VERSITA

ARS Medica Tomitana - 2012; 3(70): 140 - 145
DOI 10.2478/v10307-012-0027-5

Constandina Claudia[1], Raşcu Agripina[2], Trăilescu Ana Maria[3], Danteş Elena[1], Halichidis Stela[1], Arghir Oana Cristina[1]

Pulmonary silicotuberculosis in an electrician male - Case report and literature review

[1]Faculty of Medicine – Constanta „Ovidius" Universitatea
[2]Faculty of Medicine – „Carol Davila" University, Bucharest
[3]„Victor Babeş" Clinical Hospital Bucharest

ABSTRACT
This is a case report of a pulmonary silicotuberculosis in a former smoker, male, 43 years old, having 21 years of occupational exposure to particulate coniotic-free crystalline-silicon dioxide as an electrician, developing symptoms as fever higher than 38°C, dry cough and diffuse chest pain, being diagnosed with miliary of the lung in 2002. Silicosis was confirmed later by histological exam obtained through an exploratory thoracotomy and it was included in the transient first to second stage of pneumoconiosis. Three years later, in July 2005, Pulmonary Tuberculosis was diagnosed by acid-fast stains positive smears. The evolution of the case was to a progressive deterioration till 2008, leading to silicosis stage III. Silicosis is a pulmonary fibrosis which must be always suspected in persons working in conditions of occupational exposure to dust of silicon dioxide, having suggestive radiological changes including micronodular radiological pattern or pseudotumoral one. Once the diagnosis of silicosis is confirmed, tuberculosis may be frequently associated. The more advanced silicosis is, the more the combination of the two diseases is commonly revealing and, often, the TB morbidity among workers in the silica industry exceeds that of general population. A hint orientation for the silicotuberculosis' diagnosis, in this reported case, was represented by the radiological dynamic of the lesions. Tuberculosis lesions are less dense and imprecisely defined, located in upper lobes and develop necrotic centers.

Keywords: silicosis, miliary, pulmonary tuberculosis, exploratory thoracotomy

Arghir Oana Cristina

Faculty of Medicine Constanta;
Universitatii Street, No. 1, Campus B, Constanta, Romania;
Constanta Clinical Hospital of Pneumophtysiology;
Sentinelei Street, N0. 40, Palazu-Mare, 900002 Constanta, Romania;
E-mail address: arghir_oana@yahoo.com;
Telephone: +40722636201;
Fax: +40241636350

Introduction

Silica (silicon dioxide) is the most abundant mineral on earth, existing in both crystalline and amorphous forms. Amorphous forms, including vitreous silica and diatomite (formed from skeletons of prehistoric marine organisms), are relatively nontoxic after inhalation. In contrast, inhaled crystalline silica is associated with a spectrum of pulmonary diseases. Silicosis is referring to a group of pulmonary diseases caused by the inhalation of free crystalline silica known as silicon dioxide (SiO2 lc). The oldest written record of occupational lung disease caused by silica inhalation extends back to ancient Egypt and Greece. Despite a clear understanding of

how to prevent this disease, new cases of silicosis continue to occur. Any pneumoconiosis, but silicosis in particular, carries an increased risk of associated tuberculosis (TB). Complicated silicosis is a serious progressive disease recognizing no specific therapy. [1] Patients with this disease are constantly facing the threat of pulmonary tuberculosis. The onset of TB is often insidious and the underlying chest X-ray's image abnormalities make the diagnosis even more difficult. Living and practicing in an area with increased TB rate, the opportunity to observe complicated silicosis with pulmonary tuberculosis appears greater. The hazard of TB occurrence in patients with silicosis has been described by many researchers. Fletcher found, in 1955, tubercle bacilli and histological changes of TB in 40% of cases of progressive massive fibrosis caused by silicosis. [2] Cathcard described the form of cavitary pulmonary TB in 69% of miners (n=182/1,690). [3] Cochrane noted that large accumulations of silica increased the attack and mortality rates from TB. [4] The combined occurrence of silicosis and pulmonary TB creates a suggestive radiological pattern, frequently making early and precise diagnosis impossible.

Material and methods

S.T. was an adult male born in 1966, moderate former smoker (15 packs-year), retired medical degree in 2002 after 21 years of working as a senior professional electrician (8 years IT Republic, Metrorex 3 years, 10 years SC. Glass Art), was admitted in July, 11th 2005, to Colentina Clinical Medicine Hospital. Family history, occupational exposure history and medical history of the patient were questioned. The respiratory and general symptoms were reviewed. Clinical exam, chest X-ray, laboratory investigations were performed. Silicotuberculosis was diagnosed, anti tuberculosis therapy was recommended and, three years later, in March 2008, the patient was evaluated both by pulmonologyst and occupational disease specialist.

Results

Family History revealed no evidence of any history of chronic diseases and disorders and no Known contact or exposure to tuberculosis.

Occupational Exposure History referred to a previous profession of an electrical work practiced for a long period of 21 years, including 8 years in Republica Factory, 3 years in Metrorex and 10 years in Glass Art Factory. The last job involved working in 3 shifts, in a large hall, where the following activities were met: melting lead, making lead profiles, production of stained glass, glass cutting, grinding, rolling, welding, sandblasting glass (pistols with compressed air), electrical and mechanical repairs. The patient participated in all above mentioned operations. Occupational exposure to silicosis appears very suggestive for pneumoconiosis caused by SiO2 dust (exposure time -TE = 10 years, latency time -TL= 9 years , retention time -TR = 15 years) associated with other respiratory nuisance, gases, vapors of lead in stressful working conditions in noise, unfavorable microclimate and prolonged standing.

Medical History revealed no significant pathologic disorders before silicosis was diagnosed in October, 2002, in the Colentina Clinical Medicine Hospital, Bucharest, Romania, excepting an accident mentioned in 1996, during working time, with a left forearm ligament section consequence.

In July, 11th 2005, the patient was admitted to Colentina Clinical Medicine Hospital for prolonged productive cough with purulent expectoration, weight of loss (~ 3 kg in 3 weeks) secondary to anorexia, moderate dyspnea, sweats, bilateral interscapulovertebral pain, gradually increasing in the last three months.

Clinical exam revealed cyanotic lips and no clubbing, the frequency of respiratory movements was 21 breaths / min, and heart rate was 102 beats / min.

Spirometry showed a decrease of 31% of vital capacity and a moderate distal obstructive syndrome (MEF50 reduced by 62% of the predicted value).

Pulsoximetry (SaO2) at rest was 94% and after

6 minute walk test indicated a decreasing in saturation of arterial oxygen to 88%.

Radiological pattern of silicosis showed major changes from the initial moment of October, 2002, when silicosis stage I/II was diagnosed, having nodular opacities relatively symmetrically disposed, to clouded and quite asymmetrically nodular opacities distributed through both pulmonary fields, mainly in the upper lobes and in the lower right lobe, being very suggestive for an evolutive Pulmonary Secondary TB (Figure 1).

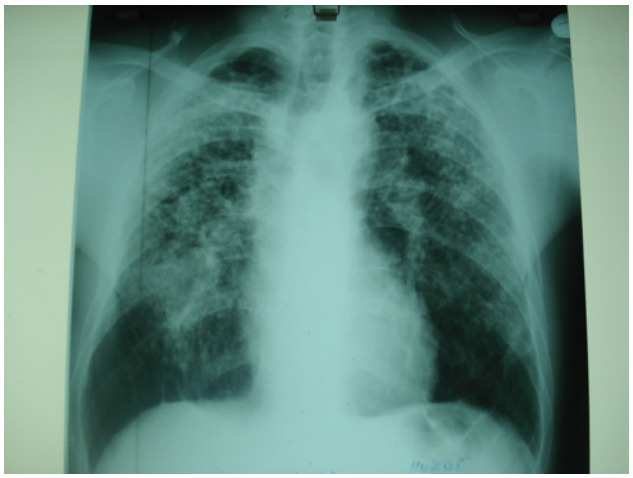

Figure 1- Male patient, S.T. – Bilateral and asymmetrical distribution of homogenous and non homogenous nodular lesions in both upper lobes: hyper transparency of the bases of the lungs, 11th of July, 2005

Sputum smears were positive for acid-fast bacilli (AFS) and, 2 months later, positive culture confirmed Pulmonary Active Tuberculosis disease.

Antituberculosis treatment was started in August 2005 with daily administration of first directly observed (DOT) regimen 4 drugs included (isoniazid 300 mg/day, ethambutol 1600 mg/day, rifampin 600 mg/day and pirazinamide 2000 mg/day) for two months.

Negative smears after 2 months of anti tuberculosis therapy were considered to be the bacteriological marker of a good evolution and intermittent DOT therapy (three times weekly-3/7) continued with isoniazid (H) 600 mg/day and rifampin (R) 600 mg/day, being well tolerated with no adverse reactions.

After three months of treatment, the patient was discharged and he continued recommended therapy with HR 3/7 up to other 9 months, symptomatic bronchodilator therapy (metilxantine), associated systemic corticosteroids, mucolytic bronchial secretion (acetylcisteine 600mg once a day) and long-term oxygen therapy (4 l/min, 12 hours/day).

The patient diagnosed with silicotuberculosis was evaluated three years latter, in March 2008, both by pulmonologyst and occupational disease' specialist, concluding to an obvious deterioration to an advanced stage III of silicosis (C, 3q, r, hi, tb, em) because an extensive progressive fibrosis mass and macro nodular lesions occurred and latent pulmonary failure was associated. The long-term evolution of tuberculosis lesions after 12 months of administered DOT was satisfactory.

Discussion

Estimating the harmful potential impact on health of a SiO2 dust's exposure is only possible through a detailed examination of the environmental and raw material content, technological processes that may cause the release of the dust. Quantitative and qualitative assessment of the dust is insufficient to characterize a professional exposure, if one doesn't know details about the profession itself. Frequently, during work, the worker is exposed to multiple jobs of varying concentrations of dust. For a glazier, the exposure to silica is incurred in preparing materials and polishing operations and silicosis is considered to be the most common disease in exposed workers of the small factories with a small-scale production type, in which the sand may still be used for grinding wheels. The enameled glass operations and all metal objects can cause significant releases of dust rich in free silica, too. The occupation in electrical work area isn't very suggestive for silicosis mainly in non-occupational specialties.

In Romania, silicosis ranks first in the occupational morbidity. [5, 6] The risk of silicosis

increases gradually, depending on the concentration of respirable dust in SiO2 and the type of silica: quartz, tridymit, cristobalite existing in the workplace's atmosphere. [3,5]. In case of concurrent or subsequent exposure to mixed dusts, mixed type of disease may appear, as silicoantracosis, silicosiderosis. [3,5-7]. In the exposed population of workers age, men are mostly exposed. By age, the highest number of cases is registered at 40-49 years, followed by the age group of 50-59 years and 30-39 years. [8]

The inhalation of any one of a wide variety of the inorganic dusts may result in the development of a diffuse interstitial radiological syndrome. [8] The chest x-ray's changes with varying degrees of impairment of pulmonary function are developing further. [9,10] The diagnosis of any of the occupational diseases named "pneumoconiosis" is usually established on the basis of a typical professional history of exposure combined with the characteristic radiological changes of the lungs.

Radiological findings in simple silicosis include the presence of widespread nodules measuring 2-5 mm in diameter, with predominance in the middle and upper lung zones. [6,9] These nodules may grow slowly in size and show the trend to conglomeration and eventually the formation of larger poorly circumscribed masses, to which the term of progressive massive fibrosis is applied. [6,9,10] These lesions are sometimes related with tuberculosis. Other manifestations include the presence of the reticular and small nodular opacities in bilateral lung areas and large round opacities observed, more commonly, on the right side. [6] Small reticular and nodular patterns are scattered diffusely throughout both lungs and provide a background for the massive conglomerate lesions, which may show cavitation even in nontuberculous cases. [9] "Eggshell" calcifications of the hilar adenopathy may be present. [9]

Tuberculosis is regarded as one of the most common complications in patients with silicosis. [4,8] It is generally accepted that silicosis facilitates the development and propagation of tuberculosis, because the prevalence of tuberculosis silicosis-related is much higher than in the general population. [1,4,8] One or more irregular, poorly circumscribed mass lesions may appear in the lung in individuals with silicosis and may represent a complicating tuberculosis disease or a progressive massive fibrosis. [6,9]

In our case, the history of the silicosis, as an occupational disease, recognizes an unusual method of diagnosing. In August 2002, the patient was hospitalized in the Clinical Infectious Diseases Department of "Victor Babeş" Hospital, Bucharest for a prolonged febrile syndrome accompanied by an intense chest pain and a persistent dry cough. The chest X-ray image on August, 15th 2002, suggested the diagnosis of a pulmonary miliary syndrom. The radiological differential diagnosis between silicosis and miliary tuberculosis, at that moment, was not done because no professional exposure to SiO2 was discussed. In order to specify the etiology of lung miliary syndrom, there were performed several investigations, such as computerized tomography of the chest, bronchoscopy with bronchial aspirate for acid-fast bacilli, with negative smears and cultures.

Respiratory functional tests showed normal ventilatory function and lung biopsy was finally performed through an open thoracotomy in the Thoracic Surgery Department of Tunari Hospital, Bucharest.

The positive diagnosis of silicosis was established by the histological exam in October 2002 and the resolution of the Medical Occupational Committee was "Pneumoconiosis: silicosis stage I/II" as painting radiological lesion (nodular opacity, uniform, dense, with different sizes, symmetrical located in both middle and upper lung fields) revealed, silicosis being staging by the radiological criteria 2-3 p, q, hi.

After tuberculosis was diagnosed in 2005, over the next 3 years, the evolution of silicosis became progressively worse and the last assessment of the Pneumoconiosis Committee (March 2008) concluded silicosis stage III (C, 3q, r, hi, tb, em), pulmonary failure.

In the twentieth century, in Europe, the frequency of the association between silicosis and tuberculosis was higher in pathological than in the clinical statistics. [1,2,4] The consequence is that the two diseases are related one to another. [5,8] In terms of evolution, the combination of these two medical conditions implies a bad prognosis. The tuberculosis' preventive treatment administered in

patients diagnosed with silicosis does not influence the disease risk. [1]

The clinical picture of silicosis is nonspecific, so symptoms often translate this morbid complication, as the most common being, pulmonary tuberculosis. Dyspnea is the most common symptom, being triggered by exercise, with a progressive decrease of the tolerance to effort. Cough is initially dry, often in the morning, caused by the exposure to dust and smoke. Chest pain is frequently present, with a bearable intensity. The signs of general fatigue and sweats during physical exertion are frequently reported.

Radiological pulmonary pattern is, along with professional history, the most suggestive method of silicosis' positive diagnosis and allows assessing staging process, evolution and detecting complications, while providing a basis for classifying the clinical and mainly radiological stages of silicosis.

Radiological signs characteristic of silicosis are homogeneous nodular opacities isolated localized in the lung or medial and basal conglomerates located mainly bilaterally, symmetrical. Silica lesions of grades I-II are usually symmetrical, their density and distribution increasing to two lower thirds.

The appearance of tuberculosis is most often in the upper thirds, first unilaterally, changing the previous radiological images which become asymmetric. [8] In the third stage of silicosis, the massive pseudotumoral masses represent the main radiological pattern. The degree of tuberculosis reactivation of post primary disseminative lesions rise to a rate of 2-5% in general population, while in grade III silicosis proportion is much higher (40-50%). [6,7,9]

If pulmonary lesions move to the top of the lung, this dynamic change may be considered a useful clue in tuberculosis' diagnosis orientation. The tuberculosis lesions are less dense and bounded towards the imprecise silica ones have a place of choice in the apical and dorsal segments of upper lobe, apical segment of the lower lobe and have a fast dynamics to ulceration, with the appearance of tuberculosis caverns.

In our case, considering the imaging appearance of pulmonary military preceding surgery, the differential diagnosis could interfere with the following conditions: pulmonary TB, pneumoconiosis, pulmonary hemosiderosis, sarcoidosis, bronchopulmonary cancer with intrapulmonary metastases, lung infections: viral, mycosis, ricketsiosis, parasite pulmonary disease (toxoplasmosis, toxocariosis, hidatic cyst), allergic disease (Löeffler syndrome). Histological examination of the lung biopsy tissue fragments collected by open lung surgery was the only clue for a positive diagnosis of silicosis.

Histological features are the silica nodules composed of silica particles surrounded by collagen fibers (concentric ridge), with macrophages, lymphocytes and fibroblasts in the peripheral area. Nodules are surrounded by bulae of emphysema, especially in areas next to pleural slide. Birefringent silicon crystals within silica nodule can be identified by polarized light microscopy or by methods with high degree of precision that combining "scanning electron microscopy" by X-ray spectroscopy. [7]

The particularity of the reported case consists in the presumptive diagnosis of pulmonary miliary which was not associated with the obvious increased occupational risk factor of silicosis. The minimized value of anamnesis - highly suggestive of professional silicosis, conducted to the open pulmonary biopsy through a thoracotomy.

The second feature of the case is represented by the associated pulmonary tuberculosis in an early stage of silicosis, since literature data show that pulmonary tuberculosis frequently occurs when lesions of silicosis are advanced (stage III) [1,9]. Both diseases affect each other adversely. In this case, tuberculosis is an aggravating factor for silicosis. In the context of epidemiological slow but continuous growth of silicosis incidence, physicians have not right to forget the huge informative value of occupational anamnesis and the relationship between silicosis and pulmonary tuberculosis. Our case was diagnosed with silicosis by an invasive method but benefited of an early treatment and diagnosis of pulmonary secondary tuberculosis. No reactivation of tuberculosis was found after first episode ended till 2008, even the evolution of silicosis was progressively unfavorable.

Conclusion

Because of the complexity of modern industrial processes, significant exposures to toxic substances or inorganic dust may occur in occupations that are seemingly far removed from such possibilities. Silicosis must be also suspected in electrical trade workers. The occupational history is of a great importance, particularly if the chest X-ray reveals the presence of a multi micronodular pattern. Silicosis is frequently complicated with pulmonary tuberculosis, especially in an endemic TB area.

References

1. Morgan E.J. (1979). Silicosis and Tuberculosis. *Chest*. 75 (2, February), 202-203. DOI:10.1378/chest.75.2.202.
2. Fletcher C.M. (1955). Epidemiological Studies of Coal Miners Pneumoconiosis in Great Britain. *Arch.Industr. Health.* 11, 29.
3. Silicosis and Silicate Disease Committee (1988). Diseases associated with exposure to silica and nonfibrous silicate minerals. *Arch. Pathol. Lab. Med*. 112, 673-720.
4. Cochrane A.Q.L. (1954). Tuberculosis and Coal Workers' Pneumoconiosis. *Brit. J. Tuberc*. 48, 274.
5. Cocârla A. (2009). *Medicina ocupațională,* Ed. Medicală Universitară "Iuliu Haţieganu" Cluj-Napoca, România.
6. Raşcu A., Artenie C., Naghi E., Şerbescu A. & Gherman D. (2004). *Silicoza.* Ed. Universitară, Bucureşti, România.
7. Naghi E. (2002). *Pneumoconiozele*. Ed. Punct, Bucureşti, România.
8. Bogdan M.A. (2008). *Pneumologia.* Ed. Universitatea "Carol Davila" Bucureşti, România.
9. Lillington G.A. (1987). *A diagnostic approach to chest diseases (IIIrd ed.)* Williams & Wilkins, Baltimore, USA.
10. Cohen R.A.C, Patel A. & Green F.H.Y. (2008). Lung Disease Caused by Exposure to Coal Mine and Silica. *Dust Semin. Respir. Crit. Care Med.* 29(6), 651-661. DOI: 10.1055/s-0028-1101275.