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Complicated Chronic Silicosis: Findings in Asymptomatic Patient

Silicosis crónica complicada, hallazgo en un paciente asintomático

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ABSTRACT

Silicosis is caused by sustained inhalation of silicon dioxide (SiO_2). Work in quarries and industries of ornamental rock, granite and slate, and marble work, are the typically described types of exposure, together with sandblasting. Denim sandblasting and the production of kitchen and bathroom countertops have emerged more recently. Clinical presentations can be classified into chronic silicosis (simple, complicated, and pulmonary fibrosis), accelerated silicosis, and acute silicosis, depending on years of exposure and individual factors. We present a clinical case of a 78-year-old male patient with complicated chronic silicosis, asymptomatic, diagnosed in the context of a pre-surgical examination.

Key words: Silicosis; Occupational diseases; Lung Diseases, Interstitial

RESUMEN

La silicosis es producida por la inhalación mantenida de dióxido de silicio (SiO_2). Los trabajos en canteras e industrias de roca ornamental, granito y pizarra, y marmolerías son las exposiciones clásicamente descriptas, junto al pulido con chorro de arena. El arenado de jeans, producción de mesadas de cocinas y baños han surgido más recientemente. Las presentaciones clínicas pueden clasificarse en silicosis crónica (simple, complicada y fibrosis pulmonar), silicosis acelerada y silicosis aguda, según los años de exposición y factores individuales. Se presenta un caso clínico de un paciente masculino de 78 años con silicosis crónica complicada, asintomático, diagnosticado en contexto de un examen prequirúrgico.

Palabras clave: Silicosis; Enfermedades profesionales; Enfermedades pulmonares intersticiales

INTRODUCTION

Silicosis is one of the oldest known occupational lung diseases. It is caused by sustained inhalation of silicon dioxide (SiO_2), mostly in the workplace, although it has also been described in the home environment. With early suspicion, the patient can be isolated from the source of exposure and change the prognosis and evolution of the disease. This work proposes the publication of this clinical case for the purpose of emphasizing the importance of patient inquiry in identifying diseases related to environmental and work exposure and obtaining an early diagnosis of diseases that are rare but can be prevented.

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CLINICAL CASE

78-year-old male patient referred by the Traumatology Service to do a pre-surgical study. The patient is a former smoker (<5 p/y) with history of arterial hypertension, and has a pacemaker due to a sinus node disease. As for his work, he is a retired police officer. At the moment of the consultation, he is medicated with aspirin and enalapril; asymptomatic in the area of the respiratory system. Oxygen saturation ($Sp0_2$) is 98% with inspired fraction of 0.21; normal breath sounds, no adventitious sounds. The general physical examination is unremarkable.

A spirometry was performed, with the following results: FEV1/FVC, 54%; FEV1, 1.93 (75%); FVC, 3.54 (107%), with evidence of mild obstruction. The chest X-ray shows heterogeneous radiopacities with tendency to confluence, with predominance of left upper lung field (Figure 1).

Due to the findings of the chest X-ray, the patient was inquired again, and he said he worked in a family-operated medal polishing shop (10 hours a week, for more than 10 years). Chest tomography requested. The results showed bilateral masses in posterior segments of upper lobes with spiculated borders, fibro-cicatricial tracts, heterogeneous, with 58 mm × 35 mm, and 35 mm × 28 mm calcifications. These images are associated with bullae plus emphysema and bilateral mediastinal adenomegalies with calcifications (Figures 2A and 2B).

DISCUSSION

Silicosis is one of the oldest known occupational lung diseases, and is included in the group of interstitial diseases. It is produced by sustained inhalation of SiO_2. Crystalline silica is a natural metal oxide. Among the different varieties, the most abundant is the quartz, found in rocks and sand. In a lower volume, there are other polymorphisms such as cristobalite and tridymite, both from volcanic rocks, and stishovite, which doesn't have fibrotic potential[1].

The silica is phagocytized by alveolar macrophages, thus activating and perpetuating the inflammatory process through inflammatory cy-

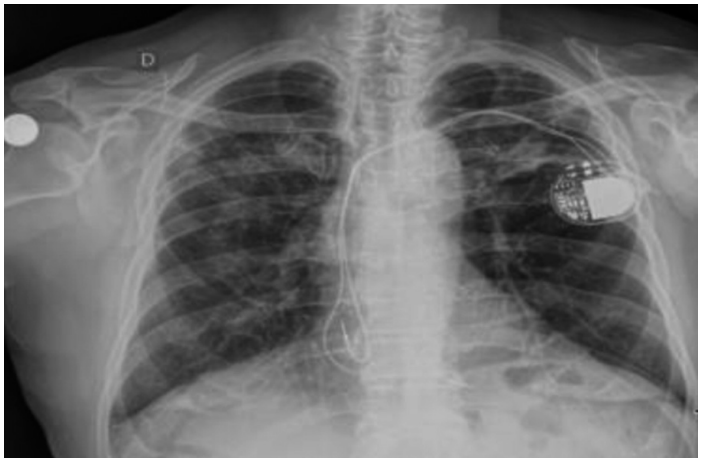

Figure 1. Front chest X-ray: heterogeneous radiopacities with tendency to confluence, with predominance of left upper lung field.

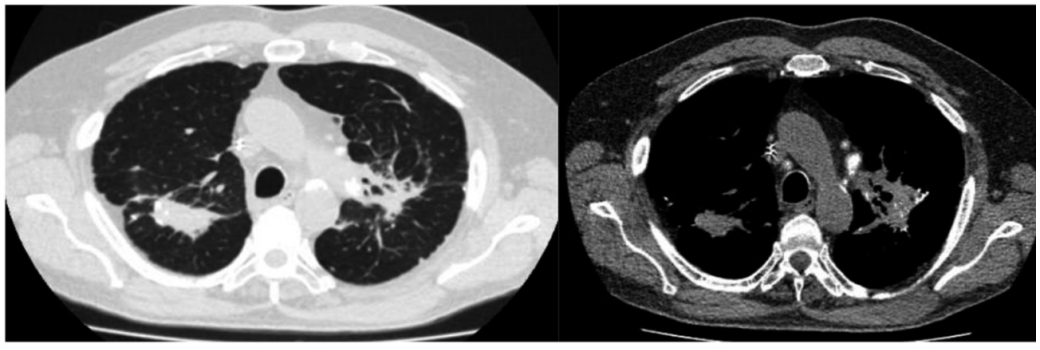

Figure 2A and 2B. Figures 2A and 2B. Chest CAT with lung window (2A) and mediastinal window (2B) showing bilateral masses in posterior segments of upper lobes with spiculated borders, fibro-cicatricial tracts, heterogeneous, with 58 mm x 35 mm, and 35 mm x 28 mm calcifications. These images are associated with bullae plus emphysema and bilateral mediastinal adenomegalies with calcifications.

1 Loading door for material to be used
2 First sand container
3 Second sand container (reserve)
4 Manual handling of raw material to be carved
5 Glass divider
6 Protective measure used
7 Sand vacuum
8 Compressor
9 General view of the shop and machinery

Figure 3. Home shop, where we can see a box with a clearly deteriorated upper glass; behind the worker, sand collecting bag and inadequate personal protective element showing signs of wear.

tokines (tumor necrosis factor-alpha [TNF-a] and interleukin-1 [IL-1]) that recruit inflammatory cells in the alveolar wall. These cells release toxic derivatives of oxygen and proteolytic enzymes that cause cellular damage and destroy the extracellular matrix, thus producing a fibrotic response in the pulmonary parenchyma[2].

There are multiple sources of exposure, mainly in the workplace, but they may also occur in the home environment (home exposure). Work in quarries and industries of ornamental rock, granite and slate, and marble work, are the typically described types of exposure, together with sandblasting.[1] In the early '90s, new industries emerged involving denim sandblasting, mostly in Turkey, also including the production of kitchen and bathroom countertops with quartz. If countertops are grinded without dust suppression, they generate a dust level 300 times above the recommended value[3]. In Israel, approximately 3,500 workers cut and process countertops that contain 93% of silica as raw material[4]. In 2000, the existence of 3.2 million people exposed to silica was reported in the European Union[5]. In 2007, a 4-year follow-up study was published in which 145 former denim sandblasting workers were evaluated, 83 of which were reevaluated in 2011; 6.2% (9 individuals) had died, with an average age of 24 years, and the prevalence of silicosis among the remaining 74 survivors increased from 55.4% to 95.9%, with evidence of radiographic progression in 82%[6]. The SWORD (Surveillance of Work-Related and Occupational Respiratory Disease) report described 216 cases of silicosis from 600,000 exposed workers between 1996 and 2017 in the United Kingdom[7]. A study conducted in 132 companies of 31 different sectors in Chile estimated that 5.4% have a high probability of exposure to silica[8]. Real figures regarding its prevalence in Argentina are unknown.

The risk of developing silicosis depends on several factors. First, the magnitude and duration of the exposure, given by the calculation of the cumulative dose (see Table 1) and individual sensitivity, which is determined by genetic and environmental factors. The silica dust, after being inhaled, is deposited in the lungs and presents a complicated process of depuration that leads to its retention, because the mechanisms of defense and ciliary clearance lose effectiveness. Those mechanisms are boosted by smoking or the presence of some respiratory underlying condition, such as COPD (chronic obstructive pulmonary disease)[1]. This justifies the fact that there isn't any safe, zero-risk exposure threshold. However, in Argentina, there is a maximum permis-

sible concentration of crystalline silica (cristobalite) regulated through Resolution N° 295/2003 of the Ministry of Labor, Employment and Social Security, which is 0.05 mg/m^3; and 0.1 mg/m^3 for quartz[1].

Clinical presentations can be classified into chronic silicosis (simple, complicated, and pulmonary fibrosis), accelerated silicosis, and acute silicosis. The first are the most common and can be observed after 10 to 15 years of exposure. Patients with simple silicosis tend to be asymptomatic, with 2-5 mm centrilobular nodule images predominating in the upper lobes and posterior segments. Thus, silicosis can progress to the complicated type in patients with dyspnea and cough, nodules bigger than 1 cm that tend to merge forming conglomerate masses of irregular shapes, marked retraction, and cicatricial emphysema. Patients usually show mediastinal adenopathies with calcifications. In pulmonary fibrosis, the predominant symptom is also dyspnea with images similar to idiopathic pulmonary fibrosis. Accelerated silicosis requires from 5 to 10 years of exposure, with features intermediate between chronic and acute silicosis, but higher frequency of progression to severe forms of the disease. Finally, acute silicosis is manifested after less than 5 years of exposure, generally very high levels of exposure, with dyspnea, weight loss and rapid progression to respiratory failure. Radiologically, we observe a bilateral perihilar alveolar pattern with "ground glass" opacities, similar to alveolar proteinosis; that is why it is also called "silicoproteinosis" (Table 2).

The diagnosis is based on the presence of exposure to silica, radiological findings and exclusion of other diseases. As regards the exposure, as we were saying, most cases occur in the workplace environment, so it is important to evaluate current and past activities and to describe the type of work that was carried out, and the mechanisms of protection used, both environmental and individual. Radiological studies (Rx-CAT) are essential for the diagnosis and evaluation of disease progression. Other diagnostic studies are indicated only in atypical cases or if the patient has a poorly-documented history of exposure. Anatomopathological studies are required in exceptional cases.

Lung function tests are necessary for follow-up and to assess possible progression. The spirometry shows the presence or absence of ventilatory defects (obstructive and non-obstructive) and facilitates the diagnosis of other diseases. A decrease in the forced expiratory volume on the first second (FEV1) can be observed in patients exposed to silica, which has a synergistic effect with smoke in smoker patients. If the spirometry is altered, we recommend to do a complete functional assessment[1]. Regarding the diffusing capacity for carbon monoxide (DLCO), it is reduced in the complicated forms of the disease. SpO_2 measured with pulse oximeter, arte-

TABLE 1. Calculation of silica cumulative dose

Silica cumulative dose = respirable dust fraction X% free silica in mg/m^3 X number in years of exposure

Guía de actuación y Diagnóstico de Enfermedades Profesionales. 05-Silicosis. Ministry of Production and Labor-Presidency of the Argentine Nation, 2018. Available at https://www.argentina.gob.ar/sites/default/files/5_guia_de_actuacion_y_diagnostico_-_silicosis_0.pd

TABLE 2. Clinical forms of silicosis

Clinical form	Exposure time	Radiology	Symptoms	Pulmonary function
Simple chronic	> 10 years	Nodules < 10 mm	None	Normal
Complicated chronic	> 10 years	Masses > 1 mm	Dyspnea, cough	Obstructive or restrictive alteration of variable severity
Interstitial Pulmonary fibrosis	> 10 years	Diffuse reticulonodular pattern	Dyspnea, cough	Restrictive alteration with decreased diffusing capacity
Accelerated	5-10 years	Nodules and masses of rapid progression	Dyspnea, cough	Rapid decline in lung function (FVC and FEV_1)
Acute	< 5 years	Bilateral acinar pattern similar to alveolar proteinosis	Dyspnea	Generally restrictive alteration with decreased diffusing capacity

Extracted from the SEPAR guidelines (Spanish Society of Pneumology and Thoracic Surgery): Normativa para el diagnóstico y seguimiento de la silicosis. R. Fernandez Älvarez et al / Arch Bronconeumol, 2015; 51(2): 86-93 (4)

rial blood gases, and exercise tests, such as the 6-minute walk test (6MWT), together with the clinical severity assessment, allow us to outline the behaviors.

There isn't any effective treatment for this disease, that is why prevention is essential. Among the preventive measures, engineering controls come first: eliminate exposure by substituting raw materials, making changes and adjustment of processes, insulation and ventilation. To control the respirable dust levels within the legal limits is an important measure, together with the use of personal protective elements, since we must remember that the damage threshold is variable among patients.

Early diagnosis and prevention of complications are part of the secondary prevention. People exposed to silica should participate in a health surveillance program that includes medical record, spirometry and chest X-ray on a periodic basis, and according to clinical symptoms.

Tertiary prevention makes reference to avoiding disease progression, treating the obstructive defect, if present, providing home oxygen supply, giving the influenza and pneumococcal vaccines and possible transplantation. We recommend the screening and treatment of the latent tuberculous infection. It is worth mentioning that association with autoimmune diseases has been described: sclerodermia, rheumatoid arthritis and systemic lupus erythematosus (SLE), among others, even lung cancer.

CONCLUSION

We present this case because it is a rare, underdiagnosed disease. In the exposed case, the patient gets to an advanced age with no symptoms.

The physician has to identify the patient's work and environmental history that could imply exposure, since it is a preventable disease that doesn't have a treatment. Decree N° 658/96 of occupational diseases enumerates the most frequent activities that involve exposure[1]. From the occupational point of view, when a patient is diagnosed with silicosis, he must avoid exposure, also the disease has to be declared as an occupational disease, and the patient must relocate his/her job or be declared as disabled. In the work environment, it would behave as a surveillance event that imposes better epidemiologic surveillance measures to prevent new cases.

Conflict of interests

The authors declare that there is no conflict of interests.

REFERENCES

1. Guía de actuación y Diagnóstico de Enfermedades Profesionales. 05- Silicosis. Ministry of Production and Labor-Presidency of the Argentine Nation, 2018. Available at https://www.argentina.gob.ar/sites/default/files/5_guia_de_actuacion_y_diagnostico_-_silicosis_0.pdf
2. Rimal B, Greenberg AK, Rom WN. Basic pathogenetic mechanisms in silicosis: current understanding. Curr Opin Pulm Med. 2005; 11: 169-73. https://doi.org/10.1097/01.mcp.0000152998.11335.24
3. Hoy RF, Chambers DC. Silica-related diseases in the modern world. Allergy. 2020; 75: 2805-17. https://doi.org/10.1111/all.14202.
4. Ophir N, Shai AB, Alkalay Y, et al. Artificial stone dust-induced functional and inflammatory abnormalities in exposed workers monitored quantitatively by biometrics. ERJ Open Res. 2016; 2: 00086-2015. https://doi.org/10.1183/23120541.00086-2015
5. Fernández Álvarez R, Martínez González C, Quero Martínez A, et al. Normativa para el diagnóstico y seguimiento de la silicosis. Arch Bronconeumol. 2015;51:86–93. https://doi.org/10.1016/j.arbres.2014.07.010
6. Akgun M, Araz O, Ucar EY, et al. Silicosis Appears Inevitable Among Former Denim Sandblasters: A 4-Year Follow-up Study. Chest. 2015; 148: 647-54. https://doi.org/10.1378/chest.14-2848.
7. Barber CM, Fishwick D, Carder M, van Tongeren M. Epidemiology of silicosis: reports from the SWORD scheme in the UK from 1996 to 2017. Occup Environ Med. 2019; 76: 17-21. https://doi.org/10.1136/oemed-2018-105337.
8. Protocolo de vigilancia del ambiente de trabajo y de la salud de los trabajadores con exposición a sílice, Ministerio de salud, Subsecretaría de salud pública, División de políticas públicas saludables y promoción, Departamento de salud ocupacional, Chile, junio 2015. Available at: https://dipol.minsal.cl/departamentos-2/salud-ocupacional/exposicion-a-silice/