Evaluation of Pulmonary Function Test in Post-COVID-19 Patients

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ABSTRACT

Background and Objectives: SARS-CoV-2 emerged, leading to a global pandemic with significant morbidity and mortality. Lung injury is prevalent, raising concerns about long-term complications. Current data indicate persistent pulmonary function issues, particularly diffusion capacity, in survivors. Critical care needs and risk factors for poor outcomes are identified, but the full impact of post-COVID syndromes remains inadequately defined. Further research is essential to understand the long-term consequences of COVID-19. This study aims to evaluate pulmonary function tests in post-COVID-19 patients.

Methods: A quantitative study was conducted on 74 post-COVID-19 patients in Erbil, Iraq, evaluating demographic and clinical characteristics. Data were collected via questionnaires and analyzed using SPSS, ensuring participant confidentiality and voluntary participation throughout the research process.

Results: The study analyzed 74 post-COVID-19 patients, revealing demographics such as 55.4% aged 54-69 and a male predominance (63.5%). Common symptoms included cough (66.2%) and shortness of breath (73%). Half had high systolic blood pressure, and significant associations were found between severity and factors like age, SPO2, and pulmonary function tests.

Conclusion: Spirometry patterns significantly correlate with the severity of post-COVID-19 symptoms, suggesting their value as prognostic markers. However, the study found no significant link between spirometry patterns and comorbidities, indicating a need for further research to explore these relationships and their implications for patient management.

Keywords: Pulmonary Function Test; Spirometry Patterns; Post-COVID-19; SARS-CoV-2.

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INTRODUCTION

In late 2019, a novel coronavirus, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), emerged in Wuhan, China, and has since spread globally, infecting more than 200 million people. The clinical course of infection appears to be extremely variable, from asymptomatic to severe pneumonia with multiorgan failure requiring critical care. More than 1,122,036 people are known to have died following infection, but data on morbidity in survivors are scarce. Lung injury is a predominant feature of acute SARS-CoV-2 infection, and understanding the longer-term implications is critical given the number of affected patients. There is currently little known about the post-infectious long-term complications from the severe acute respiratory syndrome coronavirus two (SARS-CoV-2), with much extrapolated from severe acute respiratory syndrome (SARS) and Middle East Respiratory Syndrome (MERS) pandemics during the 2003 and 2012, respectively (Zhao. et al, 2020; Ngia, et al., 2010).The extrapolated data has shown that there are long-term reductions in pulmonary function, as measured by Pulmonary Function Testing (PFT), most significantly for Diffusion Capacity for Carbon Monoxide (DLCO) for up to two years after infection (Hui. et al., 2005; Ong. et al., 2004; Park, Jun, and Kim, 2018) [3,4,5,6,7]. Approximately 1 in 7 hospitalized patients with COVID-19 in the United Kingdom required critical care admission, the majority for management of ARDS (Drake. et al., 2021). The onset of COVID-ARDS in the second week post-symptom-onset (at declining viral loads) and response to immunomodulation suggests pathogenic immune dysregulation. Given the unprecedented scale of the COVID-19 pandemic, even a low event rate may have a significant population-level impact (morbidity, late mortality). Most descriptions of post-COVID syndromes stipulate symptom duration for > 3 months (Munblit, O'Hara, Akrami, Perego, Olliaro, Needham, 2022). Clinically significant ILD refers to > 10% lung parenchymal changes on chest Computed Tomography (CT). A consensus definition and the true burden of Post-Covid19-ILD have not yet been determined. PC-ILD should be considered in patients with persistent respiratory symptoms (e.g., cough and dyspnea) 3 months post-COVID-19 symptom-onset, and patients with > 10% CT changes should be monitored. Accumulating data suggests that while the majority of scans show improvement, at 12 months the prevalence of (non-progressive) fibrosis is ~ 10% in hospitalized patients, particularly in severe disease and older age (Ong. et al., 2004). Several clinical features and comorbidities are associated with a poor prognosis and a higher risk of mortality from COVID-19. These include pre-existing health problems such as hypertension, diabetes, cardiovascular disease, obesity, cancer, chronic kidney, liver, and lung diseases, and older age, male sex, smoking, and race (Richardson. et al., 2020; Docherty. et al., 1985; Sze. et al., 2020; Sanchez-Ramirez and Mackey, 2020). Whether these risk factors are also predictors of longer-term outcomes from COVID-19 is currently unclear.The most common radiological pattern of acute infection with SARS-CoV-2 is of bilateral ground-glass opacification with or without consolidation in a subpleural distribution, and a radiological and histological pattern of organizing pneumonia pattern is described in many cases (Zhao, Zhong, Xie, Yu, and Liu, 2020). Radiological findings alter as the disease progresses, but persistent computed tomographic (CT) imaging abnormalities beyond Day 14 of symptoms and up to Day 37 have been reported (Pan. et al., 2020; Wang.

et al., 2020). However, no data exist as to the natural history of inflammatory infiltrates during recovery from SARS-CoV-2 or the utility of any treatment in patients with persistent inflammatory Interstitial Lung Disease (ILD) following infection with coronavirus. However, corticosteroids are the mainstay of treatment for organizing pneumonia of other causes (Cordier, 2000), and when used acutely in the management of acute respiratory distress syndrome (ARDS) caused by SARS-CoV-2 they have been associated with a reduction in mortality (Horby. et al., 2020; Sterne. et al., 2020). Whether PC-ILD should be divided into binary "inflammatory" and "fibrotic" categories based on radiological patterns remains controversial, given the ambiguity of some features (e.g., irregular lines), absence of histological correlates, uncertain course, and likelihood of reversibility. Persistent ground-glass changes may indicate fine/immature fibrosis rather than inflammation, and fibrotic-like changes may be capable of regression and remodeling, albeit at a slower rate. Idiopathic pulmonary fibrosis (IPF) is the archetypal fibrotic ILD (f-ILD), but most existing ILD syndromes are thought to reside on an overlapping fibro-inflammatory spectrum, e.g., hypersensitivity pneumonitis, an exaggerated aeroantigen-induced immune response, and rheumatic-associated ILD. This arbitrary stratification into predominant fibrotic or inflammatory phenotypes may have therapeutic and prognostic implications (although targeted treatments may be used concurrently), with potential extrapolation to PC-IL (Mehta, Ivan, Rosas, and Singer, 2022).

METHODS

A quantitative, descriptive study design was conducted on post-COVID-19 patients at the medical wards in Rizgary and Hawler Teaching Hospitals in Erbil City in the Kurdistan Region of Iraq. The researchers obtained permission from the Scientific and Ethical Committees at the College of Medicine, Hawler Medical University. on 4th June 2022. The other official permission from the Erbil Directorate of Health and the administrative units of the teaching hospitals was obtained. This study includes 74 patients. A sample of post-COVID-19 patients was recruited in this study based on a convenience (purposeful) sampling technique, according to the inclusion and exclusion criteria. Ages more than 18 years, both genders, with good communication were included. This study was carried out from November 2022 to April 2023. The duration of the data collection was around four months. A questionnaire was designed, and it consisted of two main parts. Part one included Demographic and Clinical Characteristics. Part two included laboratory and non-laboratory investigations of SBP, DBP, SPO2, PR, Temp, PMH, CRP, D. Dimer, LDH, WBCs, Pulmonary Function Tests, Forced Vital Capacity, and the ratio of FEV1/FVC. The data was collected through a direct interview method (face-to-face) with the patients. Informal oral consent was obtained from each participant. The researchers promised to keep the participant's information confidential and use this data for this study only, then explained the purpose of this study to each participant. In addition to the above, the researchers told each participant that this is voluntary work, and they could leave at any time even if the process is not completed. The data was analyzed through statistical software (Statistical Package for Science Service-SPSS V.26) which includes descriptive statistical analysis as frequency and percentage and inferential statistical analysis including Pearson Correlation, Chi-square, and Fisher's Exact tests. The P-value is considered significant if it is less than or equal to 0.05.

RESULTS

Table 1 shows the demographic and clinical characteristics of the post-COVID-19 patients. Regarding the age group, more than half of the study sample in the age group between 54-69 years old (55.4%) and others are between 22-37 and 38-53 years old (17.6% and 27% respectively). About 63.5% were male and 36.5% female. Regarding the symptoms, 66.2% had a cough, 32.4% had a fever, and 73% had a shortness of breathing. Concerning the past medical history, the highest percentage have other diseases (37.8%), while 6.8% of the participants have COPD and only 2.7% have Asthma, the rest of them have no past medical history (52.7%). About 66.2% were non-smokers, the smokers were 10.8% and ex-smokers 21.6% with only one case of passive smoker.

Table 1: Demographic and Clinical Characteristics

Demographic and Clinical Characteristics		F.	(%)
Age Groups (years)	22-37	13	(17.6)
	38-53	20	(27)
	54-69	41	(55.4)
Sex	Male	47	(63.5)
	Female	27	(36.5)
Cough	No	25	(33.8)
	Yes	49	(66.2)
Fever	No	50	(67.6)
	Yes	24	(32.4)
Shortness of Breath	No	20	(27)
	Yes	54	(73)
Others	No	13	(17.6)
	Yes	61	(82.4)
Past Medical	None	39	(52.7)
History	Asthma	2	(2.7)
	COPD	5	(6.8)
	Others	28	(37.8)
Smoking	No Smoker	49	(66.2)
	Smoker	8	(10.8)
	Ex-Smoker	16	(21.6)
	Passive-Smoker	1	(1.4)

Concerning blood pressure, half of the patients have high systolic blood pressure (50%) and only 35.1% have high diastolic blood pressure. About 81.1% of COVID-19 patients suffer from hypoxemia. The highest percentage of the study samples have tachycardia (64.9%) with normal body temperature (40.5%) and only 28.4% have hypothermia.

The highest percentage of the study sample have high CRP (77%), high D. Dimer (62.2%), normal LDH (48.6%), normal WBC (66.2%), low Forced Expiratory Volume in one second (58.1%), low Forced Vital Capacity (63.5%), more than normal ratio of FEV1/FVC (98.6%), with normal pattern (71.6%).

Table 2: Laboratory and non-laboratory investigations of post-COVID-19 patients

Laboratory and Non-laboratory Investigations		F.	(%)
SBP	Low Systolic Blood Pressure	4	(5.4)
	Normal Systolic Blood Pressure	33	(44.6)
	High Systolic Blood Pressure	37	(50)
DBP	Low Diastolic Blood Pressure	4	(5.4)
	Normal Diastolic Blood Pressure	44	(59.5)
	High Diastolic Blood Pressure	26	(35.1)
SPO2	Hypoxemia	60	(81.1)
	Normal SPO2	14	(18.9)
PR	Bradycardia	0	(0)
	Normal pulse rate	26	(35.1)
	Tachycardia	48	(64.9)
Temp	Hypothermia	21	(28.4)
	Normal body temperature	30	(40.5)
	Hyperthermia	23	(31.1)
CRP	Normal CRP	17	(23)
	High CRP	57	(77)
D. Dimer	Normal D dimer	28	(37.8)
	High D dimer	46	(62.2)
LDH	Low LDH	22	(29.7)
	Normal LDH	36	(48.6)
	High LDH	16	(21.6)
WBC	Leukopenia	20	(27)
	Normal WBC	49	(66.2)
	Leukocytosis	5	(6.8)
Forced Expiratory Volume	Low FEV1	43	(58.1)
in one second	Normal FEV1	30	(40.5)
	High FEV1	1	(1.4)
Forced Vital Capacity	Low FVC	47	(63.5)
	Normal FVC	24	(32.4)
	High FVC	3	(4.1)
FEV1/FVC	Airflow Limitation	1	(1.4)
	Normal FEV1/FVC	0	(0)
	More than Normal	73	(98.6)
Pattern	Normal	53	(71.6)
	Obstructive	3	(4.1)
	Restrictive	18	(24.3)

Table 3: Severity

Regarding the severity, the highest percentage was mild (43.2%) followed by moderate and severe (28.4% and 25.7% respectively).

Severity	F.	(%)
Mild	32	(43.2)
Moderate	21	(28.4)
Severe	19	(25.7)
Critical	2	(2.7)
Total	**74**	**(100)**

Concerning the association between demographic and clinical characteristics with the severity, there is a very highly significant association between age groups and SOB with the severity (P-value < 0.001). The other associations are non-significant (P-value > 0.05).

Concerning the association between investigations and severity, there is a significant association between SPO2, PR, D. Dimer, WBC, Pulmonary Function Tests, and Forced Vital Capacity with the severity (P-value ≤ 0.05). The other associations are non-significant (P-value > 0.05).

Table 4. Association between Demographic and Clinical Characteristics with Severity

Demographic and Clinical Characteristics		Severity								P-value
		Mild		Moderate		Severe		Critical		
		F.	(%)	F.	(%)	F.	(%)	F.	(%)	
Age Groups (years)	22-37	10	(76.9)	2	(15.4)	1	(7.7)	0	(0)	< 0.001 VHS
	38-53	15	(75)	2	(10)	3	(15)	0	(0)	
	54-69	7	(17.1)	17	(41.5)	15	(36.6)	2	(4.9)	
Sex	Male	19	(40.4)	16	(34)	11	(23.4)	1	(2.1)	0.550 NS
	Female	13	(48.1)	5	(18.5)	8	(29.6)	1	(3.7)	
Cough	No	14	(56)	6	(24)	4	(16)	1	(4)	0.349 NS
	Yes	18	(36.7)	15	(30.6)	15	(30.6)	1	(2)	
Fever	No	21	(42)	17	(34)	12	(24)	0	(0)	0.107 NS
	Yes	11	(45.8)	4	(16.7)	7	(29.2)	2	(8.3)	
SOB	No	15	(75)	2	(10)	1	(5)	2	(10)	< 0.001 VHS
	Yes	17	(31.5)	19	(35.2)	18	(33.3)	0	(0)	
others	No	9	(69.2)	3	(23.1)	1	(7.7)	0	(0)	0.169 NS
	Yes	23	(37.7)	18	(29.5)	18	(29.5)	2	(3.3)	
PMH	None	20	(51.3)	9	(23.1)	9	(23.1)	1	(2.6)	0.620 NS
	Asthma	1	(50)	1	(50)	0	(0)	0	(0)	
	COPD	2	(40)	3	(60)	0	(0)	0	(0)	
	Others	9	(32.1)	8	(28.6)	10	(35.7)	1	(3.6)	
Smoking	No Smoker	23	(46.9)	12	(24.5)	14	(28.6)	0	(0)	0.149 NS
	Smoker	5	(62.5)	2	(25)	0	(0)	1	(12.5)	
	Ex-Smoker	3	(18.8)	7	(43.8)	5	(31.3)	1	(6.3)	
	Passive-Smoker	1	(100)	0	(0)	0	(0)	0	(0)	

Table 5: Association between investigations with the severity

Investigations		Severity								P–value
		Mild		Moderate		Severe		Critical		
		F.	(%)	F.	(%)	F.	(%)	F.	(%)	
SBP	Low Systolic Blood Pressure	0	(0)	2	(50)	2	(50)	0	(0)	
	Normal Systolic Blood Pressure	20	(60.6)	6	(18.2)	6	(18.2)	1	(3)	**0.148 NS**
	High Systolic Blood Pressure	12	(32.4)	13	(35.1)	11	(29.7)	1	(2.7)	
DBP	Low Diastolic Blood Pressure	1	(25)	1	(25)	2	(50)	0	(0)	
	Normal Diastolic Blood Pressure	24	(54.5)	11	(25)	8	(18.2)	1	(2.3)	**0.348 NS**
	High Diastolic Blood Pressure	7	(26.9)	9	(34.6)	9	(34.6)	1	(3.8)	
SPO2	Hypoxemia	18	(30)	21	(35)	19	(31.7)	2	(3.3)	**< 0.001 VHS**
	Normal SPO2	14	(100)	0	(0)	0	(0)	0	(0)	
PR	Bradycardia	0	(0)	0	(0)	0	(0)	0	(0)	**0.002 HS**
	Normal pulse rate	18	(69.2)	7	(26.9)	1	(3.8)	0	(0)	
	Tachycardia	14	(29.2)	14	(29.2)	18	(37.5)	2	(4.2)	
Temp	Hypothermia	8	(38.1)	6	(28.6)	6	(28.6)	1	(4.8)	
	Normal body temperature	17	(56.7)	9	(30)	4	(13.3)	0	(0)	**0.306 NS**
	Hyperthermia	7	(30.4)	6	(26.1)	9	(39.1)	1	(4.3)	
CRP	Normal CRP	10	(58.8)	5	(29.4)	2	(11.8)	0	(0)	**0.320 NS**
	High CRP	22	(38.6)	16	(28.1)	17	(29.8)	2	(3.5)	
D. Dimer	Normal D dimer	19	(67.9)	3	(10.7)	4	(14.3)	2	(7.1)	**0.001 VHS**
	High D dimer	13	(28.3)	18	(39.1)	15	(32.6)	0	(0)	
LDH	Low LDH	10	(45.5)	6	(27.3)	6	(27.3)	0	(0)	**0.271 NS**
	Normal LDH	16	(44.4)	11	(30.6)	9	(25)	0	(0)	
	High LDH	6	(37.5)	4	(25)	4	(25)	2	(12.5)	
WBC	Leukopenia	8	(40)	7	(35)	5	(25)	0	(0)	**0.010 S**
	Normal WBC	24	(49)	14	(28.6)	10	(20.4)	1	(2)	
	Leukocytosis	0	(0)	0	(0)	4	(80)	1	(20)	
Pulmonary Function	Low FEV1	7	(16.3)	15	(34.9)	19	(44.2)	2	(4.7)	
	Normal FEV1	24	(80)	6	(20)	0	(0)	0	(0)	**< 0.001 VHS**
	High FEV1	1	(100)	0	(0)	0	(0)	0	(0)	
Forced Vital Capacity	Low FVC	10	(21.3)	16	(34)	19	(40.4)	2	(4.3)	
	Normal FVC	20	(83.3)	4	(16.7)	0	(0)	0	(0)	**< 0.001 VHS**
	High FVC	2	(66.7)	1	(33.3)	0	(0)	0	(0)	
FEV1FVC	Airflow Limitation	1	(100)	0	(0)	0	(0)	0	(0)	
	Normal FEV1/FVC	0	(0)	0	(0)	0	(0)	0	(0)	**0.722 NS**
	More than Normal	31	(42.5)	21	(28.8)	19	(26)	2	(2.7)	

Concerning the association between demographic and clinical characteristics and the spirometry pattern, all of the associations are non-significant (P -value > 0.05).

Table 6: Association between Demographic and Clinical Characteristics and the Spirometry Pattern

Demographic and Clinical Characteristics		Spirometry Pattern						P-value
		Normal		Obstructive		Restrictive		
		F.	(%)	F.	(%)	F.	(%)	
Age Groups (years)	22-37	11	(84.6)	0	(0)	2	(15.4)	**0.367 NS**
	38-53	16	(80)	0	(0)	4	(20)	
	54-69	26	(63.4)	3	(7.3)	12	(29.3)	
Sex	Male	34	(72.3)	3	(6.4)	10	(21.3)	**0.329 NS**
	Female	19	(70.4)	0	(0)	8	(29.6)	
Cough	No	17	(68)	1	(4)	7	(28)	**0.870 NS**
	Yes	36	(73.5)	2	(4.1)	11	(22.4)	
Fever	No	39	(78)	2	(4)	9	(18)	**0.182 NS**
	Yes	14	(58.3)	1	(4.2)	9	(37.5)	
SOB	No	15	(75)	1	(5)	4	(20)	**0.854 NS**
	Yes	38	(70.4)	2	(3.7)	14	(25.9)	
Pregnancy	No	53	(71.6)	3	(4.1)	18	(24.3)	**Constant**
	Yes	0	(0)	0	(0)	0	(0)	
SBP	Low Systolic Blood Pressure	3	(75)	0	(0)	1	(25)	
	Normal Systolic Blood Pressure	25	(75.8)	2	(6.1)	6	(18.2)	**0.775 NS**
	High Systolic Blood Pressure	25	(67.6)	1	(2.7)	11	(29.7)	
DBP	Low Diastolic Blood Pressure	3	(75)	0	(0)	1	(25)	
	Normal Diastolic Blood Pressure	32	(72.7)	2	(4.5)	10	(22.7)	**0.987 NS**
	High Diastolic Blood Pressure	18	(69.2)	1	(3.8)	7	(26.9)	
SPO2	Hypoxemia	41	(68.3)	3	(5)	16	(26.7)	**0.388 NS**
	Normal SPO2	12	(85.7)	0	(0)	2	(14.3)	
Temp	Hypothermia	15	(71.4)	2	(9.5)	4	(19)	
	Normal body temperature	23	(76.7)	1	(3.3)	6	(20)	**0.371 NS**
	Hyperthermia	15	(65.2)	0	(0)	8	(34.8)	
PMH	None	29	(74.4)	0	(0)	10	(25.6)	
	Asthma	2	(100)	0	(0)	0	(0)	**0.244 NS**
	COPD	4	(80)	1	(20)	0	(0)	
	Others	18	(64.3)	2	(7.1)	8	(28.6)	
CRP	Normal CRP	16	(94.1)	0	(0)	1	(5.9)	**0.069 NS**
	High CRP	37	(64.9)	3	(5.3)	17	(29.8)	
D. dimer	Normal D dimer	19	(67.9)	1	(3.6)	8	(28.6)	**0.799 NS**
	High D dimer	34	(73.9)	2	(4.3)	10	(21.7)	
Smoking	No Smoker	35	(71.4)	2	(4.1)	12	(24.5)	
	Smoker	5	(62.5)	1	(12.5)	2	(25)	**0.860 NS**
	Ex-Smoker	12	(75)	0	(0)	4	(25)	
	Passive-Smoker	1	(100)	0	(0)	0	(0)	

NS* Non-Significant

Concerning the association between Spirometry Pattern with the severity, there is a very highly significant association between Spirometry Pattern and the severity (P-value < 0.001).

Table 7: Association between Spirometry Pattern and Severity

Severity	Spirometry Pattern						P-value
	Normal		Obstructive		Restrictive		
	F.	(%)	F.	(%)	F.	(%)	
Mild	28	(87.5)	2	(6.3)	2	(6.3)	
Moderate	19	(90.5)	0	(0)	2	(9.5)	**< 0.001**
Severe	6	(31.6)	1	(5.3)	12	(63.2)	
Critical	0	(0)	0	(0)	2	(100)	**VHS***

VHS* Very Highly Significant

Regarding the association between the Spirometry Pattern and the Comorbidities, there is a non- significant association between Spirometry Pattern and the comorbidities (P-value 0.244).

Table 8: Association between the Spirometry Pattern and Comorbidities

PMH	Spirometry Pattern						P-value
	Normal		Obstructive		Restrictive		
	F.	(%)	F.	(%)	F.	(%)	
None	29	(74.4)	0	(0)	10	(25.6)	
Asthma	2	(100)	0	(0)	0	(0)	**0.244**
COPD	4	(80)	1	(20)	0	(0)	
Others	18	(64.3)	2	(7.1)	8	(28.6)	**NS**

NS* Non-Significant

DISCUSSION

The age distribution in this study demonstrates a predominance of patients between 54-69 years old, comprising 55.4% of the sample, which aligns with previous research indicating a higher susceptibility to severe COVID-19 outcomes among older individuals (Rothan and Byrareddy, 2020). Additionally, a higher proportion of males (63.5%) compared to females (36.5%) was observed, consistent with prior observations of COVID-19's disproportionate impact on males (Jin et al., 2020). Symptomatology among post-COVID-19 patients reveals a significant prevalence of cough (66.2%), fever (32.4%), and shortness of breath (73%), reflecting the persistence of respiratory symptoms even after recovery from acute infection, a phenomenon reported in several studies (Carfi et al., 2020; Huang et al., 2021). Comparing these findings with existing literature, several similarities and differences emerge. For instance, while our study reports a high prevalence of cough and shortness of breath, Huang et al. (2021) found a similar pattern of respiratory symptoms in their cohort of post-COVID-19 patients. However, the prevalence of fever in our study appears lower than reported by Carfi et al. (2020), suggesting potential variations in symptom presentation across different patient populations or study methodologies. Elevated blood pressure, hypoxemia, tachycardia, and abnormal biomarker levels x

(e.g., CRP, D. Dimer) indicate persistent systemic inflammation and cardiovascular involvement in post-COVID-19 patients (Nalbandian et al., 2021; Libby et al., 2021). Pulmonary function tests reveal a high prevalence of airflow limitation and restrictive patterns, indicative of lingering respiratory impairments post-recovery (George et al., 2020). When compared with similar investigations, our findings corroborate the persistence of cardiovascular and respiratory sequelae post-COVID-19. For instance, Nalbandian et al. (2021) reported a similarly high prevalence of abnormal cardiac biomarkers and pulmonary dysfunction in their study cohort. However, differences in the prevalence of specific abnormalities may reflect variations in patient demographics, disease severity, or follow-up duration across studies. The distribution of severity among post-COVID-19 patients, with mild cases being the most prevalent (43.2%), followed by moderate (28.4%) and severe (25.7%) cases. This distribution underscores the heterogeneity in symptom presentation and the need for tailored management strategies based on the severity of symptoms. the association between demographic/clinical characteristics and the severity of post-COVID-19 symptoms. Notably, age groups and shortness of breath (SOB) exhibit a highly significant association with severity (P-value < 0.001), suggesting that older age and the presence of SOB may predispose individuals to more severe outcomes. Other associations, such as sex, cough, fever, past medical history, and smoking status, appear non-significant, indicating that these factors may not independently predict the severity of post-COVID-19 symptoms in this cohort. Comparing these findings with existing literature can provide further insights. For instance, while age has consistently been identified as a significant predictor of COVID-19 severity in numerous studies (Williamson et al., 2020), the association between symptoms like cough and fever with severity may vary depending on factors like disease prevalence, population demographics, and study design. The association between investigative parameters and the severity of post-COVID-19 symptoms. Parameters such as SPO2, pulse rate (PR), D. Dimer, white blood cell count (WBC), and pulmonary function tests (FEV1 and FVC) demonstrate a significant association with severity (P-value ≤ 0.05). This suggests that abnormalities in these parameters may serve as valuable indicators of disease severity and could aid in risk stratification and clinical decision-making. A significant association between spirometry patterns and the severity of post-COVID -19 symptoms. Notably, a very highly significant association (P-value < 0.001) was observed, indicating that the type of spirometry pattern exhibited by patients is strongly correlated with the severity of their symptoms. Specifically, patients with mild or moderate symptoms predominantly displayed normal or obstructive spirometry patterns, while those with severe or critical symptoms were more likely to exhibit restrictive patterns. This suggests that spirometry patterns could serve as valuable indicators of disease severity in post-COVID-19 patients, aiding clinicians in risk stratification and prognostication. A non-significant association (P-value 0.244) between spirometry patterns and comorbidities among post-COVID-19 patients. Despite trends indicating that patients with comorbidities such as asthma and COPD tend to exhibit obstructive spirometry patterns, while those with other comorbidities display varied patterns, the lack of statistical significance suggests that spirometry patterns may not be strongly influenced by comorbidities alone in this cohort. Further research is warranted to elucidate the interplay between comorbidities

and spirometry patterns in post-COVID-19 patients. These findings align with emerging research suggesting that spirometry can serve as a valuable tool for assessing disease severity and guiding clinical management in post-COVID-19 care. Previous studies have also highlighted the utility of spirometry in identifying respiratory impairments and predicting clinical outcomes in COVID-19 patients (George et al., 2020; Torres-Castro et al., 2021). However, the association between spirometry patterns and comorbidities warrants further investigation, as it may have implications for personalized treatment approaches and long-term management strategies in post-COVID-19 care.

CONCLUSION

In conclusion, spirometry patterns exhibit a significant association with the severity of post-COVID-19 symptoms, highlighting their potential utility as prognostic markers in clinical practice. While the association between spirometry patterns and comorbidities appears non-significant in this study, further research is needed to elucidate the complex interplay between these factors and their implications for patient management.

REFERENCES

[1] Bradley BT, Maioli H, Johnston R, Chaudhry I, Fink SL, Xu H, et al. Histopathology and ultrastructural findings of fatal COVID-19 infections in Washington State: a case series. *The Lancet*. 2020 Aug 1;396(10247):320-32. https://doi.org/10.1016/s0140-6736(20)31305-2 .

[2] Carfi A, Bernabei R, Landi F. Persistent symptoms in patients after acute COVID-19. *Jama.* 2020 Aug 11;324(6):603-5. https://doi:10.1001/jama.2020.12603

[3] Cordier JF. Organising pneumonia. Thorax. 2000 Apr 1;55(4):318-28. https://doi.org/10.1136/*thorax*.55.4.318

[4] Docherty AB, Harrison EM, Green CA, Hardwick HE, Pius R, Norman L, et al. Features of 20 133 UK patients in hospital with covid-19 using the ISARIC WHO Clinical Characterisation Protocol: prospective observational cohort study. *bmj.* 2020 May 22;369. https://doi.org/10.1136/bmj.m1985.

[5] Drake TM, Riad AM, Fairfield CJ, Egan C, Knight SR, Pius R, et al. Characterisation of in-hospital complications associated with COVID-19 using the ISARIC WHO Clinical Characterisation Protocol UK: a prospective, multicentre cohort study. *The Lancet*. 2021 Jul 17;398(10296):223-37. https://doi.org/10.1016/s0140-6736(21)00799-6

[6] George PM, Wells AU, Jenkins RG. Pulmonary fibrosis and COVID-19: the potential role for antifibrotic therapy. *The Lancet Respiratory Medicine.* 2020 Aug 1;8(8):807-15. https://doi.org/10.1016/s2213-2600(20)30225-3

[7] Horby P, Lim WS, Emberson JR. RECOVERY Collaborative Group Dexamethasone in hospitalized patients with Covid-19-Preliminary report. *N Engl J Med*. https://doi.org/10.1056/nejmoa2021436

[8] Huang C, Huang L, Wang Y, Li X, Ren L, Gu X, et al. 6-month consequences of COVID-19 in patients discharged from hospital: a cohort study. *The Lancet.* 2023 Jun 17;401(10393):e21-33. https://doi.org/10.1016/S0140-6736(20)32656-8

[9] Hui DS, Wong KT, Ko FW, Tam LS, Chan DP, Woo J, et al. The 1-year impact of severe acute respiratory syndrome on pulmonary function, exercise capacity, and quality of life in a cohort of survivors. *Chest.* 2005 Oct 1;128(4):2247-61. https://doi.org/10.1378/chest.128.4.2247

[10] Jin JM, Bai P, He W, Wu F, Liu XF, Han DM, et al. Gender differences in patients with COVID-19: focus on severity and mortality. *Frontiers in Public Health*. 2020 Apr 29;8:545030. https://doi.org/10.3389/fpubh.2020.00152

[11] Libby P, Lüscher T. COVID-19 is, in the end, an endothelial disease. *European Heart Journal.* 2020 Aug 21;41(32):3038-44. https://doi.org/10.1093/eurheartj/ehaa623

[12] Lorent N, Weygaerde YV, Claeys E, Fajardo IG, De Vos N, De Wever W, et al. Prospective longitudinal evaluation of hospitalised COVID-19 survivors 3 and 12 months after discharge. *ERJ open research*. 2022 Apr 1;8(2).https://doi.org/10.1183/23120541.00004-2022

[13] Lu R, Zhao X, Li J, Niu P, Yang B, Wu H, et al. Genomic characterisation and epidemiology of 2019 novel coronavirus: implications for virus origins and receptor binding. *The Lancet*. 2020 Feb 22;395(10224):565-74. https://doi.org/10.1016/S0140-6736(20)30251-8 .

[14] Mehta P, Rosas IO, Singer M. Understanding post-COVID-19 interstitial lung disease (ILD): a new fibroinflammatory disease entity. *Intensive Care Medicine.* 2022 Dec;48(12):1803-6. https://doi.org/10.1007/s00134-022-06877-w

[15] Munblit D, O'Hara ME, Akrami A, Perego E, Olliaro P, Needham DM. Long COVID: aiming for a consensus. *The Lancet Respiratory Medicine.* 2022 Jul 1;10(7):632-4. https://doi.org/10.1016/s2213-2600(22)00135-7

[16] Nalbandian A, Sehgal K, Gupta A, Madhavan MV, McGroder C, Stevens JS, et al. Post-acute COVID-19 syndrome. *Nature Medicine*. 2021 Apr;27(4):601-15. https://doi.org/10.1038/s41591-021-01283-z

[17] Ngai JC, Ko FW, Ng SS, TO KW, Tong M, Hui DS. The long-term impact of severe acute respiratory syndrome on pulmonary function, exercise capacity and health status. *Respirology.* 2010 Apr;15(3):543-50. https://doi.org/10.1111/j.1440-1843.2010.01720.x

[18] Ong KC, Ng AK, Lee LU, Kaw G, Kwek SK, Leow MS, et al. Pulmonary function and exercise capacity in survivors of severe acute respiratory syndrome. *European Respiratory Journal.* 2004 Sep 1;24(3):436-42. https://doi.org/10.1136/thx.2004.030205

[19] Ong KC, Ng AW, Lee LS, Kaw G, Kwek SK, Leow MK, et al. 1-year pulmonary function and health status in survivors of severe acute respiratory syndrome. *Chest.* 2005 Sep 1;128(3):1393-400. https://doi.org/10.1378/chest.128.3.1393

[20] Pan F, Ye T, Sun P, Gui S, Liang B, Li L, et al. Time course of lung changes at chest CT during recovery from coronavirus disease 2019 (COVID-19). *Radiology.* 2020 Jun;295(3):715-21.https://doi.org/10.114radiol.2020200370

[21] Park WB, Jun KI, Kim G, Choi JP, Rhee JY, Cheon S, et al. Correlation between pneumonia severity and pulmonary complications in Middle East respiratory syndrome. *Journal of Korean Medical Science.* 2018 Jun 6;33(24). https://doi.org/10.3346/jkms.2018.33.e169

[22] Richardson S, Hirsch JS, Narasimhan M, Crawford JM, McGinn T, Davidson KW, et al. Presenting characteristics, comorbidities, and outcomes among 5700 patients hospitalized with COVID-19 in the New York City area. *Jama.* 2020 May 26;323(20):2052-9. https://doi.org/10.1001/jama.2020.6775

[23] Rothan HA, Byrareddy SN. The epidemiology and pathogenesis of coronavirus disease (COVID-19) outbreak. *Journal of Autoimmunity*. 2020 May 1;109:102433. https://doi.org/10.1016/j.jaut.2020.102433 .

[24] Sanchez-Ramirez DC, Mackey D. Underlying respiratory diseases, specifically COPD, and smoking are associated with severe COVID-19 outcomes: a systematic review and meta-analysis. *Respiratory Medicine*. 2020 Sep 1;171:106096. https://doi.org/10.1016/j.rmed.2020.106096

[25] Sterne JA, Murthy SW, Diaz JV, Slutsky AS, Villar JW, Angus DC, et al. WHO Rapid Evidence Appraisal for COVID-19 Therapies (REACT) Working Group. Association between administration of systemic corticosteroids and mortality among critically ill patients with COVID-19: a meta-analysis. *Jama.* 2020 Oct 6;324(13):1330-41. https://doi.org/10.1001/jama.2020.17023

[26] Sze S, Pan D, Nevill CR, Gray LJ, Martin CA, Nazareth J, et al. Ethnicity and clinical outcomes in COVID-19: a systematic review and meta-analysis. *E Clinical Medicine*. 2020 Dec 1;29. https://doi.org/10.1016/j.eclinm.2020.100630

[27] Torres-Castro R, Vasconcello-Castillo L, Alsina-Restoy X, Solis-Navarro L, Burgos F, Puppo H, et al. Respiratory function in patients post-infection by COVID-19: a systematic review and meta-analysis. *Pulmonology.* 2021 Jul 1;27(4):328-37. https://doi.org/10.1016/j.pulmoe.2020.10.013

[28] Wang Y, Dong C, Hu Y, Li C, Ren Q, Zhang X, et al. Temporal changes of CT findings in 90 patients with COVID-19 pneumonia: a longitudinal study. *Radiology*. 2020 Aug;296(2):E55-64. https://doi.org/10.1148/radiol.2020200843

[29] Williamson EJ, Walker AJ, Bhaskaran K, Bacon S, Bates C, Morton CE, et al. Factors associated with COVID-19-related death using OpenSAFELY. *Nature*. 2020 Aug 20;584(7821):430-6. https://doi.org/10.1038/s41586-020-2521-4

[30] Zhao W, Zhong Z, Xie X, Yu Q, Liu J. Relation between chest CT findings and clinical conditions of coronavirus disease (COVID-19) pneumonia: a multicenter study. *American journal of roentgenology.* 2020 May;214(5):10727.https:doi.org/10.2214ajr.20.22976