


CASE REPORT

Silicosis in a paint-production worker: Study of a lung histological specimen with scanning electron microscopy—Energy dispersive X-ray spectrometer

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Abstract

The occupational risks in silica-exposed workers have been greatly reduced over the last decades; however, only few studies investigated uncommon forms of exposure to crystalline silica as shown by this case of silicosis in a paint-production worker.

KEYWORDS

crystalline silica, paint worker, scanning electron microscopy, silicosis

1 | BACKGROUND

We reported an uncommon case of silicosis in a worker involved for 25 years in the production of paints (water-based paints and quartz paints for the building industry). The etiology was confirmed by investigating a lung histological specimen with a scanning electron microscope equipped with an energy dispersive X-ray spectrometer.

Crystalline silica is a mineral widely found on earth as part of sand, rock, and mineral ores such as quartz. The most common free crystalline forms of silica in workplaces are quartz, tridymite, and cristobalite.¹ The International Agency for Research on Cancer (IARC) in 1997 classified crystalline silica as a known carcinogen in humans (Group 1).² Recent epidemiologic studies strongly support the conclusion that

silica exposure increases the risk of lung cancer in humans independently of confounding factors such as cigarette smoking.¹

Silica exposure causes many other adverse health effects, including silicosis, cardiovascular disease, tuberculosis, autoimmune disease, and various kidney disorders.^{3,4} Together with the increase in mortality, these findings make silica exposure a priority concern for public health,^{5,6} in addition to urban air pollution.⁷⁻¹¹

Silicosis is a primary pneumoconiosis involving fibronodular lung disease caused by the inhalation of dust containing crystalline silica particles.¹ It is an irreversible, disabling, and incurable disease. There are three types of silicosis: acute (developing within weeks to a few years), accelerated (developing within 10 years), and chronic (developing more

than 10 years after initial exposure). The onset of disease is influenced by both the concentration and duration of exposure. A diagnosis of acute silicosis is supported in exposed subjects who have experienced a rapid onset and/or worsening of symptoms, including dyspnea, cough, fever, and pleuritic pain. More difficult is the diagnosis of chronic silicosis, which can be asymptomatic or may present with only mild-to-moderate exertional dyspnea.¹ The diagnosis of silicosis requires carefully documented records of occupational exposure and radiological features with the exclusion of other competing diagnoses.

Although prevention efforts have been made for many decades, silicosis remains a problem worldwide. Recent reports show that over 33 million workers in China¹² and India,¹³ over 3.2 million workers in Europe,¹⁴ and approximately 1.7 million workers in the United States¹⁵ are exposed to crystalline-free silica. Silicosis is an occupational health concern and was one of the first recognized occupational diseases in Italy. Exposure to respirable crystalline silica (aerodynamic diameter < 10 µm) occurs in many industries and occupations, such as the metallurgical sector, the extraction of coal and metal, building construction, the production of construction materials, and the paint industry.

With regard to the latter, it is known that the health effects deriving from professional exposure to paints are numerous and concern both those involved in the production of paint products and those involved in their application. Particularly affected may be the respiratory and skin systems through an irritative and allergic reaction to the chemical agents that workers transport and handle (ingredients, diluents, dyes) or that are released during processing (solvents, mists).^{16,17} To date, however, cases of pneumoconiosis in workers involved in the production of paints have not yet been reported.

2 | CASE PRESENTATION

A 49-year-old man, a former smoker for approximately 12 years of approximately 20 cigarettes/d, was a specialized worker in charge of the production of paints and employed by a company in Southern Italy that supplied water-based paints, quartz paints, and wall paints and coatings for the building industry from 1994 to 2018. The work performed by the patient for 8 h/d consisted of opening bags containing the raw paint materials in powder form (calcium carbonate, micronized talc, titanium dioxide, sand, ventilated quartz flour, silica sand, stone dust, colored pigments, etc) and pouring them manually into tanks and cisterns to mix them with water by a rotating blade placed on the bottom. After this phase, the final product is verified and packaged. In addition, the operator was tasked with carefully monitoring the evolution of the production cycle and eventually completing the composition of the final product by introducing other dusty materials. The

worker reported that his company provided him with FFP2 masks but that he only used them occasionally. Furthermore, the work environment first installed workplace dust extractors only in 2008.

The worker was healthy until 2017, the year he suffered from exertional dyspnea, dry cough, chest pain, and widespread arthralgia. At the beginning of 2018, he underwent a spirometric test with DLCO, the results of which were within normal limits, and a radiological examination (chest X-ray) with evidence of reinforcement of the peribronco-basal plot on the entire left lung and subtotal area on the right, obliterated costophrenic sinus on the left and no parenchymal outbreak lesions. The chest CT scan performed a few days later showed an isoexpanded left lung with irregular pleural thickening capturing posterobasal contrast, numerous parenchymal lung micronodules in the upper lobes, intercavo-aortic adenopathies, bilateral apical fibrosclerosis, and some thin pleural plaques. In April 2018, he underwent total body PET-TAC, which showed an accumulation of the radiopharmaceutical tracer at the level of the carinal, subcarinal, mediastinal, and anterior superior mediastinal lymph node stations. In July of the same year, he underwent atypical resection surgery of the right upper lung lobe. The histological examination performed on the pleuropulmonary fragments, on the fragments of the pulmonary nodules, and on the lymph nodes showed multiple nodular formations with subpleural, peribronchial, and perivascular sites; some were composed of aggregates of macrophages with a hint of central sclerosis, while others were composed entirely of fibrolamellar connective tissue delimited by chronic histiocytic inflammation, with a tendency of confluence. These nodules showed small refractive accumulations in the context and were extended to the contiguous parietal pleura, showing phenomena of sclerosing pleurisy. The remaining lung parenchyma showed aspects of emphysema and fibrosis of the interstitium and mid-intima of the vessel wall. The morphological picture, in consideration of the anamnestic data of professional exposure, was suggestive of pneumoconiosis and in particular of silicosis. The patient was then discharged with a diagnosis of silicosis.

The possibility that the disease was generated by the presence of inorganic particles was demonstrated by investigating the histological specimen, observed by a scanning electron microscope (SEM) equipped with an energy dispersive (ED) X-ray spectrometer. The morphology and chemical composition of the very small inorganic particles present in the histological sample were obtained by an SEM (LEO Zeiss, model EVO50XVP) coupled with an X-max (80 mm²) silicon drift Oxford spectrometer equipped with a Super Atmosphere Thin Window. Investigations were performed using the following operating conditions: 15 kV accelerating potential, 500 pA probe current, and gas chamber pressure 10 Pa.¹⁸

The SEM-EDS investigations revealed that many dusty inorganic particles were present in the histological sample,

almost exclusively silicate phases, whose dimensions varied from 0.5 to 45 μm (Figure 1); specifically, silica (SiO_2) minerals were recognized, followed more rarely by phases with compositions similar to those of feldspars (Figure 1) or micas.

To ascertain that the silica (SiO_2) minerals were not amorphous phases, it was necessary to analyze the very small particles by powder X-ray diffraction (PXRD). The paraffin-embedded sample was thus placed in a porcelain crucible and burned in a kiln (Figure 2) at 750°C and 101 kPa (1 Atm), in the presence of oxygen for 1 hour to eliminate the organic part of the sample (paraffin and human tissue) and leave only its inorganic part (silicates); the refractory inorganic component was finely ground in an agata mortar and finally positioned onto a plexiglass holder above a villiumite (NaF) support, which was also used as the standard for peak calibration. The X-ray investigations were performed using a Philips X'Pert Pro diffractometer equipped with an X'Celerator position-sensitive detector. The operating conditions were as follows: graphite-monochromated $\text{CuK}\alpha_1$ radiation, X-ray tube power supply 40 kV and 40 mA, divergence slit 1°, antiscatter slit 1/2°, programmable slit 0.2 mm, step size 0.02° 2 θ , time for step 1 seconds, and scansion range 2-70° 2 θ .

The PXRD scans (Figure 2) revealed the nearly exclusive presence of tridymite with a very low quantity of quartz; the small amounts of other silicates, identified by SEM-EDS (feldspars and micas), were present in quantities so low that they were below the detection limit of the PXRD equipment used.

In July 2018, the patient underwent further radiological and spirometric tests with DLCO, which were unchanged compared to the previous tests, and visited a specialist in occupational medicine. On this occasion, the patient was given a diagnosis of “anatomoclinical picture of bilateral pulmonary silicosis with spirometric values within the limits of the norm,” and the first certificate of occupational pathology was drawn up.

3 | DISCUSSION AND CONCLUSION

The exposure to ventilated quartz dust, silica sand, and stone dust occurred over the years from opening bags with and pouring and mixing raw materials, necessary steps in the production of paints, which appears to be the cause of the onset of silicosis. In fact, all these substances contain free crystalline silica, the known etiological agent of the pathology, and the worker did not always use personal protective equipment (PPE). Furthermore, the work environment only installed workplace dust extractors in 2008; consequently, the patient worked for approximately 14 years in the absence of this ventilation system.

However, for the present case, it was not possible to obtain up-to-date environmental monitoring data related to the concentrations of respirable silica dust. In Italy, there is no national exposure limit value for crystalline-free silica, and the threshold limit value-time-weighted average (TLV-TWA) of the American Conference of Governmental Industrial

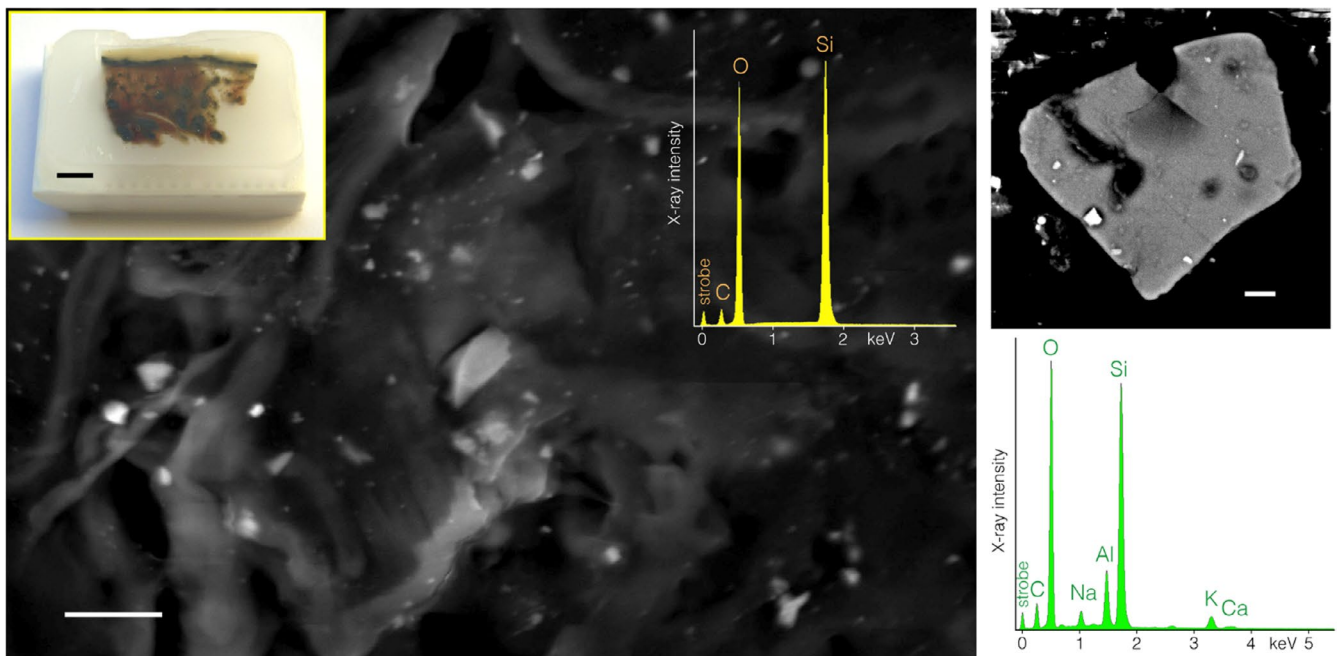


FIGURE 1 SEM images of dusty inorganic particles (almost exclusively silicate phases) present in the histological sample. Left part of the figure: particles of silica (SiO_2); right part of the figure: a particle with feldspar-like composition. Scale bar is 5 mm

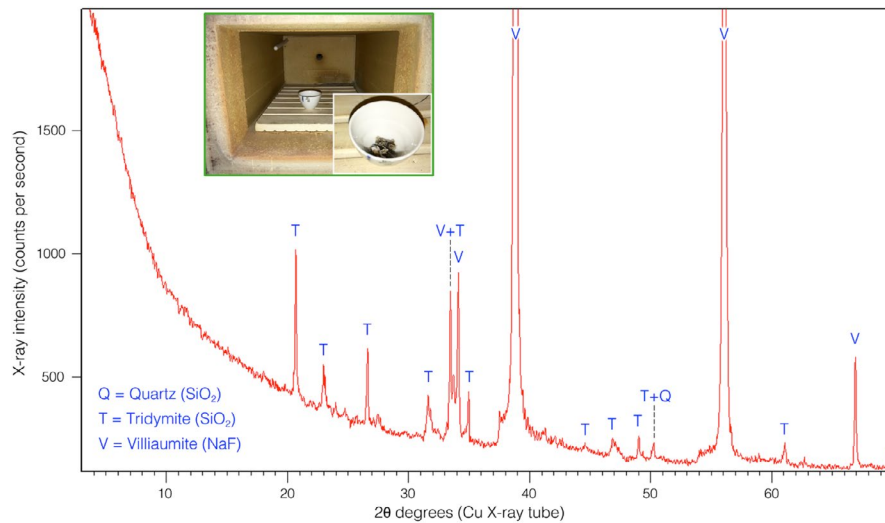


FIGURE 2 Powder X-ray diffraction scan of the silicate particles present in the histological sample revealed the nearly exclusive presence of tridymite with a very low quantity of quartz

Hygienists (ACGIH) is generally adopted. This value was lowered from 0.1 to 0.050 mg/m³ in 2000, and in 2006, it was further reduced to 0.025 mg/m³.¹⁹ Moreover, the new directive (EU) 2017/2398 adds free crystalline silica to the list of recognized carcinogenic substances and sets the occupational exposure limit value to 0.1 mg/m³.²⁰

This is the first case of silicosis ever described in a worker involved in the production of paints. Although the IARC has ascertained that workers involved in the production of paints may be exposed to free crystalline silica,²¹ there are no correlation studies in the scientific literature on the comorbid onset of pneumoconiosis, while few studies have focused on painters.²²

A general trend among paint manufacturing companies is to reduce worker exposure through the use of ventilation systems or fully closed automated production lines. However, many small companies continue to manufacture paints without such technologies. It is therefore appropriate to report cases such as this one to allow the adoption of adequate organizational and preventive measures to prevent the onset of silicosis and other similar diseases.

ACKNOWLEDGMENTS

The authors have no acknowledgments. Informed and written consent was obtained from the participant. The patient was informed that data from the research protocol would be treated in an anonymous way, with scientific methods and for scientific purposes in accordance with the principles of the Helsinki Declaration.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

AUTHOR CONTRIBUTIONS

LDM, AC, PL, and LV: designed the work. VL: acquired the data for the work. PA and MP: analyzed the data. MCD and DC: interpreted the data. All authors participated in the

drafting and revision of the work and gave the final approval of the version to be published. All authors agreed to be responsible for all aspects of the job in ensuring that issues relating to the accuracy or integrity of any part of the job are properly investigated and resolved.

ETHICAL APPROVAL

Ethical approval is not necessary because all medical and instrumental examinations were performed according to Italian laws concerning the protection of workers exposed to occupational risks (D. Lgs. 81/2008).

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