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Pneumonia due to respiratory syncytial virus does not cause long-lasting disorders in respiratory gas transport after clinical resolution in adults. Comparison *vs.* SARS-CoV-2 pneumonia

Roberto W. Dal Negro,[1] Paola Turco,[1] Massimiliano Povero[2]

[1]National Centre for Respiratory Pharmacoeconomics and Pharmacoepidemiology – CESFAR, Verona; [2]AdRes Health Economics and Outcomes Research, Turin, Italy

Correspondence: Massimiliano Povero, AdRes Health Economics and Outcomes Research, Turin, Italy. E-mail: m.povero@adreshe.com

Contributions: Roberto W. Dal Negro: planned the study and wrote the manuscript. Paola Turco: checked the data base, provided critical feedback and contributed to the final version of the manuscript. Massimiliano Povero: carried out all statistical calculations and contributed to the manuscript. All authors approved the final version of the manuscript.

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Abstract

Long-term consequences of viral pneumonia on lung function depend on virus-specific tissue injury. Persistent impairment of alveolar-blood gas transport has been described after SARS-CoV-2 pneumonia but has not been investigated following respiratory syncytial virus (RSV) pneumonia. Possible long-term effects of these two viral infections were never compared after their clinical resolution in terms of lung physiology. The aim of this paper was to compare long-term sequelae of RSV and SARS-CoV-2 pneumonia on lung function and blood gas transport. Adults (>18 years) with previous RSV or SARS-CoV-2 pneumonia were investigated after complete computed tomography scan resolution. Collected variables included demographics, body mass index, hemoglobin, SpO_2, modified British Medical Research Council dyspnea score, spirometry, diffusing capacity [diffusing capacity for carbon oxide (DL_{CO}), carbon monoxide transfer coefficient (K_{CO})], single-breath diffusing capacity for nitric oxide (DL_{NO}) and DL_{CO}, DL_{NO}/DL_{CO} ratio, and lung capillary blood volume (Vc).

38 post-SARS-CoV-2 and 37 post-RSV patients were studied. Groups were comparable and showed similar spirometric values. Compared with RSV, SARS-CoV-2 patients had significantly lower SpO_2, DL_{CO}, and K_{CO} ($p<0.01$). Vc was markedly reduced ($p<0.001$), with a corresponding increase in the DL_{NO}/DL_{CO} ratio ($p<0.001$). Only post-RSV patients were dyspnea-free. In conclusion, viral pneumonia may cause long-lasting lung function impairment depending on virus-specific tissue damage. Unlike RSV, SARS-CoV-2 pneumonia induces a persistent reduction of lung Vc, leading to impaired gas exchange despite radiological resolution.

Key words: RSV pneumonia, SARS-CoV-2 pneumonia, lung function, long-lasting sequelae, blood gas transport, single-breath simultaneous DL_{NO} and DL_{CO} diffusing capacity.

Introduction

Respiratory infections due to rhino, paramyxo, corona and influenza viruses are events that are known to commonly occur in human populations since long ago [1-3]. They are characterized by variable severity and extension from the short-term inflammation of proximal airways up to the long-term involvement of peripheral airways and of the deep lung structures, not infrequently leading to fatigue, dyspnea, hypoxia, or respiratory failure [1-3].

While some respiratory viral infections usually cause no or mild respiratory sequelae, SARS-CoV-2 and Respiratory Syncytial Virus (RSV) in particular, are described to possibly cause a substantial and persistent impact on current lung function over time, mostly related to parenchymal injury within the deep lung or to induced bronchial hyperreactivity and asthma, respectively [4-9].

Pneumonia can represent a frequent complication of respiratory viral infections also in adults [10]. Consequences on lung function are of variable impact and duration as their pattern of underlying tissular injury have been described to be virus-specific. Actually, substantial disorders in alveolar-blood gas transport have been reported to persist for several weeks after SARS-CoV-2 pneumonia in a great proportion of patients despite their stable clinical and radiological resolution [11-13]. Unfortunately, likely due to the dreadful impact of the recent pandemic, consequences over-time of non-SARS-CoV-2 respiratory viral infections, RSV included, have received less attention in adults. In particular, the possible long-term persistence of lung function sequelae due to RSV pneumonia has been scarcely investigated to our knowledge in recent years [14,15].

The aim of the study was to investigate the long-term consequences on current lung function and on blood gas transport after several weeks from the clinical and radiological resolution of RSV compared to SARS-CoV-2 pneumonia.

Materials and Methods

Subjects of both genders aged >18 years who experienced RSV pneumonia or SARS-CoV-2 pneumonia were investigated between October 1, 2023 and April 30, 2025. All subjects were recruited after 12±2 weeks from their clinical recovery and complete resolution of any parenchymal lesion as confirmed by the CT-scan available near recruitment. The etiology of pneumonia had been confirmed by nasal swab and specific serologic tests in all subjects.

Variables collected in all patients were: age; gender; BMI; blood hemoglobin (Hb, in g/L); $\%O_2$ saturation ($SpO_2\%$); the current dyspnea score graded in each patient according to the modified British Medical Research Council (mMRC) [16] the total lung capacity (TLC); the vital capacity (VC); the forced vital capacity (FVC); the forced expiratory volume in 1 sec (FEV_1); the

current diffusing capacity for carbon oxide (DL_{CO}) and Carbon Monoxide Transfer Coefficient (K_{CO}), both by 10″ breath-hold time; the single-breath simultaneous diffusing capacity for nitric oxide and carbon oxide (sDL_{NO} and sDL_{CO}), both by 5″ breath-hold time; the sDL_{NO}/sDL_{CO} ratio and the lung capillary blood volume (Vc). Except for age, gender, BMI, Hb and mMRC score, all other parameters were reported in % predicted.

Current and former-smokers were excluded together with those patients with major comorbidities affecting measurements of the lung diffusion (such as: anemia (blood Hb <12g/L); heart failure; COPD; lung fibrosis; vasculitis; renal and liver failure; diabetes); persistent COVID-related parenchymal abnormalities; physical and/or cognitive impairment that affect the procedures required for obtaining effective lung function tests). Subjects' selections were carried out automatically and anonymously from the institutional data-base. Patients were simply recruited sequentially over the study period. Only patients who did not fit with the exclusion criteria reported above were excluded.

A Plethysmography Platinum DX Elite (MedGraphics, Saint Paul, MN, USA) was used for measuring spirometrical parameters and current DLco. A "Stand-Alone" Hypair Compact System (MGC Diagnostics International, Sorinnes, Belgium) was used for the simultaneous assessment of sDL_{NO}, sDL_{CO} and of Vc as a function of the standard single-breath method. This method stems from the Roughton and Forster's principle [17] of two reactions: the first one for CO and the second one for NO, according to the values fixed in the ERS/ATS Task Force 2017 [18,19], during the usual single-breath maneuvers. The use of an electrochemical analyzer consents to reduce the usual 10 sec. apnea duration required for current DL_{CO} measures to only 5 sec. for simultaneous CO and NO measures. Two gas mixtures are required for these measures: 1) helium (He) 14%; CO 0.280%; oxygen (O_2) 18–21, and nitrogen (N_2) and, 2) nitric oxide in nitrogen (NO in N_2) 400 ppm. More technical details had been described in a previous paper [11].

The study was approved by the CASFAR Ethical Committee during the session of September 18, 2023 (N. 04/CES/2023). All patients provided their written informed consent also for using their data for scientific purposes.

Statistics

Means and standard deviation (SD) were used to summarize continuous variables, while gender was reported as absolute and relative frequencies. Differences assessed in baseline between patients who experienced RSV or SARS-CoV-2 pneumonia were tested by the non-parametric Wilcoxon test (for continuous variables) and Fisher exact test (for gender). Differences in lung function parameters were estimated by a generalized linear model (gamma

family) adjusting for population characteristics (age, gender, BMI, Hb). Model assumptions were assessed by examining deviance and Pearson residuals and by evaluating the mean–variance relationship expected under the gamma distribution. Goodness-of-fit was evaluated using deviance statistics and the Pearson chi-square divided by degrees of freedom. Results were reported as adjusted mean difference (AMD) and confidence intervals (CI). Finally, differences in the distribution of DL_{CO}% pred., K_{CO}% pred., sDL_{NO}/sDL_{CO} ratio, and Vc% pred. were visually investigated by using box plots.

A p-value < 0.05 was considered statistically significant. All statistical calculations were carried out by means of STATA (StataCorp. 2017. Stata Statistical Software: Release 15. College Station, TX: StataCorp LLC).

Results

The whole sample consisted of 75 subjects (40 males) who experienced SARS-CoV-2 (n=38) or RSV pneumonia (n=37), well matched for age, BMI and comorbidities. General data are reported in Table 1. Originally, patients of both groups suffered from pneumonia equally affecting 40-50% of their lung volume (originally CT documented at admission). During their hospitalization they received high flow oxygen of variable duration. No patient was intubated. Only the mMRC dyspnea score proved persistently higher in the post-SARS-CoV-2 pneumonia group (p<0.001). In particular, while around 40% of patients in the post- SARS-CoV-2 pneumonia group were still complaining a moderate-to-severe dyspnea for twelve weeks after their clinical and radiological recovery, the vast majority of patients in the post-RSV pneumonia group did not complain any persistent dyspnea after the same period (p<0.001). Mean values for blood hemoglobin and each lung function variables are reported in Table 2 together with the corresponding statistical comparisons (no relevant deviations from model assumptions were detected).

Both groups equally showed values of blood hemoglobulin and of current spirometrical parameters in the normal ranges (p=ns), while mean values for SpO_2% were slightly (but significantly) lower in the post-SARS-CoV-2 pneumonia group (p<0.001).

When the two groups were compared by their lung diffusion indices, mean values for current DL_{CO}% pred. and for K_{CO} resulted slightly, but significantly, lower in the post-SARS-CoV-2 group over time, though characterized by higher variability (p< 0.001 and p< 0.01, respectively). Moreover, sDL_{NO}/sDL_{CO} and Vc% pred. mean values were dramatically low in patients who experienced SARS-CoV-2 pneumonia while the vast majority of those who experienced RSV pneumonia showed corresponding mean values in the normal ranges (both p<0.001) (Table 1). The corresponding mean breath-hold times calculated for current and

simultaneous diffusive measurements (such as, 10 or 5sec, respectively) were superimposable in the groups, thus confirming the good quality and reliability of measures (both p=ns) (Table 1). To further appreciate the differences calculated, it should be considered that the reference optimal cut-off values are 113.5 (95% CI 110 to 117) for the sDL_{NO}/sDL_{CO} ratio and 58.5 (95% CI 54 to 63) for Vc% pred., respectively [20]. Finally, when the distributions of sDL_{NO}/sDL_{CO} and Vc absolute values were compared, only a few post-RSV patients were at the lower limits of the normal cut-off levels (Figure 1). Distributions of current DL_{CO}% pred., K_{CO}% pred., sDL_{NO}/sDL_{CO} ratio, and Vc% pred. are reported in Figure 2.

Discussion

Together with SARS-CoV-2 and influenza, RSV infection remains one of the most important global public health burdens affecting humans of different ages in all countries [10,21,22], reaching related hospitalizations around 10% of the total lower airway infections [23].

Human RSV infection produces a broad spectrum of clinical manifestations, ranging from mild to more serious events, such as bronchiolitis (mainly in infants and in childhood) [24,25]. Pneumonia is more frequent in adults and in elderly people and can also lead to severe outcomes [10,26].

In general, the long-term consequences of viral infections over time are described to largely depend on the peculiar injury originally caused by the specific viral etiology to different respiratory structures. In particular, while SARS-CoV-2 pneumonia, further to the damage to alveolar epithelial cells, hyperplasia of type II pneumocytes and fibrin deposition, recognizes the occurrence of microvascular thrombosis and occlusion as the major pathogenetic events [27-30], the basic RSV pattern of lesions is characterized by widespread involvement of peripheral airways, uneven proliferation and disruption of type 1 (which facilitate gas exchange) and type 2 alveolar epithelial cells (which produce surfactant and act as progenitor cells) [31], production of papillary intra-bronchiolar syncytia, deposition of intraluminal cellular debris, but with the vascular side of the alveolar structures preserved [3].

Stemming from this pattern of evidence, the true scope of the present observational study was to investigate whether or not different pathogenetic determinants of viral lung injury could differently affect the lung physiology of these patients over time.

The persistent reduction of lung capillary blood volume (Vc), such as the total volume of blood in the lung capillaries exposed to alveolar air, has been identified as the major pathophysiological disorder that characterizes the respiratory Long-COVID syndrome specifically [11-13]. As confirmed by experimental models [32-34], the substantial and persistent lung capillary rarefication underlying the SARS-CoV-2 pneumonia explains its peculiar lung function picture

that is in fact primarily characterized by a substantial drop of lung capillary blood volume and then of blood/gas transport [11-13]. Conversely, as previously mentioned, the pathogenesis usually described for RSV pneumonia is substantially different as the vascular side of the alveolar/capillary membrane results substantially not involved. Therefore, as no damage to lung microvascular structures has never been reported in adults who experienced RSV-induced pneumonia, long-term consequences due to the loss in capillary blood volume can be presumed to be absent and then the blood/gas transport preserved.

Results of the present study seem to confirm this hypothesis. Actually, thanks to the non-invasive technology pivotally used in the present investigation (that is based on the single-breath simultaneous assessment of CO and NO diffusion capacity; i.e., sDL_{NO}/sDL_{CO}) it was possible to rapidly discriminate between disorders occurred in the alveolar membrane conductance (MD) from those involving the capillary blood volume (Vc) [11-13]. In other words, while changes in sDLNO are mainly related to disorders in membrane conductance (MD), sDL_{CO} changes are primarily related to the condition of the microvasculature in the deep lung. The Roughton and Forster's formula (that is: $1/DL_{CO} = R1+R2 = 1/DM+1/\theta_{CO}\cdot Vc$) [17] regulates the relationships among variables, where DM is the diffusing capacity (that is dependent on molecular diffusion only) of the membranes separating the alveolar epithelial surface from the red cell (also called the alveolar–capillary membrane conductance); Vc is the total volume (in milliliters) of blood in the lung capillaries exposed to alveolar air, and θ_{CO} is the number of milliliters of gas taken up by the red cells in 1 mL of blood/per minute/1 mmHg of partial pressure of dissolved gas between the plasma and interior of the red cell (also called the specific conductance in the blood for CO). Indeed, as the binding of NO with intracapillary Hb is extremely faster than that one of CO, sDL_{NO} mainly informs on the condition of the epithelial surface of the alveolar membrane, while sDL_{CO} mainly informs on the vascular phase of diffusion through the membrane. Moreover, only when DL_{NO} and DL_{CO} are simultaneously measured, the sDL_{NO}/sDL_{CO} ratio can be calculated. Obviously, higher the ratio, lower the value of sDL_{CO}, and then of the lung capillary blood volume (Vc).

Differently from current lung function parameters (such as: spirometrical volumes and flows, and current measures of lung diffusion (DL_{CO}), this recent method proves particularly suitable for investigating and assessing non-invasively and in short time the possible long-lasting consequences of pneumonia caused by different respiratory viruses in the deep lung.

Unlike what was reported in some studies where the occurrence of impaired gas transfer was generically associated to viral pneumonia regardless their etiology [35], data from the present study support the hypothesis that substantial differences exist between the lung function profile of subjects who experienced RSV rather than SARS-CoV-2 pneumonia. These data are strongly

supported and largely explained by the distinct pattern of tissular lesions that are known to characterize these two respiratory viral etiologies: the prevailing lung microvascular thrombosis and capillary rarefication documented in SARS-CoV-2 pneumonia [27-30,32-34] rather than the prevailing widespread involvement of peripheral airways and the uneven proliferation and disruption of alveolar epithelial cells described in RSV infections [3,24-26,31]. Moreover, differently from the post- SARS-CoV-2 pneumonia period, residual long-lasting dyspnea was totally absent in subjects who experienced RSV pneumonia. This feature further tends to confirm also in clinical terms the effects of the pathophysiological pattern described in these patients, such as the peculiar absence of any significant microvascular involvement and blood flow drop due to RSV lung infection.

We acknowledge some point of weakness: 1) the sample studied is limited and monocentric; 2) presently, only two respiratory viruses have been investigated and compared in terms of their attitude to induce long-lasting troubles in alveolar blood-gas transport; however, a further study vs influenza viruses is already in progress 3) the non-invasive technology used in the present investigation is not yet largely distributed in lung function labs. Points of strengths are: 1) the careful selection of the subjects' sample; 2) the large utilization of current lung function variables; 3) the pivotal adoption of a novel non-invasive technology for investigating and assessing specifically the occurrence of long-term disorders in alveolar blood-gas transport following SARS-CoV-2 and RSV pneumonia.

Conclusions

Pneumonia due to respiratory viruses can lead to different long-term consequences on lung function according to its specific etiology. The technology adopted in the present investigation allows the assessment of lung function effects over time due to the underlying injury occurred within the lung structures with high sensitivity and specificity.

Differently from pneumonia caused by SARS-CoV-2 infection, RSV pneumonia proves free from long-lasting sequelae in blood/gas transport after resolution as the original RSV injury does not involve the vascular side of alveolar structures in the vast majority of cases. The high correspondence between the SARS-CoV-2 and RSV pathogenesis and their specific lung function profiles can contribute to suggest the possible etiology in these cases of pneumonia also after their clinical resolution. The diagnostic procedure pivotally adopted in the present study can be intended as a useful diagnostic support for understanding the physiological evolution of disease with high sensitivity and specificity. In other words, this procedure seems to allow the detection of the deep lung's function (such as, the alveolar phase) successfully also in clinical practice, beyond the resolution power of present CT imaging that still is known

as not able to discriminate the determinants of alveolar injury. Further larger studies are needed to confirm and support present results.

References

1. Widmer K, Zhu Y, Williams JV, et al. Rates of hospitalizations for respiratory syncytial virus, human metapneumovirus, and influenza virus in older adults. J Infect Dis 2012;206:56-62.
2. Wright AL, Taussig LM, Ray CG, et al. The Tucson Children's Respiratory Study. II. Lower respiratory tract illness in the first year of life. Am J Epidemiol 1989;129:1232-46.
3. Pickles RJ, De Vincenzo JP. Respiratory syncytial virus (RSV) and its propensity for causing bronchiolitis. J Pathol 2015;235:266-76.
4. van den Borst B, Peters JB, Brink M, et al. Comprehensive health assessment three months after recovery from acute COVID-19. Clin Infect Dis 2020;73:e1089-98.
5. Psarras S, Papadopoulos NG, Johnston SI. Athogenesis of respiratory syncytial virus bronchiolitis-related wheezing. Paediatr Respir Rev 2004;5:S179-84.
6. Lukacs NW, Smit J, Lindell D, Schaller M. Respiratory syncytial virus-induced pulmonary disease and exacerbation of allergic asthma. Contrib Microbiol 2007;14:68-82.
7. Jartti T, Gern JE. Role of viral infections in the development and exacerbation of asthma in children. J Allergy Clin Immunol 2017;140:895-06.
8. Bellan M, Soddu D, Balbo PE, et al. Respiratory and psychosocial sequelae among patients with COVID-19 four months after hospital discharge. JAMA Netw Open 2021;4:e2036142.
9. Limkar AR, Percopo CM, Redes JL, et al. Persistent airway hyperresponsiveness following recovery from infection with pneumonia virus of mice. Viruses 2021;13:728.
10. Falsey AR. Respiratory syncytial virus infection in adults. Semin Respir Crit Care Med 2007;28:171-81.
11. Dal Negro RW, Turco P, Povero M. Long-lasting dyspnoea in patients otherwise clinically and radiologically recovered from COVID pneumonia: a probe for checking persisting disorders in capillary lung volume as a cause. Multidiscip Respir Med 2022;17:875.
12. Pini L, Giordani J, Levi G, et al. Long-term alveolar-capillary diffusion impairments after severe SARS-CoV-2 pneumonia. Ann Med 2025;57:2483383.

13. Zavorsky JS, Barisone G, Gille T, et al. Enhanced detection of patients with previous COVID-19: Superiority of the double diffusion technique. BMJ Open Respir Res 2025;12:e002561.
14. Branche AR, Falsey AR. Respiratory syncytial virus infection in older adults: an under-recognized problem. Drugs Aging 2015;32:261-9.
15. Estofolete CF, Banho CA, Verro AT, et al. Clinical characterization of respiratory syncytial virus infection in adults: a neglected disease? Viruses 2023;15:1848.
16. Mahler DA, Wells CK. Evaluation of clinical methods for rating dyspnoea. Chest 1988;93:580-6.
17. Roughton FJ, Forster RE. Relative importance of diffusion and chemicalreaction in determining rate of exchange of gases in the human lung. J Appl Physiol 1957;11:290-302.
18. Graham BL, Brusasco V, Burgos F, et al. ERS/ATS standards for single-breath carbon monoxide uptake in the lung. Eur Respir J 2017;49:1600016.
19. Zavorsky GS, Hsia CCW, Hughes MB, et al. Standardisation and application of the single-breath determination of nitric oxide uptake in the lung. Eur Respir J 2017;49:1600962.
20. Dal Negro RW, Turco P, Povero M. Persistent reduction of lung capillary blood volume as the major “deep lung” disorder underlying respiratory long-COVID syndrome. Am J Med Public Health 2024;5:1059.
21. Shi T, McAllister DA, O’Brien KL, et al. Global, regional, and national disease burden estimates of acute lower respiratory infections due to respiratory syncytial virus in young children in 2015: a systematic review and modelling study. Lancet 2017;390:946-58.
22. Falsey AR, Patricia A, Hennessey RN, et al. Respiratory syncytial virus infection in elderly and high-risk adults. N Engl J Med 2005;352:1749-59.
23. Nair H, Simoes EA, Rudan I, et al. Global and regional burden of hospital admissions for severe acute lower respiratory infections in young children in 2010: a systematic analysis. Lancet 2013;381:1380-90.
24. Sweetman LL, Ng YT, Butler IJ, Bodensteiner JB. Neurologic complications associated with respiratory syncytial virus. Pediatr Neurol 2005;32:307-10.
25. van Drunen Littel-van den Hurk S, Watkiss ER. Pathogenesis of respiratory syncytial virus. Curr Opin Virol 2012;2:300-5.
26. Dowell SF, Anderson LJ, Gary HE Jr, et al. Respiratory syncytial virus is an important cause of community-acquired lower respiratory infection among hospitalized adults. J Infect Dis 1996;174:456-62.

27. Matricardi PM, Dal Negro RW, Nisini R. The first, comprehensive immunological model of COVID-19: implications for prevention, diagnosis, and public health measures. Pediatr Allergy Immunol 2020;31:454-70.
28. Chu H, Chan JFW, Wang Y, et al. Comparative replication and immune activation profiles of SARS-CoV-2 and SARS-CoV in human lungs: an ex vivo study with implications for the pathogenesis of COVID-19. Clin Infect Dis 2020;ciaa410.
29. Iba T, Connors JM, Levy JH. The coagulopathy, endotheliopathy, and vasculitis of COVID-19. Inflamm Res 2020;69:1181-9.
30. Wu X, Xiang M, Jing H, et al. Damage to endothelial barriers and its contribution to long COVID. Angiogenesis 2024;27:5-22.
31. Morris SB, Ocadiz-Ruiz R, Asai N, et al. Long-term alterations in lung epithelial cells after EL-RSV infection exacerbate allergic responses through IL-1β-induced pathways. Mucosal Immunol 2024;17:1072-88.
32. Osiaevi I, Schulze A, Evers G, et al. Persistent capillary rarefication in long COVID syndrome. Angiogenesis 2023;26:53-61.
33. Rovas A, Osiaevi I, Buscher K, et al. Microvascular dysfunction in COVID-19: the MYSTIC study. Angiogenesis 2021;24:145-57.
34. Smadja DM, Mentzer SJ, Fontenay M, et al. COVID-19 is a systemic vascular hemopathy: insight for mechanistic and clinical aspects. Angiogenesis 2021;24:755-88.
35. Fabbri L, Moss S, Khan FA, et al. Parenchymal lung abnormalities following hospitalisation for COVID-19 and viral pneumonitis: a systematic review and meta-analysis. Thorax 2023;78:191-201.

Table 1. General data of the sample (continuous data expressed as means ± SD).

	post- SARS-CoV-2 pneumonia	post-RSV pneumonia	p
n	38	37	
Age (years)	52.5±16.4	55.8±14.3	0.329
Gender (% male)	44.7	62.2	0.100
BMI	26.3±5.9	26.1±6.3	0.787
Comorbidities			
% none	76.3	81.1	0.999
% hypertension	13.2	10.8	
% atopy	2.6	2.7	
% overweight	7.9	5.4	
mMRC dyspnea score	1.3±1.1	0.1±0.3	<0.001
% no dyspnea (score 0)	28.9	91.9	<0.001
% mild dyspnea (score 1)	31.6	8.1	
% moderate dyspnea (score 2)	21.1	0	
% severe dyspnea (score 3-4)	18.4	0	

BMI, body mass index; mMRC, modified British Medical Research Council; RSV, respiratory syncytial virus.

Table 2. Mean values of blood hemoglobin and all lung function parameters collected in the two subgroups (mean ± SD) together with statistical comparisons

	post- SARS-CoV-2 pneumonia	post-RSV pneumonia	AMD (95% CI)	p
Hb (g/L)	13.9±0.5	14.0±1.1	-0.11 (-0.52 to 0.29)	0.576
SpO_2%	96.2±1.1	97.9±0.8	-1.61 (-2.04 to -1.17))	<0.001
TLC% pred.	99.8±4.8	100.1±4.2	0.12 (-2.03 to 2.26)	0.915
VC% pred.	93.1±8.0	96.1±4.1	-2.74 (-6.01 to 0.53)	0.101
FVC% pred.	91.2±10.7	93.0±5.4	-1.67 (-5.89 to 2.54)	0.436
FEV_1% pred.	90.8±8.9	93.4±4.9	-2.44 (-6.48 to 1.60)	0.236
DL_{CO}% pred. (10sec breath-hold time)	76.5±14.8	89.8±9.3	-14.63 (-20.96 to -8.29)	<0.001
K_{CO}% pred.	86.26±12.2	91.68±8.7	-6.52 (-11.48 to -1.55)	0.010
sDL_{NO}/sDL_{CO} (5sec breath-hold time)	123.1±11.3	108.1±4.8	14.94 (11.17 to 18.71)	<0.001
Vc% pred.	43.4±9.6	61.4±8.3	-18.27 (-22.89 to -13.64)	<0.001
mean breath-hold time 1	5.1±0.3	5.1±0.3	0.02 (-0.10 to 0.13)	0.780
mean breath-hold time 2	11.1±0.5	11.0±0.5	0.09 (-0.12 to 0.29)	0.401

AMD, absolute mean difference (adjusted for age, gender, BMI, and prevalence of comorbidities; DL_{CO}, diffusing capacity for carbon oxide; FVC, forced vital capacity; FEV_1, forced expiratory volume in 1 sec; Hb, hemoglobin; K_{CO}, Carbon Monoxide Transfer Coefficient; RSV, respiratory syncytial virus; sDL_{CO}, single-breath simultaneous diffusing capacity for carbon oxide; sDL_{NO}, single-breath simultaneous diffusing capacity for nitric oxide; SpO_2, O_2 saturation; TLC, total lung capacity; VC, vital capacity; Vc, lung capillary blood volume.

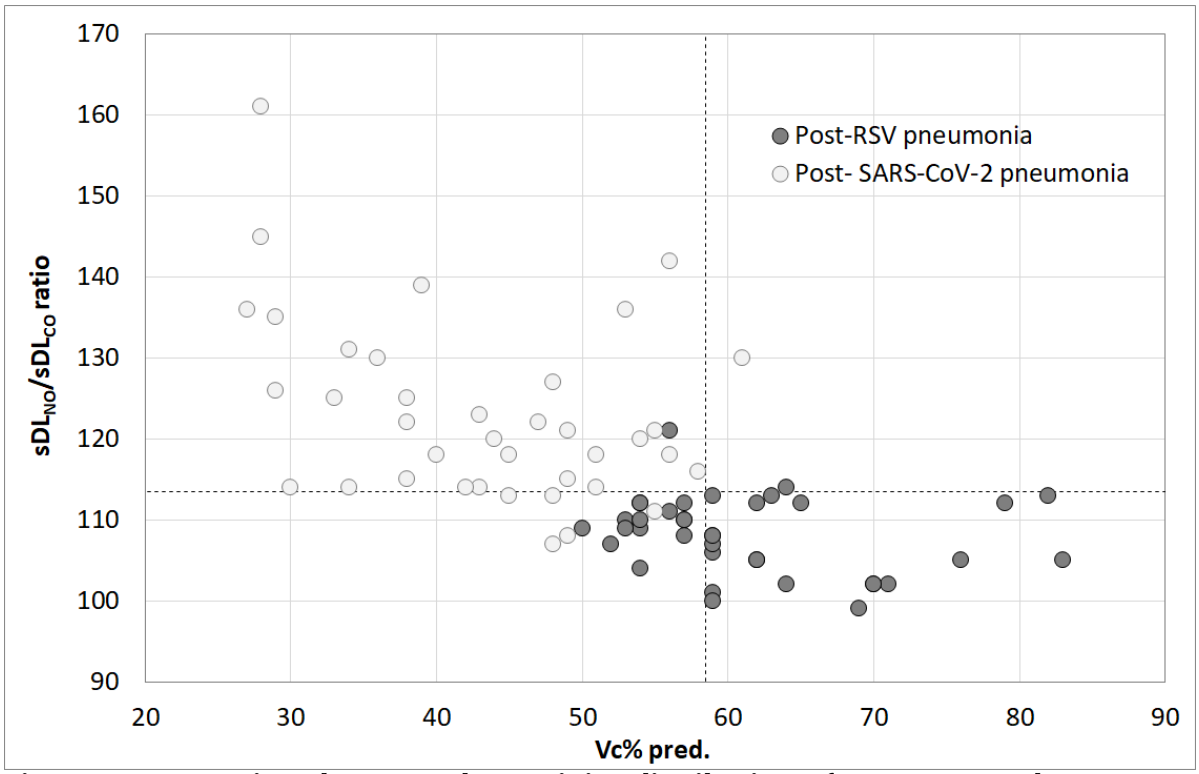

Figure 1. Comparison between the conjoint distribution of Vc% pre. and DL_{NO}/DL_{CO} ratio and the optimal cut-off previously calculated reported as dotted lines (113.5 for sDL_{NO}/sDL_{CO} ratio and 58.5 for Vc% pred.). RSV, respiratory syncytial virus; sDL_{CO}, single-breath simultaneous diffusing capacity for carbon oxide; sDL_{NO}, single-breath simultaneous diffusing capacity for nitric oxide; Vc, lung capillary blood volume.

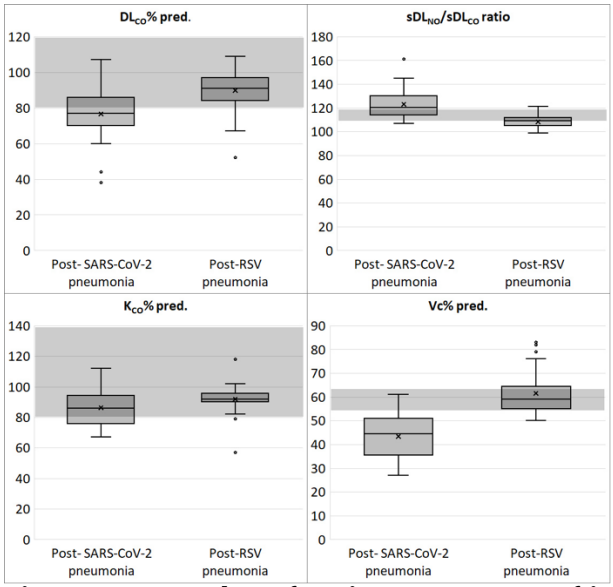

Figure 2. Box-plot of main parameters of interest (gray area represents the range of normal values). DL_{CO}, diffusing capacity for carbon oxide; K_{CO}, carbon monoxide transfer coefficient; RSV, respiratory syncytial virus; sDL_{CO}, single-breath simultaneous diffusing capacity for carbon oxide; sDL_{NO}, single-breath simultaneous diffusing capacity for nitric oxide; Vc, lung capillary blood volume.