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Case reports

From Stone Grinder to Lung Transplant Candidate: A Young Worker's Battle with Silicosis

Gabriela Roxana Louisse Neacșu[1], Agripina Rașcu[1,2], Dragoș Bumbăcea[1,3]

[1]University of Medicine and Pharmacy Carol Davila, Bucharest, Romania
[2]Colentina Clinical Hospital, Bucharest, Romania
[3]Pneumology, Elias Emergency University Hospital, Bucharest, Romania

Corresponding author
Gabriela Roxana Louisse Neacșu
louisse.neacsu@drd.umfcd.ro

Abstract

This article presents a compelling case study of a young stone grinder who, after nine years of dedicated labour, now faces the prospect of a life-altering lung transplantation due to the development of silicosis, a debilitating pulmonary disease stemming from inhalation of crystalline silica dust, commonly encountered in stone grinding industries. This article delves into the pathophysiology of silicosis, its insidious onset, and the countless challenges confronted by the young worker upon receiving this radical diagnosis.

This case highlights the critical importance of rigorous occupational safety protocols, early disease detection mechanisms, and the essential roles played by employers and healthcare professionals in preserving the well-being of individuals exposed to occupational hazards.

Keywords: *acute silicosis, occupational exposure, lung transplantation, crystalline silica, pneumoconiosis.*

Introduction

In the world of occupational health, the term "Silicosis" casts a long shadow, and rightly so. It's a lung disease that often lurks in the unseen corners of certain industries, quietly affecting those who work with materials like stone, sand, and concrete.

Silicosis isn't a household name, but it should be. It's caused by the inhalation of tiny crystalline silica particles, which are generated during activities like cutting, grinding, or drilling materials that contain silica. Over time, this exposure can lead to inflammation and scarring of the lungs, impairing breathing and, in severe cases, necessitating lung transplantation.

This young worker's journey serves as a vivid reminder that the daily labour of countless individuals can expose them to unforeseen health risks. Silicosis is a quiet menace that demands our attention. Through his story, we will not only delve into his personal struggle but also gain insight into the broader importance of occupational safety measures, early disease detection, and the vital roles played by healthcare professionals and employers in safeguarding the health of those who work in occupations filled with potential hazards.

It calls on us to reevaluate and elevate workplace safety standards, prioritize the health of our

workforce, and ensure that dedication to a craft does not come at the cost of one's well-being.

Case report

We present the case of a 32-year-old patient, a former smoker (~9 years), with occupational exposure for 9 years (2007-2016) to silica dust (formerly a stone grinder), diagnosed with mediastinal-pulmonary silicosis, acute-onset form, complicated by progressive massive fibrosis and chronic respiratory failure requiring oxygen therapy for almost 2 years.

Stone polishers typically do the following: place the blocks or slates on the workbench, fixing them between ends with the help of other workers, if necessary, to proceed with polishing. Eliminate roughness from the surface of blocks or slates, handling a grinding wheel, to give the required smoothness and contours. Polish the surfaces of blocks or slabs of granite, marble or other stones, using a polishing machine, to give them shine.

Workers involved in manufacturing, finishing and installing natural and manufactured stone are at risk for significant crystalline silica exposure. Crystalline silica commonly occurs in nature as the mineral quartz, and is found in granite, sandstone, quartzite, various other rocks, and sand. Workers who inhale very small crystalline silica particles are at risk for silicosis — an incurable, progressively disabling and sometimes fatal lung disease. Silicosis results in permanent lung damage. Silica dust particles become trapped in lung tissue, causing inflammation and scarring and reducing the lungs' ability to take in oxygen [1]. Symptoms of silicosis can include shortness of breath, cough and fatigue, and may or may not be obviously attributable to silica. Workers exposed to airborne crystalline silica also are at increased risk for lung cancer, chronic obstructive pulmonary disease (COPD), kidney disease and lung transplant [2].

Figure 1. Different-sized particles, grouped into three 'fractions' – the 'inhalable', 'thoracic', and 'respirable'

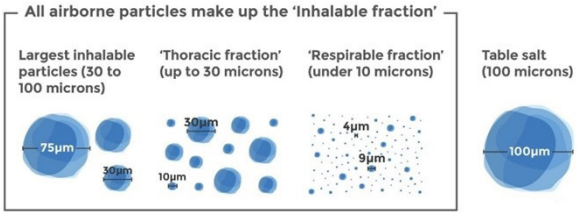


Table 1. Silica content of various types of stones [4]

Stone	Average % Silica
Engineered stone	≥93
Quartzite	95
Quarzitic sandstone	90
Sandstone	60
Granite	10-45
Slate	Varies
Soapstone	Varies

Respirable fraction of Respirable Crystalline Silica (RCS) dust (under 10 microns in diameter); the third and smallest fraction of crystalline silica particles is the respirable fraction or RCS. This fraction is small enough to penetrate the natural defences of the nose, throat and lungs, and enter the tiny alveoli deep in the lungs where gas is exchanged in the breathing process (Figure 1) [3].

The table below (Table 1) describes the typical silica/quartz content of common natural and engineered stones. Note that silica/quartz content will vary among stone varieties, and the exact content can be determined by petrographic and X-ray diffraction analysis on each rock [4].

The disease initiated in 2016 with progressive dyspnea, myalgias, and arthralgias in the lower limbs. Following a chest X-ray, and subsequently a CT scan, suspicion of pulmonary TB was raised, later disproved, leading to a decision for mediastinoscopy lymph node biopsy and right lung biopsy which supported the diagnosis of silicosis. Lymph nodes with remodelled architecture due to the presence of multiple circumscribed collagenized fibronodules, bordered by histiocytes laden with pigment described as lymph node silicosis (Figure 2).

Pulmonary parenchyma with significantly altered architecture, characterized by the presence of abundant eosinophilic material and endoalveolar microliths, some with dilated cystic spaces, associated with substantial interstitial thickening due to fibrosis and a rich diffuse lymphoplasmacytic inflammatory infiltrate; subpleurally, circumscribed collagenized fibronodules are described, bordered by anthracotic-laden histiocytes, appearance of acute pulmonary silicosis (Figure 3).

Figure 2. Lymph node silicosis (HE)

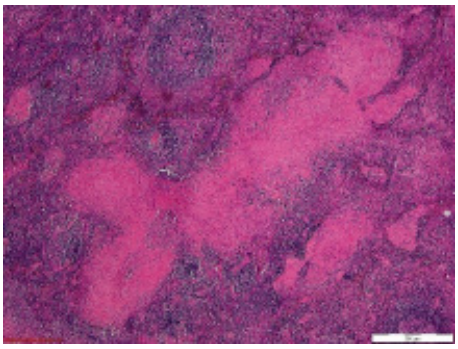
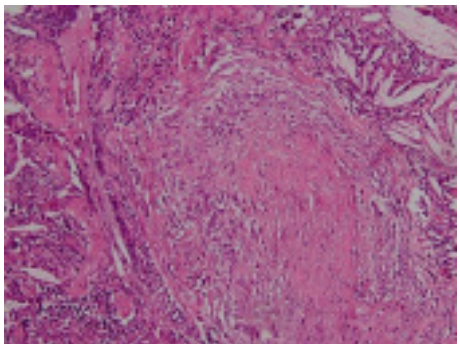


Figure 3. Acute pulmonary silicosis (HE)



Clinically, the patient presents exertional dyspnea of at least 2 years (with slight improvement under Nintedanib treatment), accompanied by arthralgias and myalgias in the lower limbs; no chest pain, cough, hemoptysis, but intermittent sensation of nasal obstruction for several years and recurrent episodes of epistaxis since 2016. Noteworthy medical history also includes a left spontaneous pneumothorax surgically drained in May 2022. On the physical examination, the general condition was affected, afebrile, lateral and suprasternal scars (post-right lung biopsy, post-left pleural drainage), digital clubbing, RR=18/min at absolute rest, but breathless with minimal exertion, bilateral diminished diffuse vesicular murmur, diffuse bilateral crackling rales, SaO2=78% in room air, at rest SaO2 92-93% with 3.5 L O2/minute/nasal cannula, regular heart sounds, HR=80/min, no detectable heart murmurs, no leg oedema, no signs of deep venous thrombosis, soft and mobile abdomen with respiration, not spontaneously or palpably painful, apparently no hepatosplenomegaly, non-palpable kidneys, conscious, cooperative, oriented to time-space and self. Normal electrocardiography with sinus rhythm.

Chest X-ray showed bilateral medium-to-high intensity large pulmonary opacities, with a central distribution, with signs of retraction and enclosed dilated bronchial lumens, associated with basal bilateral hyperinflation. Indistinct cardiac silhouette (Figure 4).

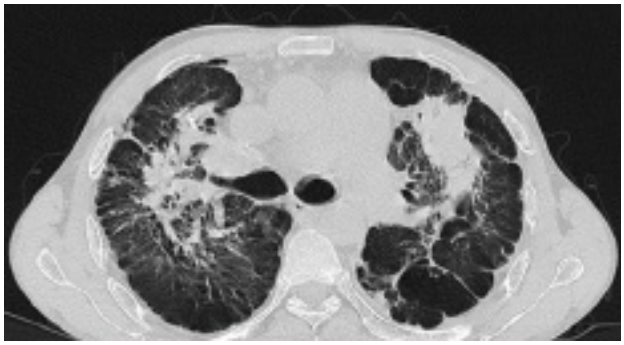
The Computed Tomography scan from 2023 showed bilateral localized fibronodular consolidations, predominantly in the central zone with fibrotic changes associated with microcalcifications.

Parenchymal emphysematous changes, centrilobular and para septal, bilateral. Generalized traction bronchiectasis with deformed walls due to fibrotic changes, associated with architectural distortions. Bilateral posterior and apical pleural thickening. Generalized lymphadenopathies, some with calcifications noted in the mediastinal region. Absence of pleural or pericardial effusions (Figure 5).

Figure 4. Chest x-ray: Acute silicosis with progressive massive fibrosis



Figure 5. Silicosis, Computed Tomography



The abdominal ultrasound revealed localized intrahepatic abnormalities, further evaluated by magnetic resonance imaging (MRI), and peri cephalic pancreatic adenopathy. Abdominal-pelvic MRI examination highlights mild hepatosplenomegaly and hepatic steatosis. Multiple hepatic focal processes with MRI characteristics of hemangiomas. A cystic lesion located in the peripheral zone of the middle lobe of the left prostate, displaying MRI characteristics of benignity, but requiring correlation with clinical echography data. The echocardiography presented an aortic bulb dilation, cardiac chambers of normal dimensions, ejection fraction normal, normal kinetics. Mild tricuspid regurgitation. Medium probability of pulmonary hypertension.

From the laboratory data on admission, the ferritin was elevated with normal C-reactive protein. The arterial blood gas test showed compensated respiratory acidosis with normal pH (7.41), elevated pCO2 (46 mmHg) and HCO3- (29.2 mmol/L), decreased pO2 (38 mmHg). The other biochemical tests were normal.

Pulmonary function tests (PFT) on admission revealed a severe mixed ventilatory dysfunction with the forced vital capacity (FVC) of 1.53 L (26% of predicted), forced expiratory volume in the first second (FEV1) 0.77 L (16% of predicted), modified Tiffeneau-Pinelli index (FEV1/FVC ratio) 50.4%, and total lung capacity (TLC) 37% of predicted.

Diffusing capacity of the lung for carbon monoxide (DLCO): DLCO = 5.77 mL/min/mmHg (16% of predicted) - severely reduced alveolar-capillary diffusion, low transfer constant.

The evolution of the PFT over the last 6 years, is presented below (Table 2).

Table 2. Pulmonary function tests over the last 6 years

PFT	aug 2017	dec 2017	jun 2018	aug 2018	2020	2022	mar 2023	jul 2023
TLC	N/A	N/A	N/A	6.27L / 83%	3.87L / 51.9%	5L / 67%	5.65L / 75.5%	2.8L / 37%
RV	N/A	N/A	N/A	3.87L / 220%	2.28L / 128.5%	3.9L / 212%	4.42L / 238%	1.27L / 68%
FEV1	N/A	N/A	N/A	1.61L / 35%	61.04%	62.18%	0.56L / 12.6%	0.77L / 16%
FEV1/FVC %	N/A	N/A	N/A	68%	21.70%	15.30%	45.26%	50.40%
FVC	N/A	N/A	N/A	1.89L / 32%	1.61L / 28.4%	1.09L / 20.5%	1.15L / 21.6%	1.53L / 26%
DLCO	4.14 / 32.9%	4.36 / 36.8%	3.71 / 29.7%	3.03 / 24.2%	1.47 / 11.9%	12.04%	N/A	5.77 / 16%

The Six-Minute Walk Test (6MWT): was performed under 1 L/min of supplementary oxygen through nasal cannula (flow needed to maintain resting SpO2 around 90-92%). The patient walked 62 meters (7.6% predicted), and stopped 67 seconds due severe dyspnea. SpO2 dropped from 90% to 73%. Dyspnea score on the BORG scale increased from 2 before test to 8 at the end of the test, and lower limb muscle fatigue score on the Borg scale from 2 before test to 8 at the end of the test [5].

A repeat 6 minute walking test was performed under oxygen titrated to maintain a SpO2 of at least 90%: This time the patient walked 220 meters (27.1% predicted [6]) and stopped after 3 minutes and 8 seconds due to severe dyspnea Borg Scale increased from 1 to 7 and lower limb muscle fatigue (Borg scale from 1 to 7). The SpO2 dropped during the test to 83% under 6l/min of oxygen.

The evolution of the Six Minute Walk Test over the last 5 years, is presented below (Table 3).

The patient with acute-onset mediastinum-pulmonary silicosis, currently under treatment with Nintedanib, exhibits severely impaired pulmonary function (FEV1 26% of predicted, FVC 37% of predicted, and DLCO 16% of predicted). The disease has clinically progressed since diagnosis. With a walking distance of only 7.6% of predicted value during the 6 minute walk test and severe hypoxia at rest, it is recommended that the patient be listed for lung transplantation.

Table 3. The evolution of the 6MWT over the years

6MWT	jun 2018	aug 2018	mar 2020	aug 2022	jul 2023	aug 2023
The distance	600m / 74.10%	568m / 71.5%	420m / 51%	N/A	62m / 7.64%	220m / 27.1%
The time stopped	6min	6min	5min, 30 sec	1min	67sec	3min, 8sec
The reason for stopping prematurely	SpO_2 <83, dyspnea	SpO_2 drops from 89% to 83%	SpO_2 drops from 90% to 74%, dyspnea	SpO_2 drops at 85%	SpO_2 <73, dyspnea, Added 1L O2	SpO_2 <83, dyspnea, chest pain, Added 6L O2
Borg Dyspnea Scale before	2	2	0	N/A	2	1
Borg Dyspnea Scale after	5	4	7	N/A	8	7

Discussion

In the journey from stone grinder to lung transplant candidate, we have witnessed the profound impact of occupational lung diseases on the lives of workers. The case of this young worker battling silicosis serves as a reminder of the urgent need for enhanced workplace safety measures and rigorous adherence to preventive guidelines. Occupational health physicians play an important role in safeguarding the well-being of workers, striving to prevent the progression of diseases like silicosis, and advocating for their patients' welfare. The consideration of lung transplantation in severe cases underlines the gravity of these conditions and the importance of early intervention. Measures include controlling dust levels below the permissible exposure limit of 50 μg/m3 (micrograms of silica per cubic meter of air) as an 8-hour time-weighted average (TWA). This is believed to be the maximum daily concentration that workers can be exposed to for a working lifetime without adverse health effects [7].

Figure 6. The workers shown above are wearing an approved N95 filtering facepiece respirator (left), a full facepiece elastomeric respirator (centre), and a loose-fitting powered air-purifying respirator (PAPR) (right) [8].



By learning from such stories, advocating for change, and embracing advancements in medicine and workplace safety, we move one step closer to a future where the journey from stone grinder to lung transplant candidate becomes a rarity, rather than a reality.

A recent article about the current global perspectives on silicosis mentions that the outbreaks of silicosis linked to the production of household kitchen countertops using artificial stone with high silica content have drawn attention due to the relatively young age of affected labourers, brief periods of exposure to RCS and the rapid advancement of the disease [9].

Global Burden of Disease study from 2017, identified a total of 23,695 incident cases of silicosis, which represents 39% of the 60,055 incident cases of pneumoconiosis.

A detailed evaluation of the patient's work-related and medical history is crucial to prevent misdiagnosis in instances of suspected silicosis. Such misdiagnosis could have significant implications for treatment, disease progression, and the overall prognosis of affected individuals.

As mentioned in a 2020 study, an incomplete history may result in a diagnosis of sarcoidosis rather than silicosis in the same patient [10].

Misdiagnosis can lead to the omission of a detailed occupational history and adequate long-term screening. This, in turn, may result in unnecessary treatments and investigations, ultimately contributing to a poorer prognosis and diminished quality of life [11].

Silicosis remains a prominent industrial health concern globally. In the current landscape, where the identification of silicosis relies solely on late and irreversible radiological alterations, there is a growing need for specific biomarkers in the screening protocol for individuals exposed to silica [12].

Considering the patient's work history, the typical clinical signs and the findings from radiological imaging, an initial diagnosis of acute silicosis was suggested and later confirmed through histological examination. The patient's condition had progressed to the point where a lung transplant was considered the only treatment option.

Key points

Addressing the prevention of pneumoconiosis resulting from exposure to modern materials, like synthetic aggregates containing elevated levels of crystalline silica, is imperative.

This case highlights an intriguing clinical challenge when distinguishing between pneumoconiosis and sarcoidosis in the differential diagnosis.

This case serves as a compelling example of a rare yet largely preventable disease with severe health consequences and an unfavourable prognosis.

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