



# Evaluation of silicosis combined with type 2 diabetes mellitus based on the quantitative CT measured parameters

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**Background:** Type 2 diabetes mellitus (T2DM) and silicosis both have significant impacts on cardiopulmonary health. However, there is a lack of research investigating whether the presence of T2DM causes additional damage to the heart and lungs of patients with silicosis. This study aims to assess the alterations in pulmonary and cardiac structures, as well as lung function, in patients with silicosis who also have T2DM, and to explore the factors influencing lung function.

**Methods:** We included 30 silicosis patients with T2DM and 30 silicosis patients without diabetes. The two groups were matched by age, silicosis stage, smoking history, and dust exposure duration. Demographic details, occupational history, hematological results, computed tomography (CT)-measured cardiac and lung parameters, and pulmonary function test (PFT) results were collected. We compared these parameters between the two groups, evaluated the correlation between lung function and CT parameters in the diabetic group, and analyzed factors affecting lung function in this group.

**Results:** The silicosis combined with T2DM group showed significantly higher values for body mass index (BMI), the longest diameters from left to right of the left ventricle (LVLR), blood glucose, and triglycerides compared to the silicosis but without T2DM group (all  $P < 0.05$ ). The silicosis combined with T2DM group showed significantly lower total lung mass, and the ratio of forced expiratory volume in one second to forced vital capacity (FEV1/FVC) compared to the silicosis but without T2DM group (all  $P < 0.05$ ). In the silicosis with T2DM group, FEV1/FVC ratio showed significant correlations with total lung mass, the longest diameters from left to right of the left atrium, LVLR, and the longest diameters from left to right of the right atrium ( $r$  values were 0.51, 0.47, 0.40, and 0.44, respectively; all  $P < 0.05$ ). Multivariate analysis revealed that BMI and the LVLR were independent determinants of FEV1/FVC ratio in the silicosis with T2DM group ( $t$  values were  $-3.367$  and  $2.471$ , respectively; all  $P < 0.05$ ).

**Conclusions:** Diabetes mellitus induces structural changes in the lungs and heart of patients with silicosis and exacerbates the impairment of lung function. BMI and LVLR are key determinants of the FEV1/FVC ratio highlighting the need for enhanced comprehensive management strategies.

**Keywords:** Silicosis; type 2 diabetes mellitus (T2DM); quantitative computed tomography (quantitative CT); pulmonary function

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## Introduction

Pneumoconiosis is a type of occupational lung disease caused by long-term dust exposure (1), and this condition has led to an approximate 0.4% decline in the gross national product of China (2). Silicosis, the primary form of pneumoconiosis, is the most serious and common occupational disease in China and accounts for 88.3% of all reported cases of pneumoconiosis (3). The prevalence of type 2 diabetes mellitus (T2DM) is increasing globally every year (4). In similarity with silicosis, the prevalence of diabetes mellitus imposes an enormous burden on society, resulting in high medical costs for patients and their families and adverse health consequences for the affected individuals (5).

On the one hand, both T2DM and silicosis can cause heart damage, and it is thought that T2DM can directly affect both the structure and function of the myocardium, a condition referred to as diabetic cardiomyopathy (6,7). On the other hand, pneumoconiosis can lead to chronic pulmonary heart disease due to its association with emphysema and pulmonary fibrosis (8). However, the alterations in the heart structure of patients with silicosis combined with diabetes mellitus remain poorly understood.

Computed tomography (CT)-measured cardiac and lung parameters have been used in patients with cardiopulmonary diseases such as pulmonary embolic disease (9), emphysema (10), and left ventricular enlargement (11). CT-measured cardiac parameters can reveal changes in cardiac structure even in non-contrast images (12) and have been shown to be related to prognosis in some cardiopulmonary diseases (13).

To our knowledge, there has been no prior research examining whether concurrent diabetes mellitus impacts cardiopulmonary structures and lung function in patients with silicosis. We posit that comorbid diabetes may affect cardiopulmonary structures and lung function in these patients. Therefore, in this study, we aimed to evaluate the differences in cardiopulmonary structural changes between silicosis patients with diabetes mellitus (SCD group) and those without diabetes mellitus (SWD group) on the basis of CT parameters and to investigate the factors influencing lung function. We present this article in accordance with the STROBE reporting checklist (available at <https://qims.amegroups.com/article/view/10.21037/qims-24-1748/rc>).

## Methods

### *Recruitment of patients and controls*

This was a retrospective study for which approval from

the Ethics Committee of the Fourth Hospital of West China, Sichuan University was obtained (No. HXS-EC-2022096). The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). Owing to the retrospective nature of the study, the requirement for individual patient consent was waived. Patients diagnosed with silicosis, staged according to a history of silica dust exposure per the China National Diagnostic Criteria for Pneumoconiosis (GBZ-2015) (14), which aligns with the International Labor Organization Classification of Pneumoconiosis (15), were included in the study between 1 January 2012 and 31 May 2024. The diagnosis of diabetes mellitus was made by an endocrinologist on the basis of the patient's blood test results. The collected data included demographic information, details of dust exposure history, chest CT images, pulmonary function test (PFT) results, and hematological results. The exclusion criteria encompassed patients with biopsy-confirmed tuberculosis as well as those diagnosed with tuberculosis based on clinical symptoms and laboratory findings.

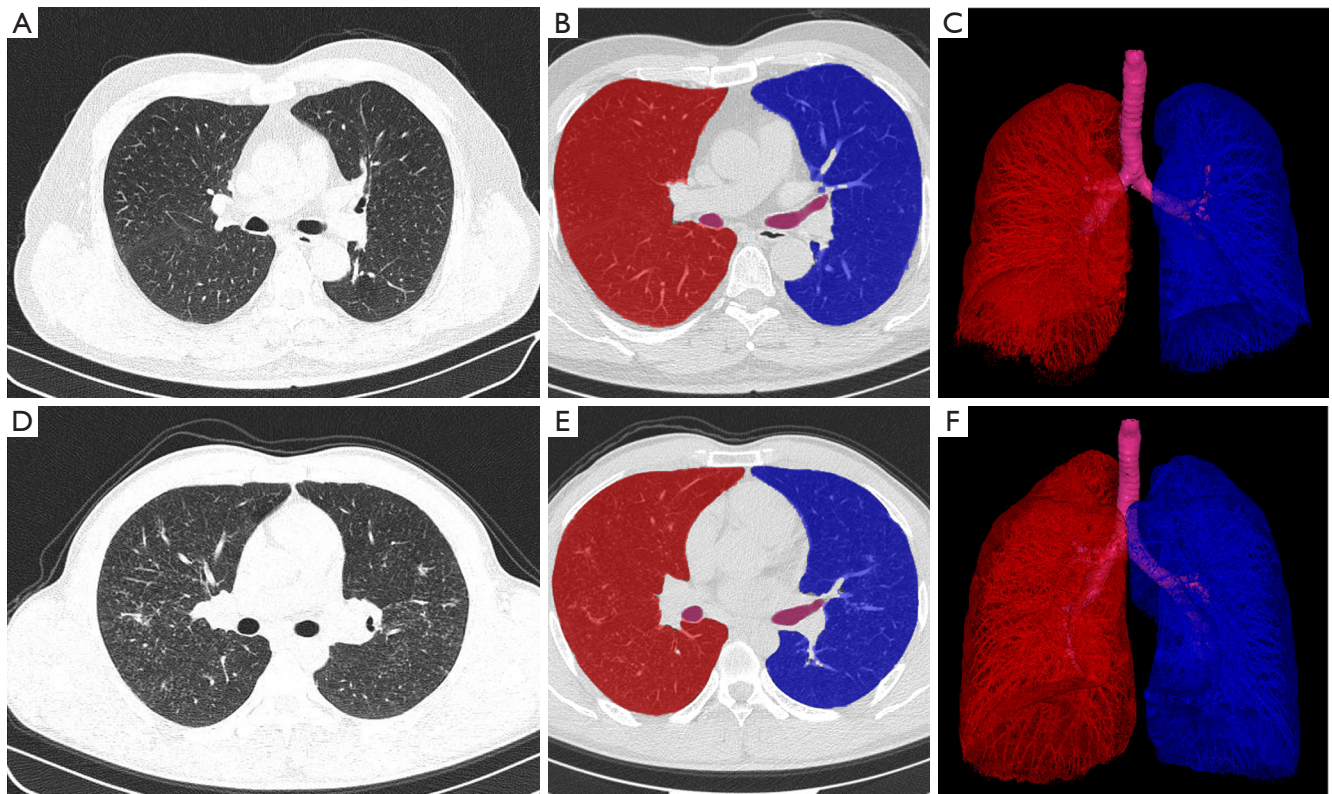
### *The CT protocols*

In this study, CT scans were conducted with a 16-channel CT scanner (Siemens Healthcare, Erlangen, Germany) and a 256-channel CT scanner (GE Healthcare, Chicago, IL, USA). Patients were placed in the supine position with their arms elevated above their heads, and scans proceeded from the apex to the base of the lungs during end-inspiratory breath-holding. Standard radiation protection measures (16) were employed throughout the scanning process to minimize the exposure of patients and staff to ionizing radiation. The non-contrast CT chest parameters for the CT scans included a tube voltage set between 120 and 130 kVp, a tube current ranging from 110 to 300 mAs, a pitch of 0.992–1.2, an image matrix configured at 512×512, and a reconstructed slice thickness of 1.5 mm.

### *The CT-measured parameters*

Three-dimensional (3D) volume and mass measurements of all the CT images were automatically calculated on high-resolution displays with an image archiving and communication system provided by CREALIFE Technology, Inc. (Beijing, China) (17). The lung parameters derived from the CT measurements included total lung volume (TLV) and total lung mass (TLM) (*Figure 1*).

The acquired image datasets were transferred to



**Figure 1** Diagram of pulmonary parameter measurements. (A,D) Non-enhanced axial chest CT images; (B,E) rendered images; (C,F) volume-rendered 3D images. (A-C) are from a 37-year-old male patient with stage 1 silicosis and diabetes, showing lower lung volume and lung mass (4,320 mL, 980 g). (D-F) are from a 29-year-old male patient with stage 1 silicosis but without diabetes, showing higher lung volume and lung mass (5,310 mL, 1,168 g). CT, computed tomography; 3D, three-dimensional.

a dedicated reprocessing workstation (CREALIFE Technology) where they were processed via volume rendering and curved planar reconstruction methods. This retrospective analysis was carried out by two radiologists employing a double-blinded methodology, and the outcomes were averaged for accuracy.

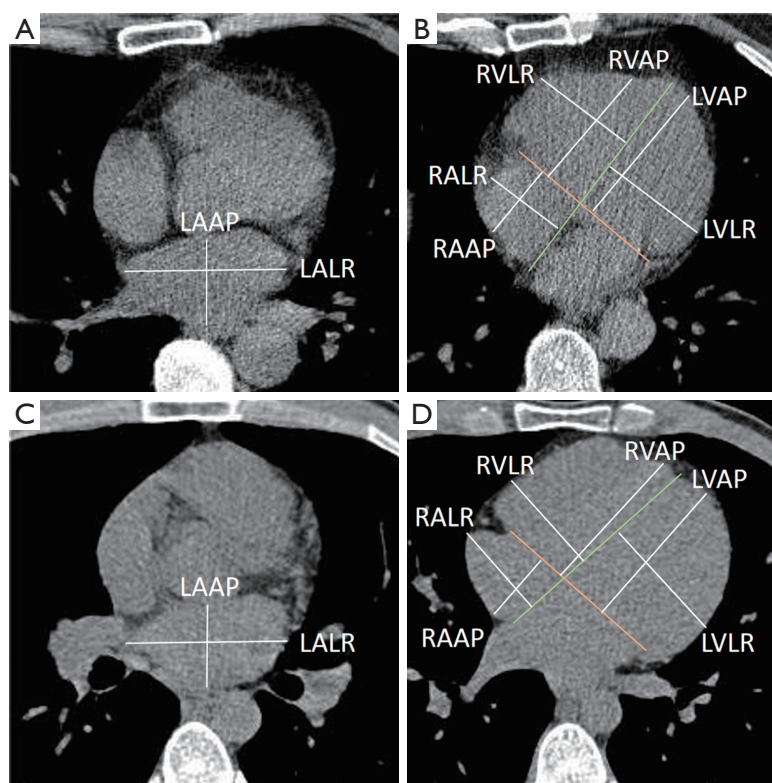
Measurements of the cardiac parameters via CT were based on methodologies suggested by Guo *et al.* (9), in which specific measurement tools were utilized. The cardiac parameters measured from CT imaging are detailed as follows (Figure 2): longest diameter from left to right of the left atrium (LALR), longest diameter from left to right of the left ventricle (LVLR), longest diameter from left to right of the right atrium (RALR), longest diameter from left to right of the right ventricle (RVLR), longest anteroposterior diameter of the left atrium (LAAP), longest anteroposterior diameter of the left ventricle (LVAP), longest anteroposterior diameter of the right atrium

(RAAP), and longest anteroposterior diameter of the right ventricle (RVAP).

### PFTs

PFTs were conducted in strict adherence to the American Thoracic Society/European Respiratory Society (ATS/ERS) guidelines (18) and were administered by specialists within the Pulmonary Function Department of the Fourth Hospital of West China, Sichuan University. These tests utilized a state-of-the-art clinical spirometer (MasterScreen PFT, Jaeger; Vyaire Medical, Höchberg, Germany) for accurate measurement. Relevant lung function metrics were retrieved from patients' medical records. The recorded lung function parameters included forced vital capacity (FVC), the ratio of FEV1/FVC, maximum vital capacity (VC MAX), residual volume (RV), total lung capacity (TLC), and the diffusing capacity of





**Figure 2** Diagram of cardiac parameter measurements. (A,C) Measurement of left atrial diameter; (B,D) measurement of left ventricle, right atrium, and right ventricle diameters. (A,B) The cardiac computed tomography parameters of a 37-year-old male patient with stage 1 silicosis and diabetes. (C,D) The cardiac CT parameters of a 29-year-old male patient with stage 1 silicosis but without diabetes. LAAP, longest anteroposterior diameter of the left atrium; LALR, the longest diameters from left to right of the left atrium; LVLR, the longest diameters from left to right of the left ventricle; RALR, the longest diameters from left to right of the right atrium; RVLR, the longest diameters from left to right of the right ventricle; LVAP, longest anteroposterior diameter of the left ventricle; RAAP, longest anteroposterior diameter of the right atrium; RVAP, and longest anteroposterior diameter of the right ventricle; CT, computed tomography.

the lungs for carbon monoxide (DLCO).

### Statistical analysis

All the statistical analyses were executed with the software SPSS 26.0 (IBM Corp., Armonk, NY, USA) and GraphPad Prism 9.0 (GraphPad Software, San Diego, CA, USA). Continuous variables were summarized as the means  $\pm$  standard deviations, whereas categorical variables were represented by counts (n) and percentages (%). Paired *t*-tests were applied for comparisons involving continuous variables, and chi-square tests were utilized for those involving categorical variables. To assess the relationships between CT-measured parameters and pulmonary function, Pearson correlation analysis was conducted. Linear regression analysis was employed to identify the

determinants of lung function impairment in the SCD group.

## Results

### Demographic characteristics

The study comprised two cohorts: the SCD group (n=30) and the comparably matched SWD group (n=30), with both groups consisting solely of male participants. Both cohorts were diagnosed by the Occupational Disease Evaluation Panel and confirmed by endocrinology specialists. No significant differences were observed between the groups regarding age, silicosis stage, smoking history, or duration of dust exposure, with all *P* values  $>0.05$ . Notably, the SCD group had a significantly higher body mass index (BMI)

**Table 1** Baseline characteristic of the study population according to whether silicosis is comorbid with diabetes mellitus

Variables	SWD group (n=30)	SCD group (n=30)	P value
Silicosis stage			>0.99
Stage I	12 (40.0)	12 (40.0)	
Stage II	9 (30.0)	9 (30.0)	
Stage III	9 (30.0)	9 (30.0)	
Age (years)	51.87±12.07	55.80±10.41	0.222
Smoking			0.754
Yes	23 (76.7)	24 (80.0)	
No	7 (23.3)	6 (20.0)	
BMI (kg/m <sup>2</sup> )	24.07±3.03	26.59±2.99	<0.001*
Years of dust exposure (years)	10.62±7.07	9.29±5.05	0.253

Data are presented as n (%) or mean ± SD. \*, P<0.05. SWD, silicosis without diabetes mellitus; SCD, silicosis combined with diabetes mellitus; BMI, body mass index; SD, standard deviation.

**Table 2** The differences of CT measured cardiac and pulmonary parameters between SWD and SCD groups

Variables	SWD group (n=30)	SCD group (n=30)	P value
TLV (mL)	4,729.63±1,192.60	4,502.59±685.35	0.399
TLM (g)	1,029.40±177.55	959.21±120.84	0.048*
LAAP (mm)	30.00±6.41	33.01±6.20	0.101
LALR (mm)	65.27±9.48	64.01±8.12	0.557
LVAP (mm)	48.92±6.25	50.45±5.37	0.342
LVLRL (mm)	65.60±10.35	70.53±6.29	0.030*
RVAP (mm)	48.84±26.80	45.82±7.49	0.521
RVLRL (mm)	55.52±8.86	58.60±4.91	0.109
RAAP (mm)	40.09±7.74	40.30±5.84	0.904
RALRL (mm)	43.35±8.29	41.48±7.32	0.308

The data are presented as mean ± standard deviation. \*, P<0.05. CT, computed tomography; SWD, silicosis without diabetes mellitus; SCD, silicosis combined with diabetes mellitus; TLV, the total lung volume; TLM, total lung mass; LAAP, anteroposterior diameters of the left atrium; LALR, the longest diameters from left to right of the left atrium; LVAP, anteroposterior diameters of the left ventricle; RVAP, anteroposterior diameters of the right ventricle; RVLRL, longest diameters from left to right of the right ventricle; LVLRL, the longest diameters from left to right of the left ventricle; RAAP, anteroposterior diameters of the right atrium; RALRL, longest diameters from left to right of the right atrium.

than did the SWD group (P<0.001) (Table 1).

### *Differences in the hematology results, CT-measured parameters, and pulmonary function results in the SCD versus SWD group*

The comparative analysis of quantitative CT lung and cardiac parameters revealed that the TLM values in the SCD group were significantly lower than those in the SWD group (P=0.048). Furthermore, the SCD group showed significantly higher values for LVLRL (70.53±6.29 mm) compared to the SWD group, which had a corresponding value of 65.60±10.35 mm (P=0.030). However, in the comparisons of the SCD and SWD groups, there was no significant difference in TLV, LAAP, LALR, LVAP, RVAP, RVLRL, RAAP, or RALRL (all P>0.05) (Table 2).

The SCD group showed significantly higher values for blood glucose and triglycerides compared to the SWD group (all P<0.05). Additionally, there were no significant differences between the SCD and SWD groups in other hematological indices such as total protein (TP) and albumin (ALB) (all P>0.05) (Table 3).

In the comparisons of lung function, the SCD group had a significantly lower FEV1/FVC (78.69%±10.96%) than did the SWD group (89.31%±7.41%, P<0.001). There were no significant differences in FVC, VC MAX, RV, TLC, or DLCO between the SCD and SWD groups (all P>0.05) (Table 4).

### *Analysis of correlations between pulmonary function and CT-measured parameters in the SCD group*

FEV1/FVC exhibited significant correlations with TLM, LALR, LVLRL, and RALRL, with correlation coefficients (r) of 0.51, 0.47, 0.40, and 0.44, respectively (all P<0.05) (Figure 3).

### *Univariate and multivariate regression analyses of the SCD group*

The univariate linear regression analysis for the SCD group revealed that silicosis stage, age, TLM, LALR, LVLRL, and RALRL were factors affecting FEV1/FVC, with all P values less than 0.05. Based on the results of univariate linear regression and previous studies (19–21), silicosis stage, age, smoking history, BMI, TLM, LALR, LVLRL, RALRL, and laboratory test results (including blood glucose, calcium, phosphorus, and iron levels) were included in the multivariate linear regression analysis. This analysis

**Table 3** Laboratory measurements according to whether silicosis is combined with diabetes mellitus

Variables	SWD group (n=30)	SCD group (n=30)	P value
TP (g/L)	66.40±6.59	67.37±5.11	0.589
ALB (g/L)	39.86±3.64	40.36±3.99	0.645
GLB (g/L)	26.53±4.74	27.02±3.07	0.641
ALT (U/L)	34.00±36.91	32.67±22.53	0.865
AST (U/L)	31.37±31.53	24.43±11.69	0.268
TBIL (μmol/L)	17.67±10.60	16.73±4.26	0.635
DBIL (μmol/L)	4.81±2.84	5.16±1.81	0.573
IBIL (μmol/L)	12.88±8.79	11.57±3.68	0.418
ALP (U/L)	68.83±25.37	70.60±21.77	0.760
GGT (U/L)	38.77±56.47	39.10±23.96	0.976
BUN (mmol/L)	5.21±1.48	5.12±1.70	0.848
CREA (μmol/L)	74.67±11.78	74.18±16.40	0.884
UA (μmol/L)	343.03±102.68	429.30±452.36	0.913
GLU (mmol/L)	5.31±0.65	8.10±2.99	<0.001*
CHOL (mmol/L)	4.68±0.91	4.80±1.05	0.640
TG (mmol/L)	1.19±0.53	1.89±0.84	0.001*
HDL-C (mmol/L)	1.33±0.40	1.16±0.28	0.092
LDL-C (mmol/L)	3.12±0.84	3.12±0.84	0.495
K (mmol/L)	4.05±0.43	3.96±0.28	0.322
Na (mmol/L)	138.80±2.27	139.33±2.20	0.388
Cl (mmol/L)	105.31±3.54	105.78±4.02	0.624
Ca (mmol/L)	2.28±0.12	2.33±0.13	0.102
P (mmol/L)	1.09±0.21	1.09±0.15	0.967
Mg (mmol/L)	0.83±0.07	0.79±0.08	0.134
Fe (μmol/L)	16.7±5.38	16.11±7.53	0.700
CO <sub>2</sub> CP (mmol/L)	24.03±2.92	23.33±3.72	0.449

The data are presented as mean ± standard deviation. \*, P<0.05. SWD, silicosis without diabetes mellitus; SCD, silicosis combined with diabetes mellitus; TP, total protein; ALB, albumin; GLB, globulin; ALT, alanine aminotransferase; AST, aspartate transaminase; TBIL, total bilirubin; DBIL, direct bilirubin; IBIL, indirect bilirubin; ALP, alkaline phosphatase; GGT, γ-glutamyl transpeptidase; BUN, blood urea nitrogen; CREA, creatinine; UA, uric acid; GLU, glucose; CHOL, total cholesterol; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; K, potassium; Na, sodium; Cl, chlorine; Ca, calcium; P, phosphorus; Mg, magnesium; Fe, iron; CO<sub>2</sub>CP, carbon-dioxide combining power.

**Table 4** The differences of pulmonary functional test results between SWD and SCD groups

Variables	SWD group (n=30)	SCD group (n=30)	P value
FVC (L)	2.54±0.98	2.67±0.93	0.616
FEV1/FVC (%)	89.31±7.41	78.69±10.96	<0.001*
VC MAX (L)	2.66±0.99	2.79±0.88	0.594
RV (L)	2.64±0.80	2.86±0.93	0.328
TLC (L)	5.33±1.22	5.71±0.87	0.201
DLCO (mmol/min/kPa)	6.29±2.75	5.64±2.02	0.311

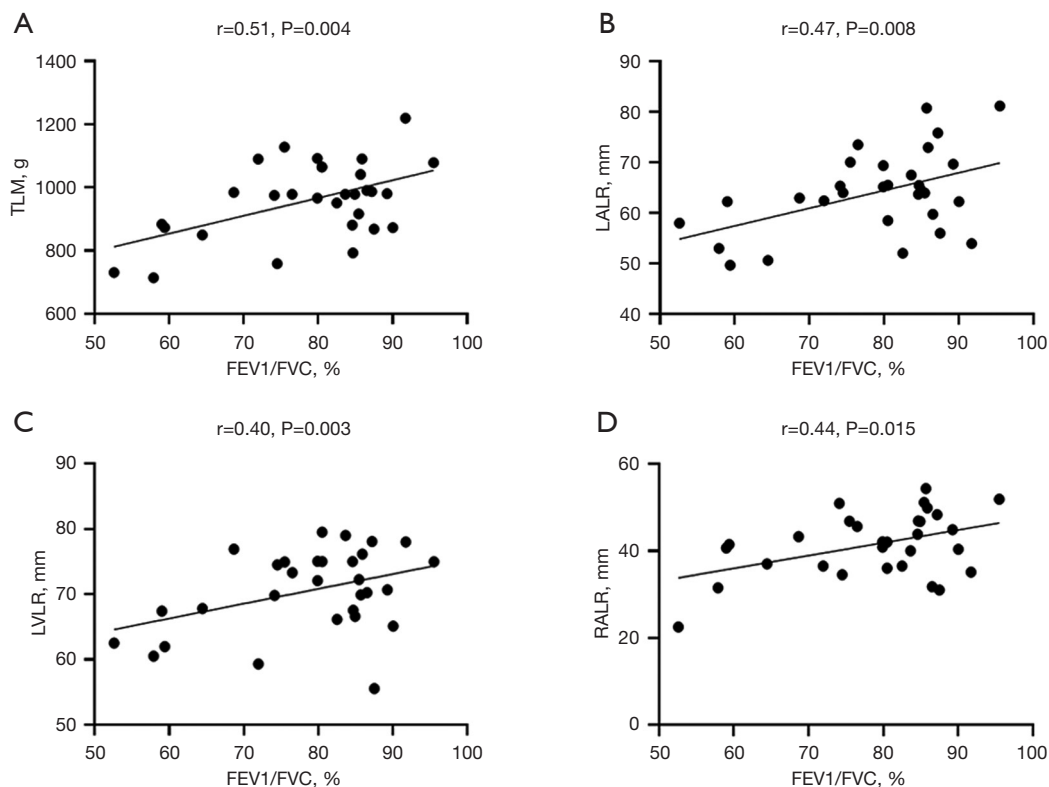
The data are presented as mean ± standard deviation. \*, P<0.05. SWD, silicosis without diabetes mellitus; SCD, silicosis combined with diabetes mellitus; FVC, forced vital capacity; FEV1/FVC, ratio of FEV1 to FVC; VC MAX, maximum vital capacity; RV, residual volume; TLC, total lung volume; DLCO, diffusing capacity of the lungs for carbon monoxide.

indicated that BMI and LVLR were independent determinants of FEV1/FVC (β coefficients of −0.506 and 0.317, respectively, with respective P values of 0.002 and 0.049) (*Table 5*).

## Discussion

The study findings showed that the SCD cohort manifested an enlarged LVLR alongside suboptimal pulmonary function indicators, such as a reduced FEV1/FVC ratio. This observation implies a probable direct reciprocity between cardiac morphology and pulmonary health, indicating mutual influence. The correlation identified between the SCD cohort's FEV1/FVC and parameters quantified by CT underscores the importance of imaging methods in evaluating respiratory system disorders. A single thoracic CT scan not only provides structural metrics but also offers predictive insights into FEV1/FVC, potentially enhancing the precision of disease diagnosis and monitoring its progression. This insight provides a novel perspective for the exploration of SCD. Furthermore, BMI and LVLR have been validated as independent determinants of pulmonary function in the SCD patient population. This might steer targeted intervention strategies for specific risk factors in clinical practice.

CT-measured lung parameters serve as dependable indices for assessing lung pathology, encompassing conditions such



**Figure 3** The correlation analysis of FEV1/FVC with cardiac and pulmonary parameters measured on CT. TLM, total lung mass; FEV1/FVC, forced expiratory volume in one second to forced vital capacity; LALR, the longest diameters from left to right of the left atrium; LVLr, the longest diameters from left to right of the left ventricle; RALR, the longest diameters from left to right of the right atrium; CT, computed tomography.

as coronavirus disease 2019 (COVID-19) (22) and chronic obstructive pulmonary disease (COPD) (23), with CT-derived lung masses demonstrating strong concordance with *in vivo* lung masses (24). Our investigation revealed a reduction in TLM and the FEV1/FVC ratio in the SCD cohort compared with those in the SWD cohort, diverging slightly from previous findings. Prior research has indicated an increase in FEV1/FVC among patients with T2DM relative to healthy controls (19); conversely, our data show a decline in FEV1/FVC within the SCD cohort vis-à-vis the SWD cohort. This discrepancy might be attributable to our focus on a population with preexisting silicosis, where the co-occurrence of T2DM potentially exacerbates the pulmonary impact of silicosis. Historically, studies have reported a decrease in lung mass among individuals with COPD, with a trend toward decreasing lung mass as COPD severity increases (23), whereas emphysema is associated with an increase in lung mass (10). Patients with pneumoconiosis are predisposed to concurrent COPD (25),

which may explain the reduced lung mass observed in the SCD cohort relative to the SWD cohort in our study.

Pulmonary dysfunction in diabetic patients predominantly involves four pivotal mechanisms: non-enzymatic glycation of lung collagen and elastin by advanced glycation end products, leading to diminished lung elasticity; thickening of the alveolar epithelial basement membrane and alterations in the microvasculature, reducing alveolar capillary blood volume and diffusion capacity; autonomic neuropathy impacting the phrenic nerve, causing decreased muscle tone and impaired diaphragmatic control; and hyperglycemia increasing the glucose concentration in airway surface liquid, fostering bacterial colonization, increasing the frequency of acute exacerbations akin to those of COPD, and worsening post-intervention treatment outcomes (19,26). All these pathways may synergize with other lung diseases, such as silicosis in our study, potentially leading to adverse consequences in patients concurrently afflicted by both conditions.

**Table 5** Multivariate regression analysis of factors affecting FEV1/FVC in SCD patients

Variables	Estimate	Standard error	t value	P value	95% confidence interval		Collinearity statistics	
					Lower	Upper	Tolerance	Variance inflation factor
Multiple regression analysis for FEV1/FVC of subjects with complete data (n=30)								
Age (years old)	-0.133	0.155	-0.856	0.404	-0.461	0.195	0.564	1.774
Silicosis stage	-3.221	2.011	-1.602	0.128	-7.463	1.022	0.511	1.956
BMI	-1.798	0.534	-3.367	0.004*	-2.924	-0.671	0.580	1.724
Smoking	7.350	3.857	1.906	0.074	-0.787	15.486	0.599	1.668
TLM	0.005	0.002	2.075	0.053	0.000	0.010	0.530	1.885
LALR	0.246	0.249	0.986	0.338	-0.280	0.771	0.361	2.772
LVLR	0.670	0.271	2.471	0.024*	0.098	1.243	0.508	1.970
RALR	0.378	0.300	1.263	0.224	-0.254	1.011	0.307	3.260
GLU	0.572	0.477	1.198	0.247	-0.435	1.578	0.726	1.377
Ca	15.134	12.200	1.240	0.232	-10.606	40.874	0.626	1.598
P	8.983	12.435	0.722	0.480	-17.252	35.218	0.422	2.371
Fe	0.272	0.228	1.192	0.249	-0.209	0.754	0.499	2.002

\*,  $P < 0.05$ . BMI, body mass index; FEV1/FVC, forced expiratory volume in one second to forced vital capacity ratio; SCD, silicosis combined with diabetes mellitus; TLM, total lung mass; LALR, the longest diameters from left to right of the left atrium; LVLR, the longest diameters from left to right of the left ventricle; RALR, the longest diameters from left to right of the right atrium; GLU, glucose; Ca, calcium; P, phosphorus; Fe, iron.

We hypothesize that in SCD patients, impaired diaphragmatic control due to phrenic nerve involvement and increased glucose content in airway surface liquid may contribute to a decrease in FEV1/FVC, favoring an obstructive phenotype.

Diabetic cardiomyopathy primarily affects the left heart (27). Cosyns *et al.* (28) and Yu *et al.* (29) reported that diabetic patients had a larger left ventricle (LV) diameter than control patients did. Gimenes *et al.* (30) and Markus *et al.* (31) found that diabetic rats had larger LV and left atrium (LA) diameters than control rats did. Zhou *et al.* (32) found that the LAAP in the diabetic rabbit group was larger than that in the control group. This study revealed that T2DM increased the LVLR in silicosis patients, which is consistent with the findings of previous studies. However, Jørgensen *et al.* (33) and Li *et al.* (34) reported that the diabetic group had a lower LV diameter than the control group did. The reason may be that diabetic cardiomyopathy is “two-faced” according to Seferović’s study. Seferović argued that diabetic cardiomyopathy presented as either dilated cardiomyopathy or restrictive cardiomyopathy (35). Our study revealed that SCD patients exhibit a dilated

cardiomyopathy phenotype.

Our study demonstrated that, within the SCD cohort, BMI and LVLR serve as independent determinants of pulmonary function, highlighting the complexity of lung function decline. An elevated BMI, indicative of increased abdominal and thoracic adiposity, exerts mechanical pressure on the diaphragm, restricting lung expansion and diminishing the lung volume. In combination with inflammatory processes and oxidative stress, this exacerbates pulmonary injury (20,21,36). An augmented LVLR signifies left ventricular dysfunction, compromising cardiac output and elevating pulmonary circulation pressure, thus hindering effective gas exchange (37,38). Overall, obesity augments cardiac workload, impairs left ventricular integrity, and engenders a vicious cycle that further deteriorates pulmonary function. Against the backdrop of silicosis, these factors intensify the deterioration of lung function. Consequently, patients with silicosis require vigilant weight and cardiovascular health management, necessitating a comprehensive intervention approach to optimize prognosis.

Our findings revealed that in the SCD cohort, the



FEV1/FVC ratios correlated moderately with TLM, LALR, LVLr, and RALR, diverging from prior studies on human immunodeficiency virus (HIV)-infected adolescents, where the lung mass and volume were moderately positively correlated with FEV1 but not with FEV1/FVC (39). This disparity could stem from differing age profiles and environmental exposures. Diabetes and silicosis in tandem might create unique cardiac-pulmonary interdependencies not observed in HIV-infected youth, as diabetes-induced cardiac alterations and silicosis-related lung fibrosis could synergistically affect the FEV1/FVC ratio (19,28,40). HIV-related lung dysfunction likely stems from viral lung injury rather than from cardiac factors. Remarkably, our data suggest that CT scans can be used to partially quantify lung function impairment in SCD patients, suggesting a potential alternative to PFTs.

There have been no previous studies on the impact of diabetes on cardiopulmonary structure and lung function. Our study contrasts the SCD and SWD groups, showing higher BMI, glucose, TG, and LVLr in the SCD group, alongside reduced lung mass and FEV1/FVC. We found a significant correlation between the FEV1/FVC and CT-obtained lung parameters of SCD patients, highlighting the link between lung function and structural changes. Importantly, BMI and LVLr were confirmed to be independent factors affecting SCD patients' lung function, indicating that metabolic health and cardiac structure play critical roles in pulmonary health. These findings enhance the understanding of the effects of systemic disease on lung function and guide health management in SCD cases; it also provides future directions for research on metabolic-cardio-respiratory disease interconnections.

Previous studies have shown that smoking increases the risk to the cardiovascular system and damages lung function (41). In our study, although there was no difference in smoking prevalence between the SCD and SWD groups, and smoking was not identified as a significant factor affecting lung function in multivariate analysis, this may have been due to the relatively small sample size. Future research with larger sample sizes should further investigate the relationship between smoking and the comorbidity of silicosis and diabetes mellitus. Although pulmonary tuberculosis has been excluded in this study, patients with silicosis and diabetes mellitus are prone to coexisting tuberculosis (42), which may affect cardio-pulmonary structure and lung function. This issue will be further explored in future studies.

We acknowledge that this study has several limitations. First, this was a single-center, cross-sectional study with a small sample size, which may have introduced some selection bias. Additionally, although the study continuously recruited silicosis patients, the participants were predominantly male. We hope that future multicenter studies will include a larger number of female patients with silicosis. Lastly, the use of different CT scanners could introduce minor measurement biases; in future studies, we will use the same model of scanner to improve consistency.

## Conclusions

Diabetes amplifies lung function decline in silicosis patients, with BMI and LVLr as key independent factors. Enhanced personalized management, focusing on weight and cardiac health, is crucial for these patients to mitigate lung function deterioration and enhance quality of life. This study deepens the understanding of SCD health profiles, offering valuable insights for future research and clinical strategies against the dual threat of diabetes and silicosis.

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