaneous emphysema for Covid-19.

Age	59.63 (±15.24)(23-93)
Female	47 (35%)
Comorbidities	Systemic Hypertension 40 % Diabetes Mellitus 20 % Chronic Lung Diseases 15 %
Day of pneumothorax after PCR positivity	16.55 (1-49) days
Radiological findings	Pneumothorax: 130 cases
	Left 42 (32.3%), Right 60 (46.2%), Bilateral 28 (21.5%)
	Bullae 8 (6%)
	Without chest drain : 2 cases (1.5%)
	Duration of chest drain: 8.54 (1-57) days
	Pneumomediastinum: 20 cases
	Subcutaneous emphysema:53 cases

Clinical follow up	Intensive Care Unit (ICU)	125 cases (94%)
	Intubated	117 cases (%88)
	Pneumothorax after intubation	6.94 days (0-43)
	ICU duration	19.2 days (1-95)
	Nonintubated	16 cases
		On admission : 7 cases
		During ICU follow up with O2 support: 6 cases
		During non-ICU follow up: 3 cases
Survival after Pneumothorax	9.99 (0-122) days	
Total hospital stay	25 (2-129) days	
Ex	116 cases (87.2%)	

Pneumothorax was detected in 7 of them when they applied to the emergency service, 6 of them in the ICU, and 3 of them while receiving oxygen support in the service. When the laboratory parameters of the cases when pneumothorax developed compared to the time they applied to hospital, it was observed that there was a significant ($p<0.001$) increase in the leukocyte and platelet counts and D-dimer levels, while no significant change was found in lymphocyte count, CRP and Ferritin values. The results of the laboratory parameters are summarized in Table 2.

Table 2: The comparison of laboratory data on admission and at time of pneumothorax.

	On Admission	At Time of Pneumothorax	P value
Leucocytes(/uL) Min-Max(Median) Mean±SD	420-35500(7790) 9076±5600	230-111980(15530) 18155±12042	<0.001
Lymphoctes(/mm³) Min-Max(Median) Mean±SD	80-46390(840) 1584±4704	10-83750(855) 1712±7325	0.864
Platelets(/uL) Min-Max(Median) Mean±SD	19500-509000(198000) 208387±78710	54000-948000(249500) 265807±130811.07	<0.001
CRP(mg/L) Min-Max(Median) Mean±SD	0.20-387.00(75) 10.46±84.77	0.20-466.00(113) 139.65±112.53	0.001
D-dimer (mg/L) Min-Max(Median) Mean±SD	0.19-52(1.1) 4.15±7.90	0.51-83(4.15) 8.76±11.67	<0.001
Ferritin(µg/L) Min-Max(Median) Mean±SD	11-9526(774.00) 1108.50±1128.12	52-100000(1329) 4516.03±12409.43	0.002

Discussion

During the COVID-19 pandemic, numerous studies have shown its typical and atypical chest CT findings. CT as a diagnostic test to identify patients with COVID-19 has a high sensitivity and a high negative predictive value. [5] The most frequent radiographical finding is ground-glass opacities occurring in the intermediate/late phase and reticular alterations occurring in the early phase. Distribution is usually bilateral, peripheral and with middle/lower predominance. Consolidation gradually occurs over time. [6] SPM, PNX, and SCE, although rare, may occur as complications of COVID-19, and result in worsening of the clinical condition. We present one of the largest reports on COVID-19 patients with SPM, PNX, and SCE. PM, also known as mediastinal emphysema, is the presence of air tracking along the mediastinum. [7] It can be spontaneous (SPM) or traumatic. Mechanical ventilation causing barotrauma, blunt or penetrating trauma to the chest or iatrogenic injury (eg, thoracic surgery) are examples of traumatic PM. SPM can be primary, in which no underlying lung disease is present, or secondary, in which an underlying lung or airway disease (eg, asthma or cystic fibrosis) that predisposes to air leak is present. The pathophysiology of SPM is based on the Macklin phenomenon, which describes the occurrence of a large pressure gradient between the marginal alveoli and the lung interstitium, resulting in air leak to the surrounding bronchovascular sheath [3].

One mechanism is an increase in intrathoracic pressure, resulting in an increase in intra-alveolar pressure and overinflation of the alveoli without corresponding expansion of the vascular lumen. This will result in a pressure gradient that can rupture the marginal alveoli, leading to air leaking to the interstitium which can track along the perivascular and peribronchial sheath to the hilum of the lung and then to the low-pressure mediastinum. Examples of such mechanisms include cough, vomiting, sneezing, defecation or asthma exacerbation. The second mechanism is caused by a reduction in the caliber of the pulmonary vessels, without a corresponding diminution of the alveolar pressure. This will increase the pressure gradient causing air leaks to the sheath. Examples include forced expiration against obstruction which will dam the blood back to the venous side decreasing the vascular caliber, but the alveolar end-expiratory pressure is higher than normal. This can increase the pressure gradient, resulting in air leaks and PM. Both mechanisms can occur simultaneously in which the alveoli are overdistended while the blood caliber is decreased. This will increase the pressure gradient significantly, inducing air leaks more easily. SPM was first described in 1819 by Laennec [8] and was further characterized in 1939 by Hamman [9] who reported a case series of patients presenting with SPM and SCE. Hamman then described the mediastinal crunching sound heard during auscultation over the cardiac apex and the left sternal border that is synchronous with the heartbeat, which is named the Hamman's sign [10].

Additionally, Muller noted the bubbling crepitations that occur with a heartbeat and the disappearance of the cardiac dullness. [11] Common presenting symptoms include chest pain (60%–100%), dyspnoea (75%), coughing spells (80%), neck pain (36%) or dysphagia, but some cases have no complaints or provoking factors. [12,13] In most cases, the pressure in the mediastinum is relieved by tracking of air to the subcutaneous tissue, resulting in SCE which is detected in 70% of patients with PM. [14] The most common site is at the root of the neck, resulting in crepitations. This is because the visceral layer of the deep cervical fascia is continuous with the mediastinum. Free air can also spread to the face, limbs, abdomen and perineum because of the connection among the facial planes. Several cases of upper and lower lung infections have been reported to cause air leaks, leading to SPM or PNX. These include fungal pneumonia, [15] Mycoplasma pneumonia, [16] staphylococcal pneumonia, [17] bronchiolitis obliterans organizing pneumonia [18] and critical pertussis. [19,20] In HIV infection, Pneumocystis carinii pneumonia and tuberculosis have been reported to cause PNX. [21] Viral respiratory infections can rarely cause SPM, PNX and SCE.

It has been reported with cytomegalovirus pneumonitis, [22] measles pneumonia [23] and influenza infections. [24-28] The pathophysiological reasons discussed in those cases follow the Macklin phenomenon. In a retrospective analysis of the SARS-CoV database, 13 patients developed SPM with or without PNX and SCE at a mean of 19.6±4.6 days from the symptom onset. [29] No patient had isolated PNX and none was related to the use of invasive or non-invasive positive pressure ventilation. Similarly, in another study, SP was diagnosed 23 days after the onset of symptoms. [30] Analyses also showed that high peak serum LDH level, which might signify cellular damage, was associated with the development of SPM. [31] In our study, SPM with or without PNX and SCE developed 16.55 (1-49) days after PCR test positivity. Consistent with previous studies, the incidence of PNX was higher in males (65%) [31-32]. On the other hand, concerning the laboratory parameters, there was a significant ($p<0.001$) increase in the leukocyte and platelet counts and D-dimer levels, while no significant change was detected in the lymphocyte count, CRP and Ferritin values at the time PNX were diagnosed. (Table 2) The pulmonary pathology of COVID-19 shows similarity to the one seen in SARS, showing diffuse alveolar damage, cellular fibromyositis exudates, evident desquamation of pneumocytes and hyaline membrane formation [33].

Furthermore, studies suggest that a dysregulation of the immune response seen with SARS-CoV- 2, SARS-CoV or MERS-CoV could be a cause of the significant lung injury causing an ARDS pattern. [34-35] In a case series of COVID-19 autopsies, it was found that the dominant process in all cases was diffuse alveolar damage with a mononuclear response around thrombosed small vessels. They suggest that the maladaptive immune response plays a significant role in severe COVID-19. This may suggest that the mechanism of air leak seen with COVID-19 could be related to significant alveolar damage, which makes the alveolar wall more prone to rupture. The rupture could be exacerbated by coughing or anything that increases the alveolar pressure. [36] According to the data of our study, although the majority of

the cases that developed PNX were detected after intubation (88%), it was determined that PNX developed in 16 cases (12%) while they were non- intubated (Table 1). Among these 16 cases, PNX was detected in 7 caseson admission, in 6 cases during follow up in ICU with just O_2 support and in 3 cases during non-ICU clinical follow up.

In this sense, although not receiving any invasive or non-invasive mechanical positive pressure support, development of PNX can be related to COVID-19 itself due to presumed pathophysiological mechanismsthat occur in pulmonary parenchyma and interstitium as mentioned above. The need for intensive care after the development of PNX is an important factor that increases mortality. As determined in our study, 94% of the cases required intensive care after the development of PNX, and after an average survival time of 9.9 days, the mortality rate of these cases was found to be 87.2%.

Limitations

There are several limitations of our study.

- Smoking is a risk factor for barotrauma, and PNX. Due to the retrospective design of the study, the smoking history of the whole study population could not be reached.
- The development of barotrauma may be caused by high pressures or inappropriate ventilatory modes. The ventilatory modes and the pressures were not recorded in our analyses.
- The use of corticosteroids for COVID-19 may be a confounding factor. The great majority of the cases have received corticosteroids, but our analyses did not include the medication records.
- The incidence of secondary bacterial infections has not been recorded.

Conclusion

An uncommon presentation of COVID-19 is the occurrence of SPM, PNX, and SCE. These unwanted clinical conditios may also develop during follow-up in the hospital or in the ICU. Mechanical ventilation is a major risk factor. As seen in the literature and our study, this is a wide spectrum, from a self-limiting condition that can be controlled with conservative treatments, to serious respiratory and circulatory pathologies and even death. For this reason, it is very important to question the symptoms, repeat the examination, laboratory and radiological imaging and closely monitor the patient who develops sudden deterioration, shortness of breath and a sudden increase in oxygen demand during clinical follow-up.

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