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COEXISTENCE OF COPD AND SILICA-RELATED PNEUMOCONIOSIS (SILICOSIS): A COMPARATIVE EVALUATION OF LUNG FUNCTION AND QUALITY OF LIFE

Respiratory Medicine

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ABSTRACT

Background: Silicosis adds parenchymal fibrosis to obstructive airway disease, potentially worsening gas exchange and health status in silica-exposed workers. Evidence in young, non-smoking Indian stone-workers remains limited. **Objectives:** To compare lung function and health-related quality of life (QoL) between COPD alone and COPD with coexisting silicosis, and to examine structure–function–symptom relationships. **Methods:** Analytical cross-sectional study of 100 consecutive male stone-workers (Group A: COPD, n=50; Group B: COPD+silicosis, n=50). Assessments: post-bronchodilator spirometry, hemoglobin-adjusted DLCO, HRCT severity scoring, CAT, SGRQ. Analyses: t-tests, ANCOVA, Spearman correlations, multivariable regression (Jamovi/SPSS; α=0.05). **Results:** Compared with COPD, COPD+silicosis had lower FEV % (61.3 vs 68.4, p=0.006) and DLCO% (60.2 vs 72.1, p<0.001), worse CAT/SGRQ (22.4/46.9 vs 18.2/37.5, p<0.001), and higher mMRC≥2 (54% vs 32%, p=0.030). **Conclusion:** In non-smoking stone-workers, silicosis coexisting with COPD confers independent, clinically meaningful decrements-predominantly diffusion impairment-and poorer QoL. Integrated pathways combining spirometry, DLCO, and standardized HRCT are warranted in Tier-II Indian occupational settings. prioritize pulmonary rehabilitation ; for public awareness, emphasize wet-cutting, fit-checked respirators, and annual lung-function surveillance.

KEYWORDS

COPD, Silicosis, DLCO, Spirometry, HRCT, CAT, SGRQ, Occupational exposure, Quality of life (QoL).

INTRODUCTION

Airflow depends on small-airway patency, elastic recoil, and gas-exchange surfaces; injury from noxious inhalants disrupts these elements, producing obstruction, hyperinflation, and impaired diffusion. In silica-exposed workers, smoking and silicosis act jointly to intensify lung damage, magnifying risks for chronic respiratory disease beyond either factor alone [1]. COPD features persistent airflow limitation from peripheral airway narrowing/remodeling and emphysematous destruction, while silicosis is a fibrotic pneumoconiosis driven by crystalline silica. Among silicotic workers, stopping smoking is associated with less airflow obstruction, emphasizing a modifiable determinant of disease trajectory [2]. Outside high-income settings, artisanal mining communities show notable rates of silicosis and impaired spirometry, underscoring surveillance gaps in exposed populations [3].

COPD's inflammatory–remodeling pathways intersect with carcinogenesis; clinical and genetic links with lung cancer frame COPD as a systemic risk state requiring vigilant assessment in exposure-affected cohorts [4]. Standardized lung-function testing—spirometry, lung volumes, and DLCO—offers objective, comparable metrics across respiratory disorders and is crucial when COPD coexists with interstitial fibrosis [5]. In silicosis, declines in FEV_1 and gas-transfer capacity relate variably to radiographic severity, arguing for integrated physiologic–radiologic evaluation rather than reliance on imaging alone [6]. High-resolution CT (HRCT) sensitively detects nodular profusion, emphysema, and fibrotic change, with qualitative and quantitative readouts that correlate with clinical limitation-thereby complementing spirometry and DLCO in exposure-related disease [7].

Within exposure-affected settings, tuberculosis intersects with silicosis epidemiology and care coordination, but does not solely drive functional decline, which remains principally exposure-mediated [8]. Clinically, health status in COPD tracks spirometric impairment, reinforcing the need to pair physiology with patient-reported outcomes in exposed cohorts [9]. Field surveys among Rajasthani stone-miners highlight unsafe work practices and poor disease awareness, signaling missed opportunities for prevention and surveillance [10]. Pulmonary rehabilitation is recommended in pneumoconiosis to improve health-related quality of life and functional capacity beyond pharmacotherapy [11]. Data in Indian construction workers show lower lung function and worse respiratory QoL versus matched controls [12].

MATERIALS AND METHOD

Study Area:
Kamla Nehru Chest Hospital, Jodhpur (Dr. S.N Medical College, Jodhpur)

Study Design: Observational study

Study Population: Males, Stone workers

Study Duration: One Year

Sampling Procedure:
Consecutive adult male stone-workers attending OPD at Kamla Nehru Chest Hospital, Jodhpur

Sampling Method:
Stratified consecutive sampling (1:1) over one year at the OPD of Kamla Nehru Chest Hospital, Jodhpur (Dr. S.N. Medical College). All consecutive adult male stone-workers (>18 years) with ≥5 years of silica exposure who attended OPD and provided consent were screened against inclusion/exclusion criteria (no active/post-tuberculosis; no other chronic lung disease or cardiac failure). After a uniform diagnostic pathway—post-bronchodilator spirometry per ATS/ERS 2019, single-breath DLCO with same-day hemoglobin adjustment per ATS/ERS 2017, and standardized HRCT chest read independently by two blinded radiologists with a semi-quantitative severity score—participants were classified as Group A (COPD without silicosis) or Group B (COPD with silicosis). Enrollment proceeded consecutively within each stratum until approximately 50 participants per group were achieved. Irredeemable test-quality failures after one repeat were replaced by the next consecutive eligible OPD attendee. A screening log documented approached, eligible, enrolled, and excluded cases with reasons.

- Group A: COPD without silicosis (≈50).
- Group B: COPD with silicosis (≈50).

Sample Size & Rationale:
Two-group comparison for DLCO % predicted. Assumptions: σ=6, Δ=4, α=0.05, power 80 percent gives ≈36 per group. Allowing unusable tests and nonresponse, total sample fixed at **100**. Calculated in G*Power 3.1 and checked in Jamovi.

Sample Selection:
Inclusion Criteria:
1. >18 years Males
2. Stone worker ≥5 year silica exposure
3. Informed Consent.

Exclusion Criteria:
1. Age < 18 years
2. Smoker (current/ever)
3. Active-TB and Post-TB
4. Other chronic lung disease/cardiac failure

Materials And Measures:
1. Spirometry post-bronchodilator per ATS/ERS.
2. Single-breath DLCO with hemoglobin adjustment.
3. HRCT chest with standardized protocol and blinded dual read plus semi-quantitative severity score.
4. Questionnaires: CAT, SGRQ, mMRC.
5. Structured case report forms and secure database.

Statistical Analysis:
Jamovi or SPSS. Descriptives as mean with SD or median with IQR and counts with percentages. Normality by Shapiro–Wilk and variance by Levene. Group comparisons by t test or Mann–Whitney. Adjusted comparisons by ANCOVA. Correlations by Spearman between HRCT severity, physiology, and quality of life. Multivariable linear regression to identify predictors of poorer quality of life. Two-sided alpha 0.05. Sensitivity analysis excluding borderline obstruction.

RESULTS

Table 1: Lung Function (post-bronchodilator) and Gas Transfer

Outcome	COPD only (n=50)	COPD + Silicosis (n=50)	Mean Difference (95% CI)	p-value
FEV % predicted	68.4 ± 12.1	61.3 ± 13.0	7.1 (2.1 to 12.1)	0.006
FEV /FVC (ratio)	0.59 ± 0.05	0.56 ± 0.05	0.03 (0.01 to 0.05)	0.003
DLCO % predicted (Hb-adjusted)	72.1 ± 10.9	60.2 ± 12.0	11.9 (7.2 to 16.6)	<0.001

Table 1: Compared with COPD alone, the COPD + silicosis group showed significantly lower FEV % (61.3 vs 68.4; Δ 7.1%, p=0.006) and FEV /FVC (0.56 vs 0.59; Δ 0.03, p=0.003), indicating added obstructive burden. The largest difference was in DLCO% (60.2 vs 72.1; Δ 11.9%, p<0.001), reflecting substantial diffusion impairment. Overall, silicosis coexisting with COPD confers clinically meaningful additional physiological deficit.

Table 2: Health Status / Quality of Life

Outcome	COPD only (n=50)	COPD + Silicosis (n=50)	Mean/Proportion Difference (95% CI)	p-value
CAT total (0–40)	18.2 ± 5.6	22.4 ± 6.1	4.2 (1.9 to 6.5)	<0.001
SGRQ total (0–100)	37.5 ± 12.8	46.9 ± 13.6	9.4 (4.1 to 14.7)	<0.001
mMRC ≥2, n (%)	16 (32.0)	27 (54.0)	+22.0% (3.1% to 40.9%)	0.030

Table 2: Quality of life was poorer when silicosis coexisted with COPD. CAT scores were higher (22.4 vs 18.2; Δ 4.2, p<0.001) and SGRQ totals were worse (46.9 vs 37.5; Δ 9.4, p<0.001), indicating greater symptom burden and health-status impairment. Dyspnea grade ≥2 was more frequent (54.0% vs 32.0%; +22.0%, p=0.030). Overall, silicosis adds clinically meaningful QoL decrement beyond COPD alone.

Table 3: Structure–Function–Symptoms: HRCT Severity Correlations (All participants, N=100)

Variable Pair (HRCT severity vs…)	Spearman ρ	95% CI for ρ	p-value
DLCO % predicted	−0.52	−0.66 to −0.34	<0.001
FEV % predicted	−0.33	−0.49 to −0.15	0.001
CAT total	+0.41	+0.23 to +0.56	<0.001
SGRQ total	+0.47	+0.30 to +0.61	<0.001

Table 3: HRCT severity correlated most strongly with impaired diffusion (DLCO; ρ=−0.52, p<0.001), modestly with airflow (FEV ; ρ=−0.33, p=0.001), and moderately with worse symptoms/health status (CAT ρ=+0.41; SGRQ ρ=+0.47; both p<0.001). This structure–function–symptom pattern indicates greater silicotic burden tracks with physiologic decline and poorer QoL, supporting its use in predictive modeling.

Figure 1: After adjustment for age, BMI, silica-exposure years, and mMRC, patients with coexisting silicosis showed significantly worse outcomes than COPD alone. FEV % predicted was lower by 5.6 points (p=0.010), and the largest decrement was in DLCO% predicted (−9.2 points; p<0.001), indicating greater diffusion impairment consistent with silicotic parenchymal involvement. SGRQ total was 6.5 units higher (p=0.003), exceeding the minimal clinically important difference, confirming a clinically meaningful deterioration in health status in this Indian worker cohort.

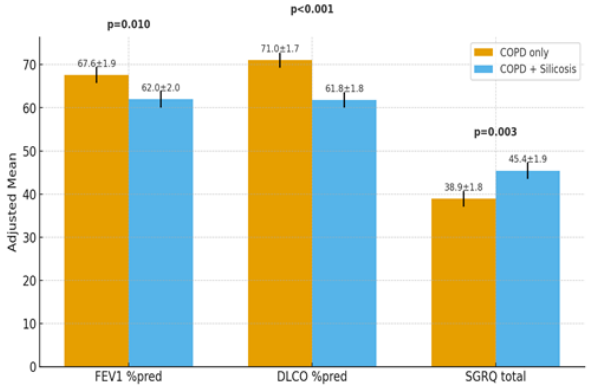

Figure 1: Adjusted Group Comparisons (ANCOVA)*

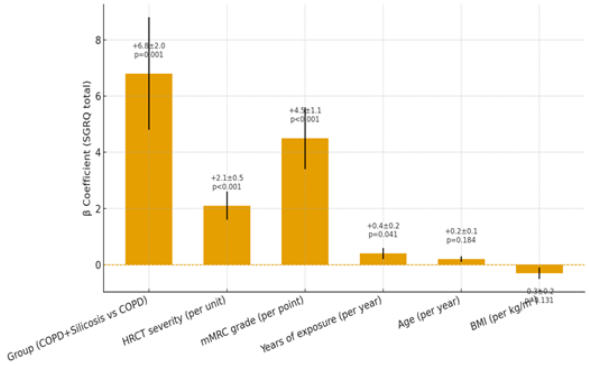

Figure 2: Predictors of Poorer Health Status (Multivariable Linear Regression

Figure 2: The bar plot of β coefficients (±SE) shows that coexisting silicosis independently worsens health status, increasing SGRQ by +6.8 points (p=0.001) versus COPD alone. Higher HRCT severity (+2.1 per unit; p<0.001) and greater dyspnea (mMRC) (+4.5 per grade; p<0.001) are strong determinants of poorer QoL. Exposure duration exerts a smaller yet significant effect (+0.4 per year; p=0.041), while age and BMI are non-significant. Collectively, structural disease burden, symptom intensity, and cumulative exposure primarily drive QoL impairment.

DISCUSSION

In this non-smoking cohort of male stone-workers with COPD, coexistence of silicosis produced a coherent, clinically important worsening across physiology and health status: FEV % was 61.3 versus 68.4 (Δ 7.1%, p=0.006), FEV /FVC 0.56 versus 0.59 (Δ 0.03, p=0.003), and DLCO% 60.2 versus 72.1 (Δ 11.9%, p<0.001), with parallel deterioration in CAT (22.4 vs 18.2; p<0.001), SGRQ (46.9 vs 37.5; p<0.001), and mMRC≥2 frequency (54.0% vs 32.0%; p=0.030). The additive effect of silica exposure on COPD physiology is consistent with exposure–impairment signals described in miner communities [3]. Health-status measures tracking lung-function impairment in Indian tertiary care reinforce the clinical salience of these differences [9]. The workplace context of stone-mining, including suboptimal dust control and limited awareness, plausibly contributes to the observed physiological gap [10]. Our TB-excluded design isolates the silica-related component while acknowledging tuberculosis as a relevant cofactor in similar populations [8].

The disproportionate diffusion loss is physiologically coherent: silicotic nodular fibrosis thickens the alveolar-capillary membrane and reduces capillary blood volume, and concomitant emphysematous destruction decreases effective surface area-together yielding a larger deficit in gas transfer than in airflow limitation [5]. HRCT severity aligned most with DLCO and also with symptoms, supporting a structure–function–symptom continuum suitable for predictive triage

[7]. For practice and policy in Tier-II services, the data support a diffusion-first pathway: routine post-bronchodilator spirometry with haemoglobin-adjusted DLCO at index assessment, standardized HRCT when diffusion is disproportionately reduced, and systematic CAT/SGRQ monitoring to guide escalation [9]. Rehabilitation should be offered given the quantified QoL burden and expected gains in pneumoconiosis [11]. Preventive emphasis belongs at the workplace-wet-cutting and fit-checked respirators-to address exposure roots, with periodic lung-function surveillance demonstrating exposure-linked decrements in Indian workers over time [12].

CONCLUSION

The coexistence of COPD and silicosis in non-smoking male stone-workers resulted in significant physiological impairment and poorer quality of life compared to COPD alone. The COPD + silicosis group showed lower FEV % (61.3 vs 68.4), reduced FEV /FVC (0.56 vs 0.59), and a greater decline in DLCO% (60.2 vs 72.1), along with worse CAT and SGRQ scores and increased dyspnoea severity (54.0% vs 32.0%). These findings align with studies in mining populations, where dust exposure leads to reduced lung function and impaired quality of life. The greater DLCO impairment underscores the need for diffusion-first diagnostic approaches. The results advocate for integrating Post- bronchodilator spirometry, DLCO, and standardized HRCT in clinical settings to enable early diagnosis and intervention. Pulmonary rehabilitation should be prioritized to improve quality of life, considering the significant symptom burden. Preventive measures like wet-cutting, fit-checked respirators, and regular lung-function surveillance are essential in reducing exposure and improving health outcomes. This study highlights the importance of early identification, rehabilitation, and workplace safety, informing policy and practice in occupational health for silica-exposed workers.

LIMITATION

Here are the limitations in of the study
1. Single-Center Design
2. Limited Sample Size**:**
3. Non-Inclusion of Female stone workers
4. No Longitudinal Follow-Up
5. Lack of Biomass Smoke and Second-Hand Smoke Data

Conflict Of Interest: None Declared

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