

A Study to Evaluate Pulmonary Function Test in Post Covid Patients

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ABSTRACT

Background: Coronavirus disease 2019 (COVID-19), caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), primarily affects the respiratory system, and emerging evidence suggests that pulmonary impairment may persist well beyond the acute illness. Spirometric indices, particularly forced vital capacity (FVC), forced expiratory volume in 0.5 second (FEV0.5) and forced expiratory volume in one second (FEV1), provide an objective, reproducible means of quantifying such residual dysfunction. **Aim:** To evaluate and compare FVC, FEV0.5 and FEV1 in patients with a past history of moderate-to-severe COVID-19 infection against age and sex-comparable controls without such history. **Materials and Methods:** A retrospective case-control study was conducted on 70 subjects (35 cases with a past history of RT-PCR/rapid-antigen-test-confirmed COVID-19 infection and 35 controls), aged 18–55 years, recruited over one year. Smokers and individuals with pre-existing pulmonary, cardiac or malignant disease were excluded. Spirometry was performed using a computerised spirometer (RMS-HELIOS-702), and the best of three technically acceptable efforts was recorded. Continuous variables were expressed as mean values and compared using analysis of variance (ANOVA) with the least-significant-difference (LSD) post-hoc test; $p < 0.05$ was taken as significant. **Results:** The mean FVC was significantly lower in cases (2.47 L) than controls (2.80 L; $p = 0.049$). FEV0.5 was significantly reduced in cases (1.82 L) compared with controls (2.13 L; $p = 0.018$), and FEV1 was significantly lower in cases (2.30 L) than controls (2.67 L; $p = 0.033$). Other spirometric indices; FEV3, peak expiratory flow rate, FEF25–75%, FEF25%, FEF50%, FEF75%, the derived ratios (FEV0.5/FVC, FEV1/FVC, FEV3/FVC), maximum voluntary ventilation and its time, FVC time and extrapolated time did not differ significantly between groups. **Conclusion:** A past history of moderate-to-severe COVID-19 infection is associated with a significant, restrictive-pattern reduction in FVC, FEV0.5 and FEV1 that persists for months after recovery, while flow-dependent and ratio-based indices remain relatively preserved. These findings support structured post-COVID pulmonary follow-up and reinforce the value of simple, low-cost spirometric screening in survivors.

Keywords: COVID-19; spirometry; forced vital capacity; forced expiratory volume; pulmonary function tests; post-COVID sequelae.

INTRODUCTION

Spirometry is a physiological test that measures the volume and flow of air that can be inhaled and exhaled in relation to time, and it remains the most widely used screening tool for general respiratory health.¹ The

manoeuvre comprises three phases: maximal inspiration, a forceful ‘blast’ of exhalation, and continued exhalation to the end of test and is governed by well-defined criteria of within-manoeuvre acceptability and between-manoeuvre reproducibility.¹ Pulmonary function tests

(PFTs) allow physicians to objectively evaluate respiratory function; although the results are effort-dependent, they are reproducible and, when interpreted together with history, physical examination and laboratory data, help clinicians reach a diagnosis, quantify disease severity, follow its course over time, and assess response to treatment.² Among the several objective techniques used for functional respiratory evaluation, spirometry, lung volume measurement and diffusion capacity spirometry remains the most accessible and widely applied.³ Interest in the quantitative evaluation of pulmonary function in cardiopulmonary disease has grown steadily over the past several decades,⁴ and this interest has been further amplified by the emergence of coronavirus disease 2019 (COVID-19).

The measurement of lung function has a long developmental history. Hutchinson first devised the spirometer and measured vital capacity, demonstrating its relationship to height, age, weight and disease.⁵ Gaensler subsequently proposed measuring forced expiratory volume over a constant time interval the 'timed vital capacity' and the ratio of this timed capacity to total vital capacity, a concept that evolved into the modern FEV1/FVC ratio.⁶ Tiffeneau and Pinelli proposed that the volume expired in the first second of a forced vital-capacity manoeuvre was the key ventilatory index for evaluating pulmonary function in chronic respiratory disease,⁷ while Strohl advocated the use of forced vital capacity itself as a standard determinant of lung function.⁸ These early observations laid the foundation for the modern, standardised approach to spirometric testing later codified by international bodies such as the American Thoracic Society and European Respiratory Society.² Today, FVC the maximal volume of air that can be forcefully and rapidly exhaled following maximal inspiration together with the forced expiratory volumes at fixed time points, particularly FEV0.5 and FEV1, remain the principal indices used to characterise ventilatory function and to distinguish obstructive from restrictive patterns of lung disease.⁹

On 11 March 2020, the World Health Organization declared COVID-19 a pandemic.¹⁰ COVID-19 is a respiratory illness caused by the novel coronavirus SARS-CoV-2, and early epidemiological data indicated that a substantial proportion of hospitalised patients developed rapid and progressive respiratory failure resembling acute respiratory distress syndrome.^{11,12} Radiological studies described characteristic ground-glass opacities and consolidative changes on chest computed tomography even in asymptomatic individuals,¹³ raising early concern that pulmonary parenchymal injury might evolve into fibrosis and that lung function should be assessed together with imaging

follow-up.^{14,15} A growing body of literature has since examined pulmonary function in COVID-19 survivors. Mo et al. reported that impairment of diffusing capacity was the commonest abnormality among discharged patients, followed by a restrictive ventilatory defect, both of which correlated with disease severity.¹⁶ You et al. described divergent trajectories of recovery in critically ill survivors, related to age.¹⁷ Frija-Masson et al. found that more than half of patients with COVID-19 pneumonia had abnormal lung function 30 days after symptom onset, independent of the radiological extent of pneumonia.¹⁸ Torres-Castro et al., in a systematic review, concluded that diffusion capacity was the most frequently affected parameter, being reduced in nearly 40% of patients, although restrictive and obstructive spirometric patterns were also documented.¹⁹ Huang et al. observed a reduction of diffusing capacity in 75% of patients at 30-day follow-up, and Zhao et al. reported a comparable reduction in diffusing capacity in 25% of patients at three months.^{20,21} Persistent symptoms lasting a mean of 60 days after onset were described by Carfi et al. in patients discharged after COVID-19 hospitalisation.²² More directly relevant to the present work, Fumagalli et al. found clinically relevant, mainly restrictive alterations in pulmonary function tests among COVID-19 pneumonia survivors, Soleymani Babadi et al. reported a restrictive pattern in over 40% of patients six to twelve weeks after discharge for severe disease, and Stylemans et al. observed that pulmonary restriction could persist, or even worsen, beyond six months in a subset of patients despite near-normalisation of chest CT changes.^{23,24,25} In contrast, Modi et al., and Lewis et al. reported essentially preserved spirometric values in non-critically ill cohorts, illustrating that the degree of residual impairment is heterogeneous and appears to depend on illness severity, timing of assessment and the population studied.^{26,27} Most recently, Antje et al. found that symptomatic post-COVID patients had significantly lower FVC and total lung capacity than asymptomatic patients, underscoring a dose-response relationship between clinical burden and residual lung-function deficit.²⁸

Experience from earlier coronavirus epidemics reinforces the plausibility of durable pulmonary sequelae. Survivors of severe acute respiratory syndrome (SARS) showed persistently reduced exercise capacity and quality of life at six months and one year after recovery, with a measurable minority exhibiting impaired diffusing capacity.^{29,30} Ong et al. similarly documented reduced exercise capacity and persistent pulmonary function impairment in about one-third of SARS survivors at one year and Ngai et al. showed that reductions in diffusing capacity could persist for up to two years after infection.³¹⁻³³ These precedents suggest

that SARS-CoV-2 infection, like its predecessor coronaviruses, has the potential to produce measurable and clinically relevant long-term pulmonary sequelae, plausibly related to diffuse alveolar epithelial and endothelial injury with secondary fibroproliferation and a predisposition to chronic alveolar remodelling and fibrosis.³⁴ Despite this expanding literature, data specifically examining the two shortest-interval expiratory indices FEV0.5 and FEV1 alongside FVC in an Indian case–control population remain limited. As the lungs are among the organs most severely affected by COVID-19, and given the heterogeneity of findings across published cohorts, the present study was undertaken to evaluate alterations in pulmonary function, with particular attention to FVC, FEV0.5 and FEV1, in patients with a past history of moderate-to-severe COVID-19 infection compared with COVID-19-naïve controls.

MATERIALS AND METHODS

This was a retrospective case–control study conducted in the Postgraduate Department of Physiology, Government Medical College and Associated Hospital, Jammu, over a period of one year, after obtaining approval from the Institutional Ethics Committee (IEC/GMC/2022/870, dated 10/02/2022).

Study population: A total of 70 subjects were enrolled 35 cases and 35 controls, matched for the 18–55 year age range. The case (study) group comprised patients with a past history of COVID-19 confirmed by reverse-transcriptase polymerase chain reaction (RT-PCR) or rapid antigen test (RAT), who had experienced moderate-to-severe symptoms as defined by national criteria (moderate disease: respiratory rate >24/min or SpO₂ 90–<93% on room air; severe disease: respiratory rate >30/min or SpO₂ <90% on room air). The control group was drawn from outpatients attending the Departments of ENT, Ophthalmology, Dermatology, Surgery and Orthopaedics of the same institution, who had no history of COVID-19 infection.

Inclusion criteria: patients diagnosed with COVID-19 with moderate-to-severe symptoms; both sexes; and age between 18 and 55 years were included

Exclusion criteria: were current smokers; a history of chronic obstructive pulmonary disease, pulmonary tuberculosis or other chronic lung disease; a history of cardiac illness; malignancy; and unwillingness to participate. All subjects gave written informed consent after a detailed explanation of the study procedure.

Clinical and anthropometric assessment: A detailed history and general physical and chest examination (including chest radiography where indicated) were carried out for every subject. Age was recorded in completed years, weight was measured on a calibrated weighing machine with the subject barefoot and standing with weight evenly distributed and height was measured with a standardised stadiometer.³⁵ Body mass index was calculated using the Quetelet index (weight in kg / height in m²), and body surface area was calculated using the Mosteller formula.

Spirometric measurement: Pulmonary function testing was performed using a computerised spirometer (RMS-HELIOS-702), a portable device with battery back-up, built-in thermal printer and pre/post-medication comparison capability, already available in the department. Subjects were first familiarised with the mouthpiece and breathing manoeuvre before formal recording. Testing was carried out in the sitting position at the same time of day for each subject, and the best of three technically acceptable efforts was taken for analysis, in accordance with standard recommendations.^{2,10}

The principal parameters recorded were:

- Forced vital capacity (FVC), the maximal volume of air that can be exhaled as forcefully and rapidly as possible following a maximal inspiration.¹⁰
- Forced expiratory volume in 0.5 second (FEV0.5), the volume exhaled in the first half-second of the forced manoeuvre.
- Forced expiratory volume in one second (FEV1), the volume of air that can be forcefully and maximally exhaled in the first second after maximal inspiration; FEV1 normally represents 70–80% of FVC (age-dependent) and is regarded as the single most validated and clinically useful index for assessing air-flow limitation and for distinguishing obstructive from restrictive lung disease.²

In addition, forced expiratory volume in three seconds (FEV3), peak expiratory flow rate (PEFR), mean forced expiratory flow between 25% and 75% of FVC (FEF25–75%), instantaneous flows at 25%, 50% and 75% of FVC (FEF25%, FEF50%, FEF75%), the timed-volume-to-FVC ratios (FEV0.5/FVC, FEV1/FVC, FEV3/FVC), maximum voluntary ventilation (MVV) and its associated time, FVC time, and extrapolated time were recorded for completeness and for comparison with the literature, although the present paper focuses on FVC, FEV0.5 and FEV1 as the primary indices of interest. The FEV1/FVC ratio (Tiffeneau–Pinelli index) is conventionally reported in the range of 75–85% and is used to differentiate an obstructive pattern, in which the ratio falls because FEV1 declines disproportionately to

FVC, from a restrictive pattern, in which the ratio remains normal or increases because the fall in FVC equals or exceeds the fall in FEV1.^{36,37}

Statistical analysis: Data were entered in Microsoft Excel and analysed using SPSS version 20.0 (SPSS Inc., Chicago, IL, USA). Continuous variables were expressed as mean values, and categorical variables as percentages. Analysis of variance (ANOVA) was used for inter-group comparison, with the least-significant-difference (LSD) test applied for multiple comparisons; the chi-square test or Fisher's exact test was used for categorical variables as appropriate. A two-tailed p-value of less than 0.05 was considered statistically significant.

RESULTS

Seventy subjects completed the study, 35 cases with a past history of moderate-to-severe COVID-19 infection and 35 controls without such history. The two groups were comparable for age, with no statistically significant difference in mean age between them, so that the observed differences in pulmonary function could not be attributed to age mismatch.

Of all the spirometric parameters assessed, three showed a statistically significant reduction in the case group compared with controls: FVC, FEV0.5 and FEV1. The remaining parameters FEV3, PEFR, FEF25–75%, FEF25%, FEF50%, FEF75%, FEV0.5/FVC%, FEV1/FVC%, FEV3/FVC%, MVV, MVV time, FVC time and extrapolated time did not differ significantly between the two groups.

Forced vital capacity (FVC): The mean FVC was 2.47 L in the case group and 2.80 L in the control group. This inter-group difference was statistically significant ($p =$

0.049), indicating a measurable reduction in vital capacity among individuals with a history of moderate-to-severe COVID-19 infection.

Forced expiratory volume in 0.5 second (FEV0.5): The mean FEV0.5 was 1.82 L in cases and 2.13 L in controls. This difference was statistically significant ($p = 0.018$), representing the parameter with the strongest statistical separation between groups among the three primary indices studied.

Forced expiratory volume in one second (FEV1): The mean FEV1 was 2.30 L in cases and 2.67 L in controls. This difference was also statistically significant ($p = 0.033$).

For comparison, the mean FEV3 was 2.47 L in cases and 2.76 L in controls (not significant); mean PEFR was 5.60 L/s in cases and 5.75 L/s in controls (not significant); mean FEF25–75% was 4.03 L/s versus 4.19 L/s (not significant); FEF25%, FEF50% and FEF75% were 5.29, 4.26 and 2.62 L/s in cases versus 5.56, 4.49 and 3.01 L/s in controls respectively (all not significant); FEV0.5/FVC% was 75.13% versus 79.61%; FEV1/FVC% was 93.33% versus 97.22%; and FEV3/FVC% was 99.98% versus 100% (all not significant). MVV (67.78 vs 61.71 L/min), MVV time (0.82 vs 0.77 s) and extrapolated time (0.13 vs 0.11 s) likewise showed no significant inter-group difference. Because the FEV1/FVC ratio itself was not reduced, indeed it remained within the physiologically expected range in both groups, the pattern of change observed (a proportionate fall in FVC accompanied by a fall in the timed expiratory volumes, with a preserved or high timed-volume-to-FVC ratio) is characteristic of a restrictive, rather than obstructive, ventilatory defect.

Table 1. Comparison of significance of key spirometric parameters between the present study and selected published post-COVID-19 cohorts (S = statistically significant; NS = not significant; DNF = data not found in the cited study).

Parameter	Fumagalli et al. ²³ (N=13)	Modi et al. ²⁶ (N=52)	Lewis et al. ²⁷ (N=80, retrospective)	Present study (Cases=35, Controls=35)
FVC	S	NS	NS	S
FEV0.5	DNF	DNF	DNF	S
FEV1	S	NS	NS	S
FEV3	NS	NS	NS	NS
PEFR	NS	NS	NS	NS
FEF25–75%	NS	NS	NS	NS
FEV1/FVC%	NS	NS	NS	NS
MVV	NS	NS	NS	NS

Table 1 places the present findings for FVC and FEV1 in the context of previously published case series and cohort studies of post-COVID-19 pulmonary function; comparable published data for FEV0.5 in this setting were not identified, underscoring the relative novelty of reporting this specific index in the post-COVID-19 population.

DISCUSSION

The present case-control study found statistically significant reductions in FVC, FEV0.5 and FEV1 in patients with a past history of moderate-to-severe COVID-19 infection compared with controls, while all flow-dependent parameters (PEFR, FEF25–75%, FEF25%, FEF50%, FEF75%), the timed-volume ratios (FEV0.5/FVC%, FEV1/FVC%, FEV3/FVC%), FEV3, MVV and its associated timing indices were unaffected. This pattern, a fall in volume-based indices with preservation of flow-dependent indices and ratios is the classical spirometric signature of a restrictive, rather than obstructive, ventilatory defect and is consistent with the hypothesis that COVID-19 pneumonia produces residual parenchymal restriction, plausibly on a background of alveolar epithelial and endothelial injury with fibroproliferative repair, rather than airway obstruction.^{2,37,38}

Forced vital capacity is considered the single most sensitive spirometric index of alterations in lung elasticity and mechanical properties, and is of established value in the diagnosis, prognosis and monitoring of respiratory insufficiency.³⁷ The significant reduction in FVC observed here (2.47 L in cases versus 2.80 L in controls; $p = 0.049$) is concordant with the findings of Fumagalli et al., who similarly reported significantly reduced FVC among COVID-19 pneumonia and is broadly consistent with the restrictive pattern described by Torres-Castro et al. and Soleymani Babadi et al.^{19,23,24} It contrasts, however, with the normal FVC values reported by Modi et al. and Lewis et al.^{26,27} This divergence most plausibly reflects differences in illness severity and follow-up interval across studies: the present cohort was recruited from patients hospitalised with moderate-to-severe disease and tested roughly six months after recovery, whereas cohorts reporting normal spirometry often included a larger proportion of non-critically-ill or milder illness and, in the case of Lewis et al., a retrospective design with variable timing of assessment.²⁷ These observations align with Antje et al.'s demonstration of a graded relationship between symptom burden and the degree of residual FVC and total-lung-capacity deficit, suggesting that the severity of the acute illness rather than infection per se is the principal determinant of persistent volume restriction.²⁸

FEV0.5 showed the largest relative separation between groups ($p = 0.018$) among the three significant parameters, yet comparable data for this index in post-COVID-19 populations could not be identified in the

reviewed literature, most published series having reported FEV1 and FVC without a distinct 0.5-second value. Because FEV0.5 captures the earliest phase of forced expiration and is less dependent on total lung volume than FEV1, its significant reduction in the present cohort strengthens the inference that early expiratory flow, and not merely total lung capacity, is affected in convalescent COVID-19 patients, and suggests that FEV0.5 may merit wider reporting as a sensitive, easily obtained marker of post-COVID-19 pulmonary compromise, particularly in settings where diffusion-capacity testing is unavailable. The reduction in FEV1 (2.30 L versus 2.67 L; $p = 0.033$) mirrors the pattern seen for FVC and is again concordant with Fumagalli et al but discordant with Modi et al and Lewis et al., for the reasons discussed above.^{23,26,27} Because the FEV1/FVC ratio was not reduced in the present study (93.33% in cases versus 97.22% in controls, both within or above the conventionally cited normal range of 75–85%³⁶), the fall in FEV1 appears to be a consequence of the fall in FVC rather than of airway obstruction, reinforcing the interpretation of a predominantly restrictive functional pattern. FEV1 is regarded as the single most validated and clinically useful spirometric index for assessing air-flow limitation and its reduction here, together with the concordant reduction in FVC and FEV0.5, provides internally consistent evidence of restrictive impairment rather than an isolated or artefactual finding in any one parameter.²

The absence of significant change in FEV3, PEFR, the mid-expiratory flows (FEF25–75%, FEF25%, FEF50%, FEF75%) and MVV suggests that the mid-to-late phase of forced expiration and overall ventilatory reserve were relatively preserved, which is compatible with restrictive rather than obstructive pathology, since obstructive disease characteristically produces disproportionate reductions in flow rates and in the FEV1/FVC ratio.³⁷ An isolated reduction in the FEV3/FVC ratio has previously been proposed as a marker of early, mild lung injury with hyperinflation or air-trapping; because FEV3/FVC was essentially unchanged in the present study (99.98% versus 100%), there was no spirometric evidence of an early obstructive or air-trapping component in this cohort. These findings are broadly consistent with the pattern of pulmonary sequelae described after earlier coronavirus epidemics. Survivors of SARS demonstrated persistently reduced exercise capacity and, in a minority, impaired gas-transfer function at six months to one year after recovery and

reductions in diffusing capacity have been documented for up to two years after SARS infection.²⁹⁻³³ Although the present study did not measure diffusing capacity, the demonstration of restrictive volume loss on simple spirometry six months after COVID-19 recovery parallels this precedent and supports the recommendation, echoed across the post-COVID literature that survivors of moderate-to-severe disease should undergo structured, longitudinal pulmonary function follow-up rather than a single post-discharge assessment, since Stylemans et al. showed that restriction identified at an early follow-up visit could persist, or even worsen, beyond six months despite radiological improvement.^{18,25}

Limitations of study

The present findings should be interpreted in light of several limitations. The study was restricted to a defined age band of 18–55 years, so the applicability of these findings to children and older adults, who may show different patterns of recovery, is uncertain; widening the age range in future work would be informative. Testing was performed at a single time point, approximately six months after recovery from infection, rather than with paired pre-infection and post-infection measurements in the same subjects, which was not feasible within the resources of the present laboratory; a paired design, as used in some previous studies, would more precisely quantify the magnitude of individual decline. In addition, only standard spirometric parameters were evaluated, and the possible influence of bronchodilator or other medication use on the observed indices was not separately assessed; future studies incorporating bronchodilator-response testing may help clarify whether any reversible component contributes to the deficits observed.

Strengths of the Study

The strengths of this study include a case–control design with an age-comparable control group, objective assessment using spirometry, and evaluation of multiple pulmonary function parameters. In particular, assessment of FEV0.5 provides additional information on early expiratory function, which has been less frequently reported in post-COVID-19 studies.

Future Implications

Future studies should include larger and more diverse populations with longitudinal follow-up and, where feasible, pre-infection or pre-COVID-19 pulmonary function measurements. Incorporation of lung-volume measurements and diffusion capacity testing may further clarify the nature and reversibility of post-COVID-19 pulmonary impairment.

CONCLUSION

In this retrospective case–control study, a past history of moderate-to-severe COVID-19 infection was associated with statistically significant reductions in three key spirometric indices forced vital capacity, forced expiratory volume in 0.5 second, and forced expiratory volume in one second — approximately six months after recovery, while flow-dependent parameters, timed-volume ratios and maximum voluntary ventilation remained unaffected. This pattern is consistent with a predominantly restrictive ventilatory defect, most likely reflecting residual parenchymal changes such as fibroproliferation following COVID-19 pneumonia, and is in keeping with, and extends, prior reports describing restrictive pulmonary impairment in COVID-19 survivors. These results support the incorporation of simple, low-cost spirometric assessment including the less commonly reported FEV0.5 into structured post-COVID-19 follow-up protocols, and highlight the need for larger, longitudinal studies across a wider age range to define the natural history, reversibility and clinical significance of these residual deficits.

Declarations

Ethical Approval

The study was conducted in accordance with the ethical principles applicable to research involving human participants and was approved by the Institutional Ethics Committee of Government Medical College and Associated Hospital, Jammu (Approval No. IEC/GMC/2022/870, dated 10 February 2022).

Informed Consent

Written informed consent was obtained from all participants before enrolment in the study. Participants were informed about the study procedures and their right to participate voluntarily.

Consent for Publication

Not applicable. No personally identifiable information or individual patient images are included in this manuscript.

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Conflict of Interest

The authors declare that they have no conflict of interest.

Author Contributions

All authors contributed to the conception and design of the study, data collection, analysis and interpretation of the findings, drafting or critical revision of the

manuscript, and approval of the final version for publication.

Data Availability Statement

The data supporting the findings of this study are available from the corresponding author upon reasonable request, subject to institutional and ethical considerations.

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