Case Report

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"Porcelain In The Lung": Apropos Of A Young Woman With Complicated Chronic Silicosis.

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ABSTRACT

Silicosis is a lung disease caused by the inhalation of crystalline silica particles included in the group of pneumoconiosis or occupational lung diseases, which, in turn, are included among the diffuse interstitial lung diseases. The risk of disease occurrence is related to the amount of silica inhaled throughout the working life and, once established, no effective treatment is available. Respirable dust control and early diagnosis are the most effective measures against this condition. The sources of occupational exposure to silica inhalation are very numerous, including the particular ones derived from porcelain. We present the case of a young woman who developed chronic conglomerate variety pulmonary silicosis after working in the ceramics industry for more than 20 years, correlating chest imaging findings with pulmonary function tests.

Categories: Internal Medicine, Pulmonology, Occupational Health.

Keywords : Pneumoconioses, Silicosis, Silica, Occupational lung disease, Interstitial lung disease.

INTRODUCTION

Silica, composed of silicon and oxygen as silicon dioxide (SiO2), exist in two specific and distinct forms: crystalline (highly toxic) and amorphous or noncrystalline (lower toxicity). Silica dust is the main constituent of sand, hence exposure is most prevalent among gold miners, sandstone and granite cutters, foundry workers, miners and potters [1]. Quartz is the most common form of crystalline silica, being the second most common mineral in the earth's crust, available in almost all types of natural rock. Cristobalite and tridymite are two other forms of crystalline silica which, although not abundant in nature, are also found in some volcanic rocks [2].

According to the International Labor Office (ILO), silicosis is characterized by progressive, sclerosant fibrotic lung changes, presented as specific radiographic signs, with or without respiratory functional impairment [1][3]. Diagnosis requires a work history of exposure to crystalline silica or free silica (SiO2) like quartz, sand or granite (60% SiO2) alongside characteristic clinical, functional and radiological findings while ruling out other diffuse interstitial lung diseases, taking into consideration a variable latency period depending on the magnitude of exposure. Notably, silicosis has been linked to some autoimmune disorders such as systemic sclerosis, as observed by Bramwell in 1914 in stone polishers and later by Erasmus among South African gold miners [4].

High-risk sources of exposure include the extraction, processing and use of quartz as a raw material or component in other industries such as glass, sanitary ceramics and refractory materials, industrial activities that are certainly frequent in our environment [5]. The intensity of exposure is quantified as [5][6]:

Accumulated silica dose = fraction of respirable dust × percentage of free silica in mg/m3 × number of years of exposure. The fraction of respirable dust refers to the particles that can reach the alveoli, specifically 30% of particles

measuring 5 microns and 100% of those measuring 1 micron. Individual susceptibility is related to the deposition and persistence of inhaled dust in the body, due to the loss of the efficiency of the defense and mucociliary clearance mechanisms [6]. This may be influenced either by genetic factors or by other factors: smoking and/or the presence of respiratory diseases such as chronic obstructive pulmonary disease (COPD), which explains cases of particular susceptibility at low doses of exposure and vice versa [3-6]. In general, it is recommended that exposure should not exceed 50 µg/m3 during an 8-hour working day. The environmental limit value (TLV) has been defined as 0.025 mg/m3 for crystalline silica (quartz and cristobalite): if these values were not exceeded, a majority of workers exposed throughout their working life would not suffer adverse health effects. Particles smaller than 5 micrometers are likely to reach the alveoli, while those larger than 10 micrometers are generally trapped in the upper airways due to impaction.

Patho-physiologically, crystalline silica disrupts the phagolysosome formation in alveolar macrophages through the macrophage receptor with a collagen structure (MARCO). This subsequently leads to formation of reactive oxygen species (ROS) and reactive nitrogen species (RNS), activating multiple biological reactions that result in inflammation and direct damage to lung tissue [1][7]. Additionally, the lysosomal content, when in contact with the cytoplasm, triggers the inflammasome (a multiprotein complex that acts as a sensor and mediator of inflammation development through type PPR receptors) [7], leading to the release of the proinflammatory cytokine IL-1β, and furthermore a Th1 type inflammatory cascade, generating a granulomatous reaction around the silica deposits, a typical finding of silicotic nodules. Additionally, alveolar macrophages tend to dedifferentiate into fibroblasts through molecular pathways mediated by caspase-1, which stimulates the production of fibroblast growth factor (FGF) contributing to fibrosis [7][8].

Pulmonary silicosis presents in various forms, classified by exposure duration, radiological findings and symptoms reported by the patient and the pulmonary function tests (PFTs). According to the ILO (Internacional Labour Office), risk factors for disease progression include high levels of exposure, previous history of pulmonary tuberculosis, active smoking, and the severity of lesions on a chest X-ray (parenchymal opacities and pleural involvement) at the time of diagnosis [8].

We present the case of a young woman with a diagnosis of complicated chronic pulmonary silicosis - conglomerate variety - (known cause of progressive pulmonary fibrosis) [9] associated with the ceramics industry and its correlation with the findings in pulmonary function tests, in which an isolated restrictive pattern does not necessarily predominate and can be mixed [10] probably depending on the degree of peribronchovascular involvement, as in the case presented here, where a moderate obstructive component was also confirmed both by pre- and post-bronchodilator spirometry and lung volumes by plethysmography.

CASE PRESENTATION

A 49-year-old woman with a 26-year history of exposure to porcelain waste in the ceramic industry began experiencing lower respiratory symptoms eight years ago. These included dyspnea on exertion (DOE) and radiological evidence of conglomerate silicosis (pseudomasses with bilateral perihilar distribution extending to the apex). She consulted for 1 week of increasing dyspnea and subjective fever, preceded by two days of diarrhea without mucus or blood.

Physical examination revealed wheezing and coarse rhonchi bilaterally, with oxygen saturation at 86% on room air. Initial lab results showed no leukocytosis, anemia, or elevated C-reactive protein (CRP), while arterial gases indicated mild hypoxemia (pO2: 93 mmHg, pCO2: 40 mmHg, FiO2 0.32, Pao2/FiO2: 290). The stool test was inflammatory, with a negative stool culture after 48 hours.

Chest X-ray (**Figure 1**) and CT (**Figure 2**) were obtained, which showed pseudomasses, consistent with conglomerate silicosis, with no evidence of concomitant pleural effusion.

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Figure 1

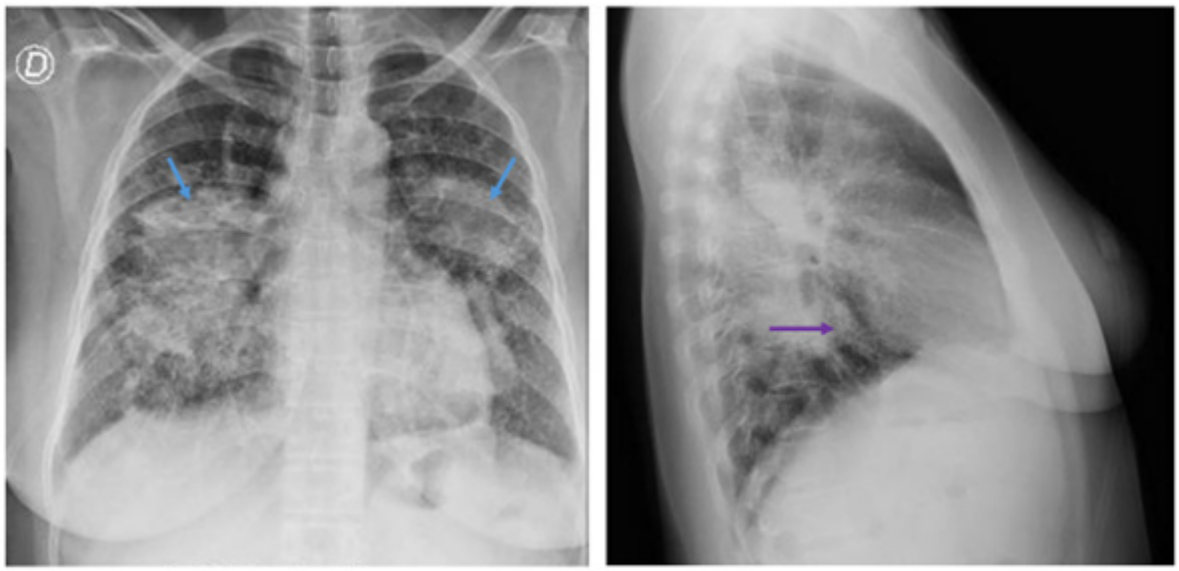


Figure 1. Multiple small nodules are observed in both lungs predominantly in lower segments of both upper lobes and middle lobes, with extensive heterogeneous opacities (>10mm) that have the appearance of "pseudomasses" (confluent silicotic nodules) of bilateral perihilar distribution (blue arrow). There is discrete right bronchial retraction in the lateral projection, with extensive retrocardiac reticulation (purple arrow).

Figure 2



Figure 2. Extensive opacities with the appearance of pseudomasses of perihilar distribution, symmetrical, with high density (95 HU, approximately) (black arrow) with perivascular mediastinal nodules, without evidence of adjacent calcified lymph nodes. Some nodules of perilymphatic distribution are observed, in addition to thickening of interlobular septa. It is considered a case of complicated pulmonary silicosis conglomerate variety (previously, progressive massive fibrosis).

The patient was initially treated with sultamicillin BID orally for 48 hours without clinical improvement, so it was escalated to cefepime 2 g BID IV, due to known risk factors for P. aeruginosa (microbiological isolation was not achieved from the induced sputum sample), oral corticosteroids (prednisolone 50 mg/day), and short-acting bronchodilator therapy, resulting in a complete clinical recovery within five days.

She had recent pulmonary function tests: pre- and post-bronchodilator spirometry with mixed pattern (FEV1/FVC: 0.71) with moderate obstructive pattern (FEV1 47% of theoretical; FVC 53% of theoretical) not reversible with bronchodilator, in addition to a moderately decreased diffusion of monoxide (DLCO), low VA and normal kCO and lung volumes by plethysmography with

Journal of Respiratory Medicine and Research

moderate simple restriction (**Figures 3 and 4**). She is currently under option for lung transplantation.

	Pre – Bronch				Post - Bronch			
	LIN	**Real**	**Teórico**	**% Teórico**	**Z Score**	**Real**	**% Teórico**	**% Cambio**
---SPIROMETRY---								
FVC (L)	1.95	1.31	2.47	53	-3.67	1.36	54	+3
FEV I (L)	1.53	0.94	1.97	47	-3.86	1.02	51	+8
FEV1 / FVC (%)	0.72	0.71	0.81	87	-1.78	0.75	91	+s
FEP 50% (L/Sec)	1.25	0.63	2.86	22	-2.28	1.00	35	+59
FEF 75% (L/Sec)	-0.07	0.31	1.02	30	-1.07	0.38	37	+21
FEF 25 - 75% (L/Sec)	l.35	0.63	2.33	27	-2.86	0.84	36	+33
PEF (L/min)		178.1				194.6		+9
FIF 50% (L/sec)	1.85	2.15	329	65	-1.31	2.22	67	-3
Expiratory ‘lime (sec)		7.10				6.82		-3
Back Extrap Vol (L)		0.02				0.02		-16
--LUNG VOLUMES-								
TLC (Pleth) (L)	2.62	2.30	3.69	62	-2.59			
SVC (L)	1.95	1.33	2.47	53	-3.61			
IC(L)		0.93	1.63	56				
FRC (SB) (L)		1.13						
RV (Pleth) (L)	0.55	0.85	1.31	64	-1.22			
RV / TLC (Pleth) (%)	0.24	0.37	0.35	105	+0.36			
TGV (L)	0.95	1.50	l.99	75	-0.95			
	Pre – Bronch				**Post - Bronch**			
	LIN	**Real**	**Teórico**	**% Teórico**	**Z Score**	**Real**	**% Teórico**	**% Cambio**
BHT (sec)		10.28						
---RESISTANCE---								
Raw (cmH2O / L/s)	1.15	3.43	1.86	184	+3.67			
Gaw (L/S/cmH2O)		0.29	1.03	28				
sRaw (cmH20*s)		6.61	<4.76					
sGaw (I / cmH20*s)	0.14	0.15	0.20	73	-1.48			
-- DIFFUSION---								
DLCO unc (ml/min/mmHg)	11.07	9.24	15.16	60	-3.23			
DLCO cor (ml/min/mmHg)	11.07		15.16	107				
DL / VA (ml/min/mmHg/L)		0.05	0.05					
IVC (L)		1.28						
VA (L)	2.65	1.85	3.26	56	-4.40			
BHT (sec)		10.28						

Journal of Respiratory Medicine and Research

Figure 3

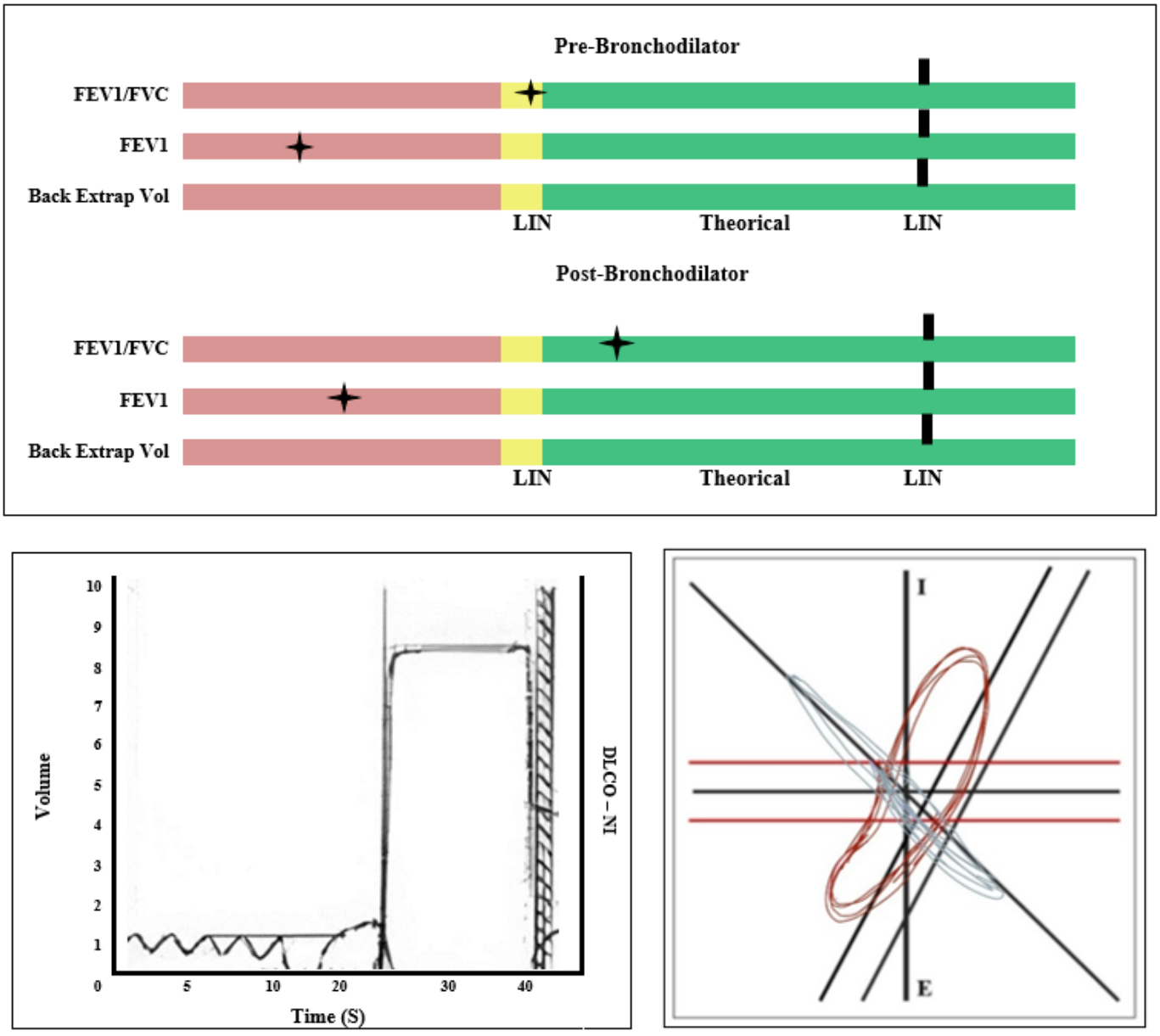
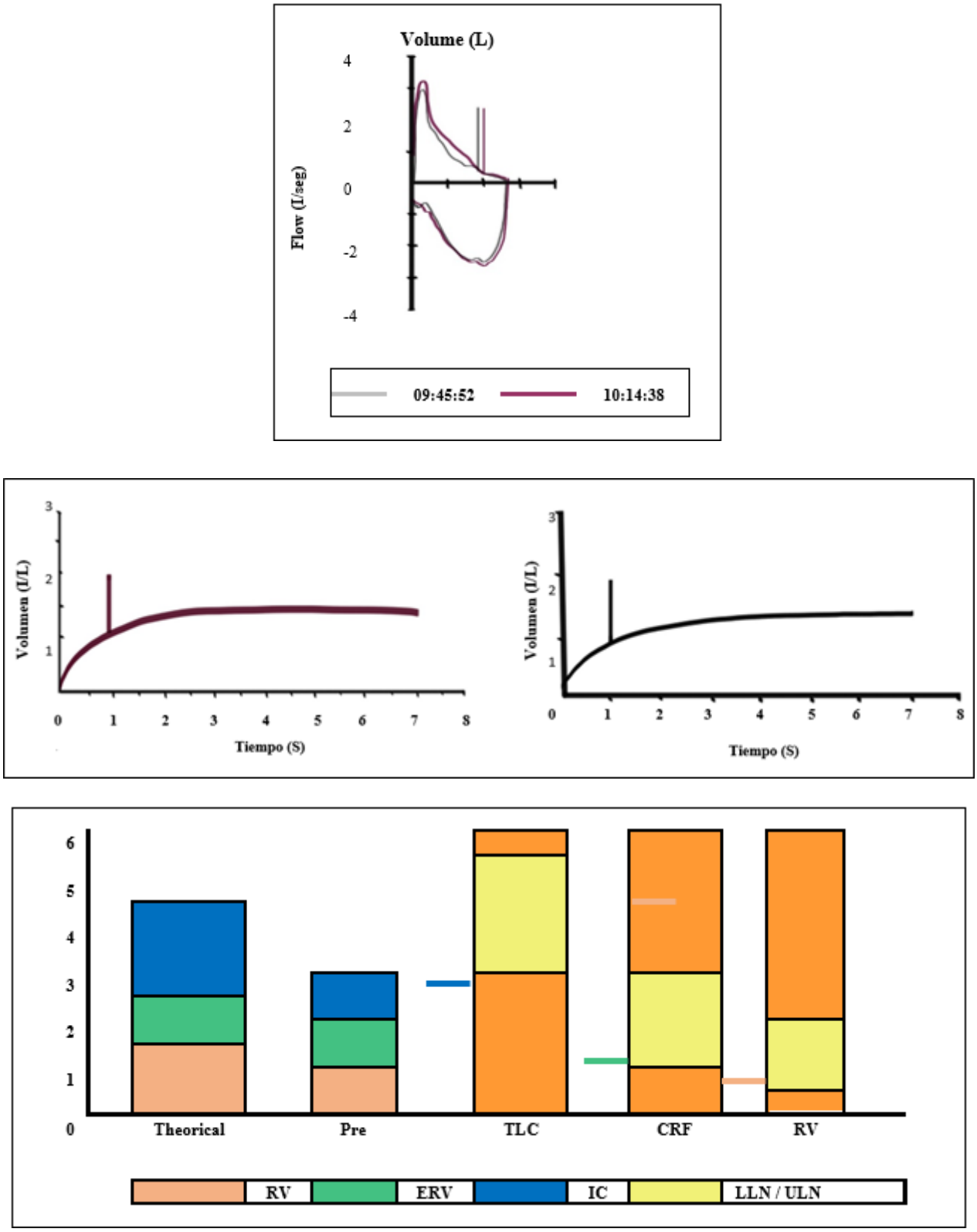


Figure 3. Spirometry pre and post bronchodilator with mixed pattern (FEV1/FVC: 0.71), with moderate obstructive pattern (FEV1 47% of the theoretical; FVC 53% of theoretical), non-reversible with bronchodilator. DLCO: Diffusion with moderate decrease, low VA and normal kCO. Nomenclature: FVC: Forced Vital Capacity, FEV1: Forced Expiratory Volume in 1 Second, FEF 50%: Forced Expiratory Flow at 50%, FEF 75%: Forced Expiratory Flow at 75%, FEF 25-75%: Forced Expiratory Flow between 25% and 75%, FIF50%: Flow to Forced Inspiratory Flow, FEV6: Forced Expiratory Volume in 6 Seconds, PEF: Peak Expiratory Flow, FIF 50%: Forced Inspiratory Flow at 50%, Back Extrap Vol: Back Extrapolated Volume, TLC (Pleth): Total Lung Capacity measured by Plethysmography, SVC: Slow Vital Capacity, IC: Inspiratory Capacity, FRC (SB): Functional Residual Capacity measured by Single Breath, RV (Pleth): Residual Volume measured by Plethysmography, RV: Residual Volume, TGV (L): Thoracic Gas Volume, BHT: Breath-Holding Time, Raw: Airway Resistance, Gaw: Airway Conductance, sRaw: Specific Airway Resistance, sGaw: Specific Airway Conductance, DLCO unc: Diffusing Capacity of the Lung for Carbon Monoxide, uncorrected, DLCO cor: Diffusing Capacity of the Lung for Carbon Monoxide, corrected, DL: Diffusing Capacity, IVC: Inspiratory Vital Capacity, VA: Alveolar Volume, BHT: Breath-Holding Time.

Journal of Respiratory Medicine and Research

Figure 4. Lung volumes by plethysmography with moderate simple restriction.



Journal of Respiratory Medicine and Research

DISCUSSION

Silicosis is an interstitial lung disease caused by the inhalation and deposits of crystalline silica dust, typically developing over more than 10 years. Clinical manifestations can range from asymptomatic to chronic severe respiratory failure in cases of accelerated silicosis (**Table 1**). Newly fractured and dry silica dust is the most harmful and can lead to acute silicosis with high risk of imminent ventilation failure [10][11]. Diagnosis relies on occupational history, symptoms and radiological findings, without the need of histopathological confirmation, as no effective treatment exists beyond removing patients from any source of exposure and ongoing monitoring to evaluate a potential lung transplant [11].

Table 1. According to the clinical, radiological and functional data, we can differentiate some forms of presentation of the disease that we classify as: chronic silicosis (simple, complicated and interstitial pulmonary fibrosis), accelerated silicosis and acute silicosis. Adapted from [12]

Clinical form	Time of exposure	Radiological findings	Symptoms	Pulmonary Function Tests
Acute Silicosis	< 5 years	Bilateral acinar pattern, with "cobblestone" or "crazy paving" pattern	Dyspnea	Generally restrictive alteration with decreased DLCO
Accelerated Silicosis	5 - 10 years	Rapidly progressive nodules and masses	Dyspnea, coughing	Rapid deterioration of lung function (FVC, FEV1)
Interstitial pulmonary fibrosis	> 10 years	Diffuse reticulo-nodular pattern	Dyspnea, coughing	Restrictive alteration with decrease in DLCO
Simple Chronic Silicosis	> 10 years	Nodules < 1 cm (10 mm)	Asymptomatic	Normal
Complicated Chronic Silicosis	> 10 years	Masses > 1 cm (10 mm)	Dyspnea, coughing	Obstructive or restrictive impairment of variable severity

Spirometry is crucial in monitoring lung function for the possible effect of silica inhalation over time. It can identify normal ventilatory patterns, obstructive patterns, non-obstructive alterations (restrictive) or mixed patterns [10][13]. Individuals with silicosis face a threefold increased risk of pulmonary tuberculosis, necessitating screening for latent infection at the time of diagnosis with tuberculin skin test (PPD) or interferon-gamma release assay (IGRA) [14][15]. The onset of tuberculosis increases the risk of progression of silicosis and vice versa. Periodic monitoring of the PPD is useful for detecting latent and active tuberculosis, especially in the high-risk group with more than 10 years of exposure to silica. A hardening of 10 mm or more is considered diagnostic of latent tuberculosis, and it is recommended that patients with silicosis and positive tuberculin test results receive Isoniazid 300 mg/day (or 10 mg/kg/day) for six months or more recent schemes with Rifampicin or Rifapentine [15][16]. Silicoproteinosis is a rare and acute variant of silicosis that typically manifests with a shorter latency period, ranging from a few months to five years following initial exposure to crystalline silica. This condition leads to a rapid decline in lung function due to the accumulation of undegraded surfactant within alveolar macrophages [17]. The pulmonary toxicity of silica (SiO_2) is dose-dependent, meaning that even a relatively brief exposure to high levels can precipitate this uncommon complication. [17][18].

In advanced cases of silicosis, the coalescence of perinodular emphysematous regions (paracicatricial emphysema) can lead to the formation of bullae, significantly increasing the risk of spontaneous rupture and subsequent pneumothorax [19]. Reports have noted generally unilateral spontaneous pneumothorax in chronic silicosis patients, with incidence rates as high as 44% in a series of 50 patients from India [20]. Furthermore, the International Agency for Research on Cancer (IARC), part of the WHO, classifies silica as a proven carcinogenic in humans (Group I). This classification underscores the importance of performing chest CT scans in cases with a high suspicion of primary lung neoplasia alongside the characteristic silicosis lesions. The presence of masses or asymmetry in the pseudomasses on chest CT scans should raise the suspicion of concomitant pulmonary neoplasia. Distinguishing complicated silicosis from pulmonary sarcoidosis can be aided by radiological features, as the latter typically presents with centrally distributed confluent perilymphatic nodules that rarely calcify or cavitate [12][18].

While silicosis is commonly associated with quarry workers and those in the ornamental rock industry (granite and slate), recent reports have identified cases among workers in stone workshops exposed to silica dust from handling quartz agglomerates—commonly used in interior design, kitchens and sanitary ware—that can contain 70% to 90% crystalline

silica. This encourages the need for strict adherence to international occupational medicine regulations, such as those established by the ILO [18][20], to prevent new cases of this prevalent and often underdiagnosed pneumoconiosis in developing countries.

CONCLUSIONS

Silicosis is a diffuse interstitial disease resulting from prolonged inhalation of crystalline silica (SiO2), characterized by fibrotic changes in the pulmonary parenchyma. It falls under the category of pneumoconioses— lung diseases induced by inhalation of mineral dust. Pulmonary biopsy should only be reserved for cases where there is diagnostic uncertainty regarding primary pulmonary neoplasia, suspicion of silicosis-related tuberculosis (silico-TB) or acute presentations (such as silicoproteinosis). In such scenarios, a comprehensive immunoserological panel should also be conducted to assess the established correlation between prolonged silica exposure and autoimmune disorders.

Additional Information

Human Subjects: Informed consent was obtained or waived for all participants involved in this study.

Conflicts of Interest: In accordance with the ICMJE uniform disclosure form, all authors affirm the following:

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- **Other Relationships:** All authors disclose no other relationships or activities that might appear to affect the integrity of the submitted work.

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