Spontaneous Pneumothorax and Pneumomediastinum in COVID-19 Pneumonia

COVID-19 Pnömonisinde Spontan Pnömotoraks ve Pnömomediastinum

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ABSTRACT

Introduction: Spontaneous pneumothorax and pneumomediastinum are uncommon complications of COVID-19 viral pneumonia and these complications remain unknown largely. This study aimed to determine the relationship between pneumothorax, pneumomediastinum, and COVID-19 prognosis.

Methods: Between March 2020 and January 2021, 82 COVID-19 (+) patients diagnosed with pneumothorax and pneumomediastinum were evaluated retrospectively. Data were obtained from the medical records of the patients, including demographic information, laboratory evaluations, radiological evaluations (PA lung, Thorax CT), clinical management, prognosis, and survival.

Results: While 74 (90.2%) of the patients had COVID-19 proven by the laboratory, 8 (9.8%) patients were diagnosed based on their clinical picture and computed tomography (CT) findings. Seventy-six patients (92.7%) had pneumothorax, while 10 (12.1%) had additional pneumomediastinum and 6 patients (7.3%) isolated pneumomediastinum. There was no significant difference in the median duration of pneumothorax based on the presence (median: 8.55, IQR: 13) days) or absence (median: 2.5, IQR: 10) of mechanical ventilation (Mann-Whitney U Z=1.548, p=0.122). Most of the inflammatory markers as well as blood gas values differed significantly between the deceased and survived patients ($p<0.05$). Age, treatment groups, and the presence of comorbidities were the significant variables associated with survival in univariate analyses. A multivariate analysis revealed pH and sex as the only significant independent predictors of survival.

Conclusion: Spontaneous pneumothorax and pneumomediastinum are rare complications of COVID-19 viral pneumonia. They can occur at any time during the course of the disease. In general, elderly patients with comorbidities who are exposed to mechanical ventilation seem to be at increased risk.

Key words: SARS-CoV-2, Pneumothorax, Pneumomediastinum, Complications, Mortality

ÖZET

Giriş: Spontan pnömotoraks ve pnömomediastinum, COVID-19 viral pnömonisinin nadir görülen komplikasyonlarıdır ve bu komplikasyonlar büyük ölçüde bilinmemektedir. Bu çalışmada pnömotoraks, pnömomediastinum ve COVID-19 prognozu arasındaki ilişkiyi belirlemeyi amaçladık.

Yöntemler: Mart 2020 ile Ocak 2021 arasında pnömotoraks ve pnömomediastinum tanısı alan 82 COVID-19 (+) hasta retrospektif olarak değerlendirildi. Demografik bilgiler, laboratuvar değerlendirmeleri, radyolojik değerlendirmeler (PA akciğer, Toraks BT), klinik yönetim, prognoz ve sağkalım dahil olmak üzere hastaların tıbbi kayıtlarından veriler elde edildi.

Bulgular: Hastaların 74'ünde (%90,2) laboratuvar tarafından kanıtlanmış COVID-19 bulunurken, 8 (%9,8) hastaya klinik tablo ve bilgisayarlı tomografi (BT) bulgularına göre tanı konuldu. Yetmiş altı hastada (%92,7) pnömotoraks, 10 hastada (%12,1) ek olarak pnömomediastinum varken ve 6 hastada (%7,3) izole pnömomediasten vardı. Pnömotoraks süresinde mekanik ventilasyon varlığına (medyan: 8,55, IQR: 13) gün) veya yokluğuna (medyan: 2,5, IQR: 10) göre istatiksel anlamlı bir fark yoktu (Mann-Whitney UZ=1,548, p= 0,122). İnflamatuvar belirteçlerin çoğu ve kan gazı değerleri, ölen ve hayatta kalan hastalar arasında önemli ölçüde farklılık gösterdi ($p<0,05$). Tek değişkenli analizde yaş, tedavi grupları ve komorbiditelerin varlığı sağkalım ile ilişkili önemli değişkenlerdi. Çok değişkenli analizlerde, pH ve cinsiyetin hayatta kalmanın tek önemli bağımsız belirleyicisi olduğunu ortaya çıkardı.

Sonuç: Spontan pnömotoraks ve pnömomediastinum, COVID-19 viral pnömonisinin nadir komplikasyonlarıdır. Hastalığın seyri sırasında herhangi bir zamanda ortaya çıkabilirler. Genel olarak, mekanik ventilasyona maruz kalan komorbiditeleri olan yaşlı hastalar artmış risk altında görünmektedir.

Anahtar Kelimeler: SARS-CoV-2; pnömotoraks; pnömomediastinum; komplikasyon; mortalite

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Eskisehir Med. J. 2022; 3(2):167-175.

Received date:16.03.2022 Accepted date:30.05.2022

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INTRODUCTION

Caused by the SARS-CoV-2 agent, the COVID-19 disease was first reported in Wuhan, China, in December 2019 and was declared by the World Health Organization as a viral pneumonia pandemic in March 2020 (1). Pleural effusion, lymphadenopathy, pericardial effusion, lung cavitation, CT halo sign, and pneumothorax are possible but rare findings during the disease course (2).

However, until recently, the relationship between COVID-19 and pneumothorax has been reported rarely, and therefore, information on the subject is minimal(3,4). Whereas, studies have revealed that COVID-19 shares more than 88% homology with the coronaviruses associated with the severe acute respiratory syndrome (SARS) from bats. Additionally, in the SARS epidemic study in 2003, spontaneous pneumothorax was observed in 1.7% of critically ill patients(5). In support of this, autopsy studies have shown that patients who died due to SARS-CoV-2 (COVID-19) showed diffuse alveolar damage at different stages and high frequency of macro and microvascular thrombosis, which were held responsible for the ARDS findings(6). These structural changes in the lung parenchyma are thought to be related to the spontaneous pneumothorax mechanism. Over time, increased intrathoracic pressure, particularly from prolonged coughing and/or mechanical ventilation, produces cystic and fibrotic changes that lead to alveolar tears, which in turn lead to pneumothorax formation(7).

Invasive mechanical ventilation (MV) was needed in 22% of patients treated for COVID-19 at the University of New York (8). Therefore, this exposes COVID-19 patients to high barotrauma (9). In this study, we hypothesized that pneumothorax is more common among COVID-19 patients, and thus, a factor associated with worse prognosis.

This study aimed to determine the relationship between pneumothorax, pneumomediastinum and COVID-19 prognosis in a large case series from two centers serving as pandemic hospitals in Turkey.

METHODS

Study Design

A cross-sectional descriptive study was planned. The study was approved by Bursa City Hospital Clinical Research Ethics Committee (IRB date: 07.04.2021 - Number: 2021-6/12).

Participants

Patients diagnosed with COVID-19 at Bursa City Hospital and Canakkale Onsekiz Mart University Faculty of Medicine were evaluated retrospectively. Between March 2020 and January 2021, 16.838 patients were diagnosed with COVID-19. 82 (0.48%) COVID-19 (+) patients diagnosed with pneumothorax and pneumomediastinum were evaluated retrospectively.

Diagnosis of the patients was made based on polymerase chain reaction (PCR) tests (RT-PCR[Biospeedy®]) of the nasopharyngeal swab samples supported by thorax CT (Toshiba Asteion Super 4 Slice CT Scanner). Those with positive CT chest findings were later verified with PCR. Thorax CT findings of coronavirus pneumonia at the time of diagnosis and during the follow-up are well defined (2,10). The characteristic features are bilateral multilobar ground-glass opacification, especially in the lower lobes, with a peripheral or posterior dominant distribution (2,10–12). Thirteen patients were excluded due to missing data (Figure 1).The patients included in the study were followed up for at least 30 days. No underlying bulla, c yst, or severe emphysema was observed in the anamnesis and/or pneumothorax or first CT scan examinations of our patients.

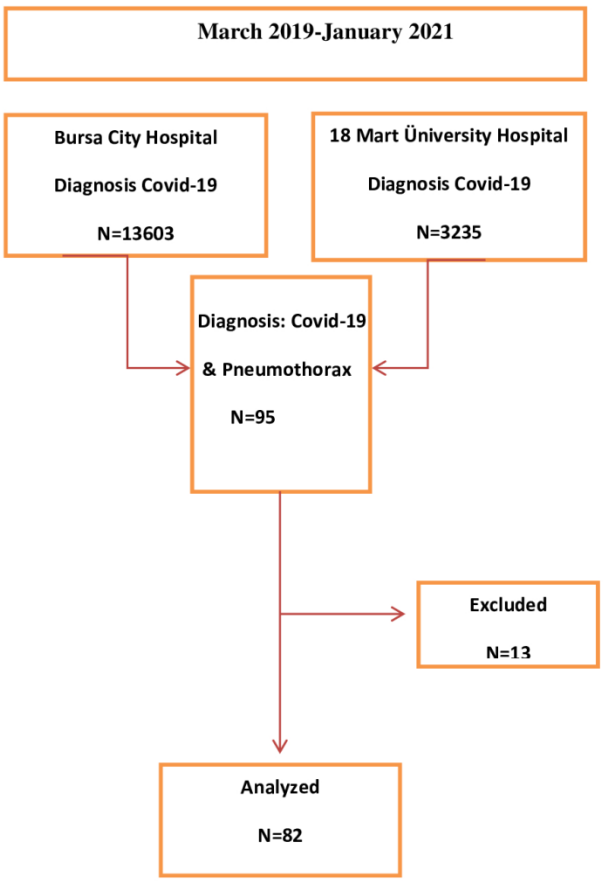

Figure 1. Participant flow diagram

Variables

Data were obtained from the medical records of the patients, including demographic information, comorbidities, laboratory evaluations (complete blood count, blood gases, D-dimer, C-reactive protein (CRP), LDH, Ferritin, IL-6), radiological evaluations (PA lung, Thorax CT), clinical management, prognosis, and survival.

All patients with tube thoracostomy were supported with closed underwater drainage systems (CUDS) ports high-efficiency particulate air (HEPA) filters.

Statistical Analysis

The data were analyzed using the Statistical Package for the Social Sciences (SPSS) version 25.0 software (SPSS Inc., Chicago, IL, USA). The results were presented as frequencies, percentages, means, medians, standard deviations (SD), and interquartile range (IQR). The Kolmogorov–Smirnov test was performed to test if the numerical variables were normally distributed. The independent samples t-test was used to compare data meeting parametric assumptions. The Mann-Whitney *U* test was used for skewed variables, and the Chi-Square test (or Fisher's exact test) was used for categorical variables. The Cox regression analysis was used to check for significant factors affecting survival. A p-value of <0.05 was considered statistically significant.

RESULTS

Of the participants, 72% (n=59) were males. The most common comorbidities were combined hypertension-diabetes and Alzheimer's disease. Seventy-six patients (92.7%) had pneumothorax, while 10 (12.1%) had additional pneumomediastinum and 6 patients (7.3%) isolated pneumomediastinum. (Table 1, Figure 2). While tube thoracostomy was applied to 73 (96%) patients, 3 (4%) patients were followed conservatively without surgical intervention. The median duration of hospitalization was 17 days (min. 1, max. 181 days).

While 53 (91.4%) of the patients under mechanical ventilation (MV) had pneumothorax, 23 (95.8%) of those without MV had this complication. There was no significant difference in the pneumothorax proportions of patients with and without mechanical ventilation (Fisher's exact test Chi-Square=0.497, p=0.666). Also, there was no significant difference between men (93.2%, n=55) and women (91.3%, n=21) concerning pneumothorax proportions (Fisher's exact test Chi-Square=0.090, p=1.000). There was no significant difference in the median duration (IQR) of pneumothorax based on the presence (median: 8.55, IQR: 13) days) or absence (median: 2.5, IQR: 10) of mechanical ventilation (Mann-Whitney U Z=1.548, p=0.122).

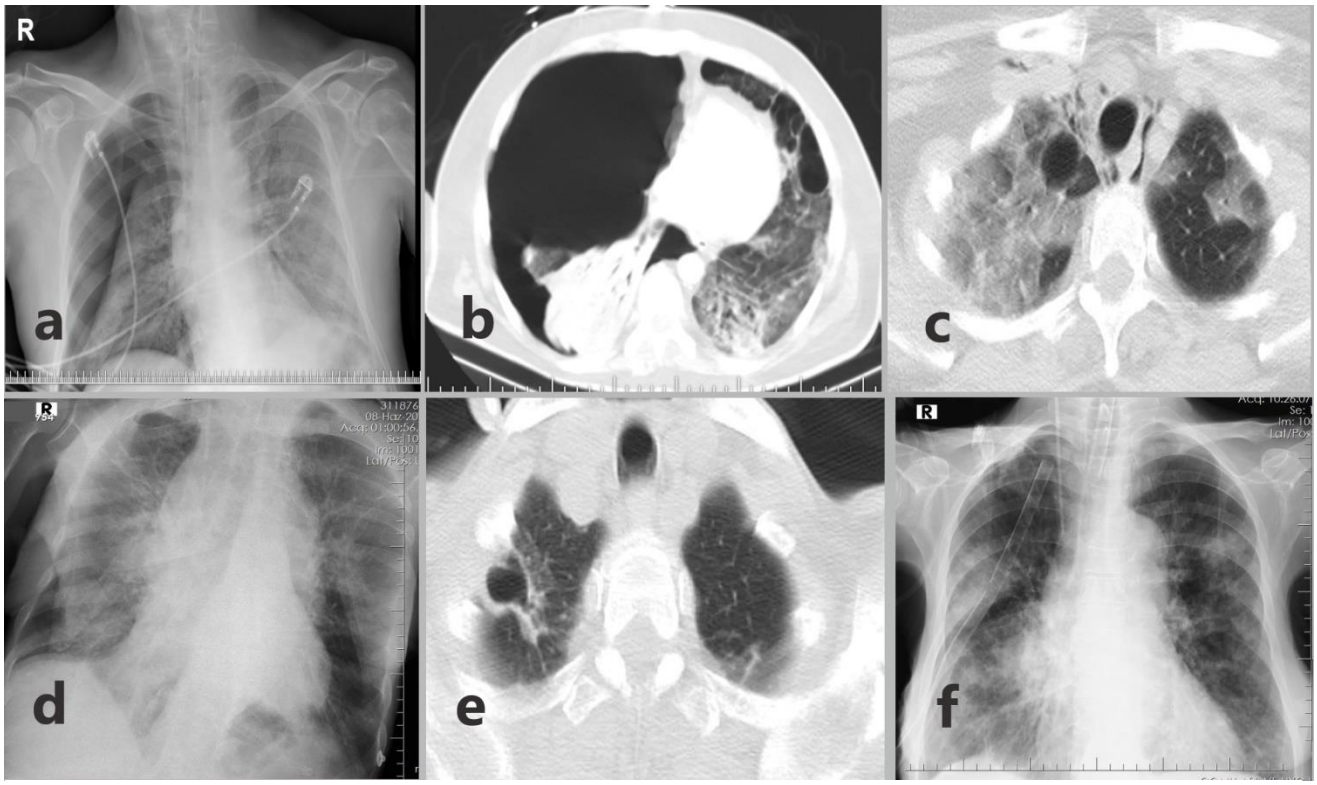

Figure 2. Figure 2a: chest radiograph (anteroposterior projection)-Large Right-sided pneumothorax Figure 2b: CT Chest- Right pneumothorax with atelectasis of right lower lobe and Left-sided pneumatocele/bullae seen with associated left lower lobe ground glass opacities. Figure 2c: Chest CT- showing pneumomediastinum and Right-sided pneumatocele/bullae seen with associated bilateral superior lobe ground glass opacities. Figure 2d: chest radiograph (anteroposterior projection): a female is her fifty-five presenting with a right pneumothorax. Figure 2e: CT Chest-Right-sided pneumatocele/bullae seen (before pneumothorax). Figure 2f: chest radiograph (anteroposterior projection) after pneumothorax developed and chest tubes were placed.

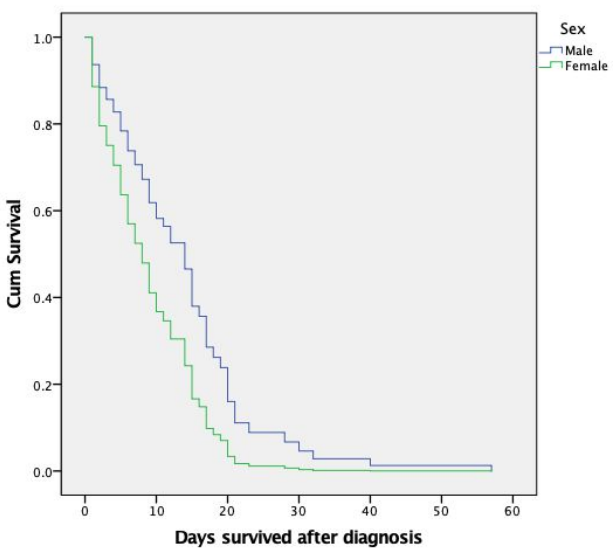

Figure 3. Comparison of the survival curves between males and females. (Sex: p=0.059, pH: p=0.030)

While 74 (90.2%) of the patients had COVID-19 proven by the laboratory, 8 (9.8%) patients were diagnosed based on their clinical picture and computed tomography (CT) findings. Age, treatment groups, and the presence of comorbidities were the significant variables associated with survival in univariate analyses (Table 2).

Most of the inflammatory markers (WBC, CRP, D-Dimer, Ferritin, and IL6) as well as blood gas values ($PaCO_2$ and pH) differed significantly between the deceased and survived patients (Table 3).

The median survival time was 10 days (95% CI: 6.3-13.6). A Cox regression model was bu ilt including variables with <0.1 significance levels. Due to sample size concerns, only D-dimer from the inflammatory markers and pH was added to the model, which

revealed pH (p=0.030, HR= 0.202) and sex (p=0.059, HR= 1.852) (borderline significant) as the only significant independent predictors of survival (Figure 3).

Table 1. Demographic information and clinical characteristics of the patients

		n	%
Sex	Male	59	72.0
	Female	23	28.0
Comorbidity	None	32	39.0
	Hypertension	8	9.8
	Diabetes	7	8.5
	Heart disease	5	6.1
	Alzheimer	11	13.4
	Cerebrovascular accident	2	2.4
	Hypertension and diabetes	13	15.9
	Other	4	4.8
Additional respiratory disease	None	64	78.0
	Asthma	2	2.4
	COPD	8	9.8
	Emphysema	3	3.7
	Lung cancer	4	4.9
	Bronchiectasis	0	0.0
	Tuberculosis	1	1.2
Place of diagnosis	Outpatient	5	6.1
	Transfer to another clinic	20	24.4
	Intensive care unit	57	69.5
Pneumothorax	Yes	76	92.7
	No	6	7.3
Pneumothorax side	Right	38	50.0
	Left	30	39.5
	Bilateral	8	10.5
Pneumomediastinum	Yes	6	7.3
	No	76	92.7
Pneumothorax + Pneumomediastinum	Yes	10	12.1
	No	72	87.9
Treatment	Spontaneous	2	2.4
	Nasal O_2	21	25.6
	MV	58	70.7
	CPAP	1	1.2
Subcutaneous emphysema	Yes	21	25.6
	No	61	74.4
Outcome	Died	55	67.1
	Alive	27	32.9

DISCUSSION

The human coronavirus is one of the main pathogens of respiratory tract infection (13). While most people infected with the virus (approximately 80%) have no or only mild symptoms, some cases may require hospitalization and mechanical ventilation, and in some patients with severe respiratory failure, such a course is associated with high mortality (14).

It has been reported that structural cystic and fibrotic changes in the lung parenchyma that occur early during the course of COVID-19 may cause a vulnerability for pneumothorax (3). This may cause pneumothorax to develop in approximately 1% of COVID-19 patients (15). In our study, the pneumothorax developed in approximately 0.45% of COVİD-19 patients.

Pneumothorax is a potentially fatal complication in patients with ARDS. The incidence varies greatly from 1.7% to 10%, especially if exposed to mechanical ventilation (16). ARDS-pneumothorax coexistence is particularly high with high peak inspiratory pressures (PIP), high positive end-expiratory pressure (PEEP), high tidal volumes, and minute ventilation situations (17), which is usually associated with barotrauma caused by high airway pressures (3). In a study conducted in the USA, 89 of 601 COVID-19 patients (15%) barotrauma were observed due to invasive mechanical ventilation. Particularly, the frequency of pneumothorax in ARDS patients who underwent mechanical ventilation was significantly higher than in the pre-COVID-19 period (9). In our study, there was no significant difference in the pneumothorax proportions of patients with and without mechanical ventilation.

Pulmonary cystic lesions and pneumothorax observed in the course of the disease can also be seen in patients who have not been exposed to mechanical ventilation (18). While pneumothorax develops in some patients with COVID-19 in relation to risk factors such as mechanical ventilation, the only factor in others can be viral pneumonia itself (19).

This clearly shows that barotrauma alone cannot cause cyst formation. The fact that we reported a large number of pneumothorax cases not exposed to mechanical ventilation in our study supports this view.

However, we consider that COVID-19 may cause vulnerability to barotrauma. For this reason, we believe that lower airway pressures should be targeted in these patients. It is also important to note tha t to this date, there are no clear guidelines on the timing and settings of mechanical ventilation in patients with COVID-19 pneumonia.

Table 2. Univariate comparison of the relationship of the studied factors with survival.

		Died		Survived			
		n/Mean/ Median	%/SD/ IQR	n/Mean /Median	%/SD/ IQR	Test	p
Age (years)	(mean±SD)	68.4	12.6	58.7	14.6	3.092	**0.003**[*]
	(median, IQR)	69.5	16.0	57.0	16.0		
Length of hospital stay (days)	(mean±SD)	20.2	13.4	27.7	33.8	0.681	0.496[#]
	(median, IQR)	17.0	19.0	18.0	25.0		
Pneumothorax duration (days)	(mean±SD)	10.2	9.2	8.4	12.3	1.608	0.108[#]
	(median, IQR)	10.0	13.0	4.0	12.0		
Duration to tube removal (days)	(mean±SD)	6.6	7.1	4.9	5.7	0.952	0.341[#]
	(median, IQR)	5.0	9.0	4.0	8.0		
Sex (n, %)	Male	36	61.0	23	39.0	3.493	0.062[&]
	Female	19	82.6	4	17.4		
Pneumothorax (n, %)	Yes	52	68.4	24	31.6	0.854	0.390[$]
	No	3	50.0	3	50.0		
Pneumothorax+ pneumomediastinum (n, %)	Yes	6	60.0	4	40.0	0.258	0.723[$]
	No	49	68.1	23	31.9		
Pneumothorax side (n, %)	Right	29	76.3	9	23.7	3.175	0.204[&]
	Left	17	56.7	13	43.3		
	Bilateral	6	75.0	2	25.0		
Treatment (n, %)	Other	7	29.2	17	70.8	22.076	**<0.001**[$]
	MV	48	82.8	10	17.2		
Subcutaneous emphysema (n, %)	Yes	15	71.4	6	28.6	0.242	0.622[&]
	No	40	65.6	21	34.4		
İsolated Pneumomediastinum (n, %)	Yes	5	83.3	1	16.7	1.054	0.304[&]
	No	50	66.0	26	34.0		
Pleural effusion (n, %)	Yes	4	80.0	1	20.0	0.403	1.000[$]
	No	51	66.2	26	33.8		
Additional respiratory disease (n, %)	No	43	67.2	21	32.8	0.002	0.967[&]
	Yes	12	66.7	6	33.3		
Comorbidity (n, %)	No	16	50.0	16	50.0	6.927	**0.008**[&]
	Yes	39	78.0	11	22.0		

[*]Independent samples t-test. [#]Mann-Whitney U test. [&]Chi-Square test. [$]Fisher's exact test. MV: Mechanical ventilation.

We have given advice on the use of low volume and pressure strategies as lung protective strategies for branches other than anesthesia working in the intensive care unit, especially during peak periods. High-flow oxygen therapy may be a safer alte rnative to avoid potential complications of mechanical ventilation in

Table 3. Comparison of inflammatory markers and blood gas values with survival status.

	Died				Survived					
	Mean	SD	Median	IQR	Mean	SD	Median	IQR	Test	p
WBC	13.3	5.3	13.5	8.8	10.9	3.8	11.0	5.5	2.058	**0.043**[*]
Lymphocytes	1.2	1.1	0.8	0.7	1.4	0.9	1.1	0.6	0.872	0.386[*]
Neutrophils	24.7	26.7	14.5	6.6	24.9	28.6	13.2	6.0	1.896	0.058[#]
CRP	75.1	69.1	45.6	96.5	31.4	36.3	17.0	34.4	2.975	**0.003**[#]
LDH	468.8	178.3	441.0	236.0	356.0	135.4	350.0	156.0	2.898	**0.005**[*]
D-Dimer	23.5	152.9	2.8	2.1	2.1	1.1	2.0	1.5	2.655	**0.008**[#]
Ferritin	816.8	477.8	778	578.0	546.2	414.0	404.0	348.0	2.514	**0.014**[*]
IL6	24.4	18.0	21.2	32.4	10.4	15.0	5.8	4.7	3.331	**0.001**[#]
PaO_2	71.2	27.2	66.4	34.8	74.4	27.1	76.6	36.3	0.497	0.620[*]
$PaCO_2$	61.6	20.9	57.5	20.1	45.5	14.7	43.3	13.2	3.575	**0.001**[*]
HCO_3	25.8	7.9	26.3	9.6	24.5	5.4	22.5	8.1	0.771	0.443[*]
SaO_2	81.4	16.0	86.5	22.4	87.2	13.9	93.8	12.6	1.589	0.116[*]
pH	7.2	0.2	7.2	0.2	7.4	0.11	7.4	0.1	3.963	**<0.001**[#]

*Independent samples t-test. #Mann-Whitney U test.

these patients. In our study, mortality was significantly higher in pneumothorax cases, especially in the mechanical ventilation-dependent patient group.

Although Martinelli et al. found a higher incidence of pneumothorax in males (3), we found no significant difference between the sexes in terms of pneumothorax in our study. Nevertheless, contrary to Martinelli et al, found survival no significant difference between the sex (3). We found borderline significantly higher survival in female patients.

Although PA chest radiography was used in the initial evaluation of all our patients, we considered that though rare, cystic parenchymal changes can easily be overlooked in standard chest x-rays. Due to the sensitivity and ease of use of these features, which are important radiological manifestations of COVID-19, thorax CT is an essential screening tool for patients with suspected COVID-19 (20). For this reason, we used Thorax CT in every patient without exception, not only for diagnosis but also for monitoring the progression of the disease and shaping the medical treatment.

The history of pneumothorax and/or lung disease was low in our study. Zanta et al. reported the presence of lymphopenia and high inflammatory markers, including CRP, LDH, Ferritin, D-dimer, and IL-6 levels in almost all patients who developed spontaneous pneumothorax, as an important finding in their study (21). In our study, most of the inflammatory markers (WBC, CRP, D-Dimer, Ferritin, and IL6) differed significantly between the deceased and survived patients. The results were similar to those reported by Zanta et al.

Mediastinal emphysema occurs as a result of a sudden increase in alveolar pressure, alveolar rupture, and air leakage with interstitial emphysema (22). Mediastinal emphysema was also observed in some of our cases, especially due to the high pressure caused by mechanical ventilation. However, Wang et al. reported spontaneous pneumothorax, pneumomediastinum, and

Table 4. Cox regression analysis computer output showing variables predicting survival.

	B	Wald	p	Exp(B)	95.0% CI for Exp(B) Lower	Upper
Age	0.021	2.742	0.098	1.021	0.996	1.047
Sex (Women vs. Men)	0.616	3.568	**0.059**	1.852	0.977	3.510
Comorbidity (Present vs. Absent)	-0.411	1.154	0.283	0.663	0.313	1.403
MV vs. no MV	0.636	2.263	0.133	1.890	0.825	4.331
pH	-1.597	4.688	**0.030**	0.202	0.048	0.860
D-Dimer	-0.001	0.765	0.382	0.999	0.997	1.001

MV: Mechanical ventilation

subcutaneous emphysema in a patient who was not mechanically ventilated (23).

The survival rate was lower, especially in intubated cases. While mortality was 82.8% in intubated patients, this rate was 29.2% in other patients. Imam et al. reported that advanced age and an increasing number of co-morbidities were independent predictors of hospital mortality among COVID-19 patients (24). In our study, age, treatment groups, and presence of comorbidities were important variables associated with survival in univariate analyzes.

Filice et al. (25) reported that long-term air leakage developed in all SARS patients who underwent tube thoracostomy, and the improvement of air leaks took 14 to 31 days (mean 23.5 days). In our study, the mean leakage duration in patients who underwent tube thoracostomy was 10.6 days. From this point of view, long-term air leakage seems predictable due to lung parenchyma inflammation and damage. Severe pneumonic conditions may also preclude the patient's chances for surgical treatment.

Negative aspiration was applied to the Gomco® suction device in cases of especially advanced lung involvement who had no expansion after tube thoracostomy, and the lung was kept as expanded as possible, which we think has a positive effect on our treatment.

Interestingly, none of our cases progressed, requiring operative intervention. In parallel with the patients who responded to the treatment, sometimes late improvements in pneumothorax were observed. This means that the symptoms and treatment modalities of COVID-19 pneumonia with pneumothorax must be handled with absolute care.

Limitations

Since our study was limited to two centers, we cannot generalize our comments regarding the incidence of pneumothorax. The fact that our data were obtained from retrospectively evaluated hospital records carries the limitations of retrospective studies.

CONCLUSION

Spontaneous pneumothorax and pneumomediastinum are rare complications of COVID-19 viral pneumonia. It can occur at any time during the course of the disease. In general, patients with elderly and comorbidities and exposed to mechanical ventilation seem to be at increased mortality risk.

High inflammatory markers (WBC, CRP, LDH, D-Dimer, Ferritin, and IL6) and blood gas values (high PaCO2 and low pH) differed significantly between deceased and surviving patients.

Clinicians should be as active and careful as possible in the diagnosis and treatment of this complication. Therefore, early diagnosis and timely treatment of COVID-19 complications can increase the therapeutic effect and reduce mortality.

Conflict of Interest: The Authors state that they have no conflicts of interest in regard to this study.

Financial Disclosure: Any company or institution has not financially contributed to the study.

REFERENCES

1.World Health Organization. Available from: https://www.who.int/docs/default-source/coronaviruse/situation-reports/20200531- covid-19-sitrep-132.pdf? Sfvrsn=d9c2eaef_2. . Accessed: November 10, 2020.

2.Salehi S, Abedi A, Balakrishnan S, Gholamrezanezhad A. Coronavirus disease 2019 (COVID-19): A systematic review of imaging findings in 919 patients. AJR Am J Roentgenol .2020;215(1):87-93

3.Martinelli AW, Ingle T, Newman J, et al. COVID-19 and pneumothorax: A multicentre retrospective case series. Eur Respir J. 2020;56(5).

4.Flower L, Carter JPL, Rosales Lopez J, Henry AM. Tension pneumothorax in a patient with COVID-19. BMJ Case Rep. 2020;13(5): e235861.

5.Lu R, Zhao X, Li J, et al. Genomic characterisation and epidemiology of 2019 novel coronavirus: implications for virus origins and receptor binding. Lancet. 2020;395(10224):565–74.

6.De Michele S, Sun Y, Yilmaz MM, et al. Forty Postmortem Examinations in COVID-19 Patients. Am J Clin Pathol. 2020;154(6):748–60.

7.Wang W, Gao R, Zheng Y, Jiang L. COVID-19 with spontaneous pneumothorax, pneumomediastinum and subcutaneous emphysema. J Travel Med. 2020;27(5).

8.Petrilli CM, Jones SA, Yang J, et al. Factors associated with hospital admission and critical illness among 5279 people with coronavirus disease 2019 in New York City: Prospective cohort study. BMJ. 2020;369.

9.McGuinness G, Zhan C, Rosenberg N, et al. Increased incidence of barotrauma in patients with COVID-19 on invasive mechanical ventilation. Radiology. 2020;297(2): E252–62.

10.Ai T, Yang Z, Hou H, et al. Correlation of Chest CT and RT-PCR Testing for Coronavirus Disease 2019 (COVID-19) in China: A Report of 1014 Cases. Radiology. 2020;296(2): E32–40.

11.Sun R, Liu H, Wang X. Mediastinal emphysema, giant bulla, and pneumothorax developed during the course of COVID-19 Pneumonia. Korean J Radiol. 2020;21(5):541–4.

12.Huang C, Wang Y, Li X, et al. Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. Lancet. 2020;395(10223):497–506.

13.Hu B, Zeng LP, Yang X Lou, et al. Discovery of a rich gene pool of bat SARS-related coronaviruses provides new insights into the origin of SARS coronavirus. PLoS Pathog . 2017;13(11):e1006698.

14.Wang D, Hu B, Hu C, et al. Clinical Characteristics of 138 Hospitalized Patients with 2019 Novel Coronavirus-Infected Pneumonia in Wuhan, China. JAMA - J Am Med Assoc. 2020;323(11):1061–9.

15.Chen N, Zhou M, Dong X, 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.

16.Sihoe ADL, Wong RHL, Lee ATH, et al. Severe acute respiratory syndrome complicated by spontaneous pneumothorax. Chest. 2004;125(6):2345–51.

17.Ioannidis G, Lazaridis G, Baka S, et al. Barotrauma and pneumothorax. J Thorac Dis. 2015;7(Suppl 1):S38-43.

18.Sheard S, Rao P, Devaraj A. Imaging of acute respiratory distress syndrome. Respir Care . 2012;57(4):607-12.

19.Ucpinar BA, Sahin C, Yanc U. Spontaneous pneumothorax and subcutaneous emphysema in COVID-19 patient: Case report. J Infect Public Health.2020;13(6):887–9.

20.Lin X, Gong Z, Xiao Z, Xiong J, Fan B, Liu J. Novel coronavirus pneumonia outbreak in 2019: Computed tomographic findings in two cases. Korean J Radiol. 2020;21(3):365–8.

21.Zantah M, Dominguez Castillo E, Townsend R, Dikengil F, Criner GJ. Pneumothorax in COVID-19 disease- incidence and clinical characteristics. Respir Res. 2020;21(1):236.

22.Park SJ, Park JY, Jung J, Park SY. Clinical manifestations of spontaneous pneumomediastinum. Korean J Thorac Cardiovasc Surg. 2016;49(4):287–91.

23.Wang W, Gao R, Zheng Y, Jiang L. COVID-19 with spontaneous pneumothorax, pneumomediastinum and subcutaneous emphysema. J Travel Med . 2020;27(5):taaa062.

24.Imam Z, Odish F, Gill I, et al. Older age and comorbidity are independent mortality predictors in a large cohort of 1305 COVID-19 patients in Michigan, United States. J Intern M ed. 2020;288(4):469–76.

25.Filice GA. SARS, pneumothorax, and our response to epidemics. Chest . 2004;125(6):1982-4.

Cite as: Pekcolaklar A, Kapicibasi HO. Spontaneous Pneumothorax and Pneumomediastinum in COVID-19 Pneumonia. Eskisehir Med J. 2022;3(2):167-175.