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Case Report | Olgu Sunumu

# INSIDIOUS DISEASE DIAGNOSED 20 YEARS AFTER EXPOSURE: SILICOSIS, A CASE REPORT

## *MARUZ KALIMDAN 20 YIL SONRA TANI ALAN SİNSİ HASTALIK: SİLİKOZİS, BİR OLGU SUNUMU*

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**ABSTRACT**

Pneumoconiosis is defined as an irreversible disease characterized by the accumulation of inorganic dust in the lungs and the fibrotic tissue response to the accumulated dust. The occurrence of the disease is directly related to the duration of exposure to dust, the amount of dust, its physicochemical properties and dust control measures of the environment. The cause is obvious and it is a completely preventable disease. It is one of the important problems in silicosis, the most common and known type of pneumoconiosis, that it continues to progress even after dust exposure ends. Our case is a 46-year-old male patient who was mainly exposed to silica dust for a total of five years. The detailed history, physical examination, imaging, and laboratory results of the patient who applied for exertional dyspnea after many years of silence were evaluated. In the patient's anamnesis, the history of working in a quartz mine 20 years ago played a key role in the diagnosis together with radiological imaging, and it was decided that he had silicosis after excluding some diseases included in the differential diagnosis. We wanted to draw attention to the fact that in such cases, the progression of the disease after exposure, the ability to remain silent for decades, and most importantly, the detailed work history of the patients helps the diagnosis.
**Keywords:** Pneumoconiosis, silicosis, exertional dyspnea, quartz, silica

**ÖZ**

Pnömokonyoz, inorganik tozların akciğerlerde birikmesi, biriken toza karşı akciğerlerde gelişen fibrotik doku yanıtıyla karakterize, geri dönüşümsüz bir hastalık olarak tanımlanır. Hastalığın meydana gelmesi, toza maruz kalınan süre, tozun miktarı, fizikokimyasal özellikleri ve ortamın toz kontrol tedbirleri ile doğrudan ilişkilidir. Nedeni bilinen ve tamamen önlenebilir bir hastalıktır. Pnömokonyozun en yaygın ve bilinen türü olan silikozisde toz maruziyeti sonlansa bile progresyonuna devam etmesi önemli sorunlardan biridir. Olgumuz, 46 yaşında erkek hasta olup toplam beş yıl boyunca ağırlıklı olarak silika tozuna maruz kalmıştır. Sessiz geçen uzun yılların ardından efor dispnesi nedeniyle başvuran hastanın detaylı öykü, fizik muayene, görüntüleme, laboratuvar sonuçları değerlendirildi. Hastanın anamnezinde 20 yıl önce kuvars madeninde çalışma öyküsü radyolojik görüntülemeyle birlikte tanıda anahtor rol oynadı ve ayrıcı tanıya giren bazı hastalıkların da dışlanması ile silikozis olduğuna karar verildi. Olguyla, bu tür durumlarda maruziyet sonrası hastalığın progresyonunun devam etmesi, dekatlar boyu sessiz kalabilmesi ve en önemlisi, hastaların detaylı iş öyküsünün alınmasının tanıya yardımcı olmasına dikkat çekmek istedik.
**Anahtar Kelimeler:** Pnömokonyoz, silikozis, efor dispnesi, kuvars, silika

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## Introduction

Silicosis is one of the oldest occupational diseases known to man, caused by breathing in crystalline silica dust. This progressive disease causes death due to pulmonary fibrosis and unfortunately there is no current treatment.[1] The most common forms of crystalline silica in occupational exposures are quartz, tridymite and cristobalite. The duration of exposure to silica can lead to one of three forms of disease; chronic silicosis (after exposure to respirable dust containing less than 30% quartz for more than 10 years), subacute or accelerated silicosis (after exposure to a heavier dust load within 2-5 years), and acute silicosis (usually within a few months after exposure to finer and denser dust). Workers with a high potential for contracting the disease; miners, foundry workers, tunnel drillers, quarry workers, stone carvers, ceramic workers, silica flour production workers and sandblasters.[2] The diagnosis of silicosis is usually based on a history of significant exposure to silica dust and concordant radiological features, with the exclusion of other diagnoses such as miliary tuberculosis, fungal infections, sarcoidosis, idiopathic pulmonary fibrosis, rheumatoid nodules, other interstitial lung diseases, and carcinomatosis. Chest X-ray consists of generally faint-looking opacities, 1-10 mm in diameter, located in the upper lobe and posterior part of the lung. High Resolution Computed Tomography (HRCT) scan findings; upper lobe posteriorly located uniform, centrilobular and subpleural small nodules, hilar and mediastinal lymphadenopathies (some in the form of "eggshell"), pleural-parenchymal bands, emphysema, etc. occurs. Some nodules may be calcified.[3,4] There is no specific treatment method. The primary treatment procedure is to eliminate the source of exposure and to enroll patients in a pulmonary rehabilitation program.[4]

## Case Report

Our case is a 46-year-old male patient who applied with the complaint of exertional dyspnea, which has been increasing for the last 10 years. Although he applied to hospitals with this complaint several times before, a clear diagnosis was not made. When the patient's work history was taken, he worked as a vibratory drill and dynamite worker in an iron-oxide ore mine between 1992-1995 and in a quartz mine between 1997-1999. After drilling the rocks with a drill and placing dynamite, he performs the blasting operation and then makes the necessary controls. He said there was serious dust around. For the last 20 years, he has been working part-time as a cook in a restaurant. He described no dust exposure in the restaurant. The patient has a history of diabetes mellitus and hypertension. The father died of lung cancer. No history of drug use. Although he has a 10 pack-year smoking history, he quit 16 years ago. No ral or rhonchi was heard in the physical examination. Pulmonary Function Test (SFT): FEV1/FVC: 76%, FEV1: 3.38 L (88%), FVC: 4.41 L (94%), DLCO_SB: 24.5 ml/(min*mmHg) (77%), KCO_SB: 4.79 ml/(min*mmHg*L) (107%), TLC_SB: 5.31 L (74%). He finished the six-minute walking test at 356 meters without getting tired (before: BORG: 2, saturation: 99%, heart rate: 92/min; after: BORG: 3, saturation: 99%, heart rate: 104/min). Biochemistry, hemogram, sedimentation, C-reactive protein and rheumatological tests were within normal values. HRCT was requested after faint nodules were seen in the upper zones in the patient's chest X-ray (Figure 1).

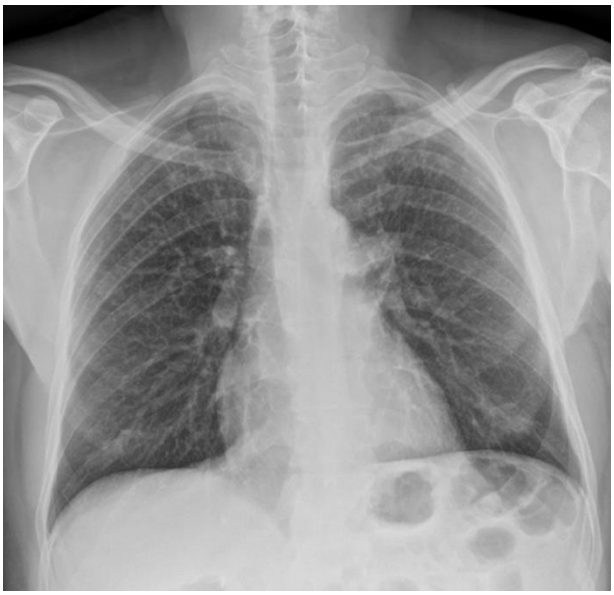

**Figure 1.** Chest X-ray of the patient

HRCT of the patient was reported as multiple lymphadenopathies, some eggshell-shaped, in the mediastinum and hilar region, multiple small-sized pulmonary nodules with perilymphatic distribution pattern, prominent in the upper lobes of both lungs, pleural-parenchymal thickenings, and fibrotic changes accompanying the nodules (Figure 2). After excluding the diseases included in the differential diagnosis (such as sarcoidosis, rheumatoid nodules, tuberculosis and fungal infections), we accepted the case as chronic complicated silicosis and we evaluated the posteroanterior chest X-ray as q/q 2/2 at, ax, hi, es according to the International ILO (International Labor Organization) Pneumoconiosis Standardization.

## Discussion

Our case worked as a vibratory drill and dynamite worker in the mine for a total of about five years, two years apart. As a result of the detailed occupational anamnesis, imaging findings, biochemical markers, pulmonary diseases and radiology consultations of the patient who applied to us for the investigation of dyspnea that occurred years later, we decided that he had chronic complicated silicosis, excluding other causes.

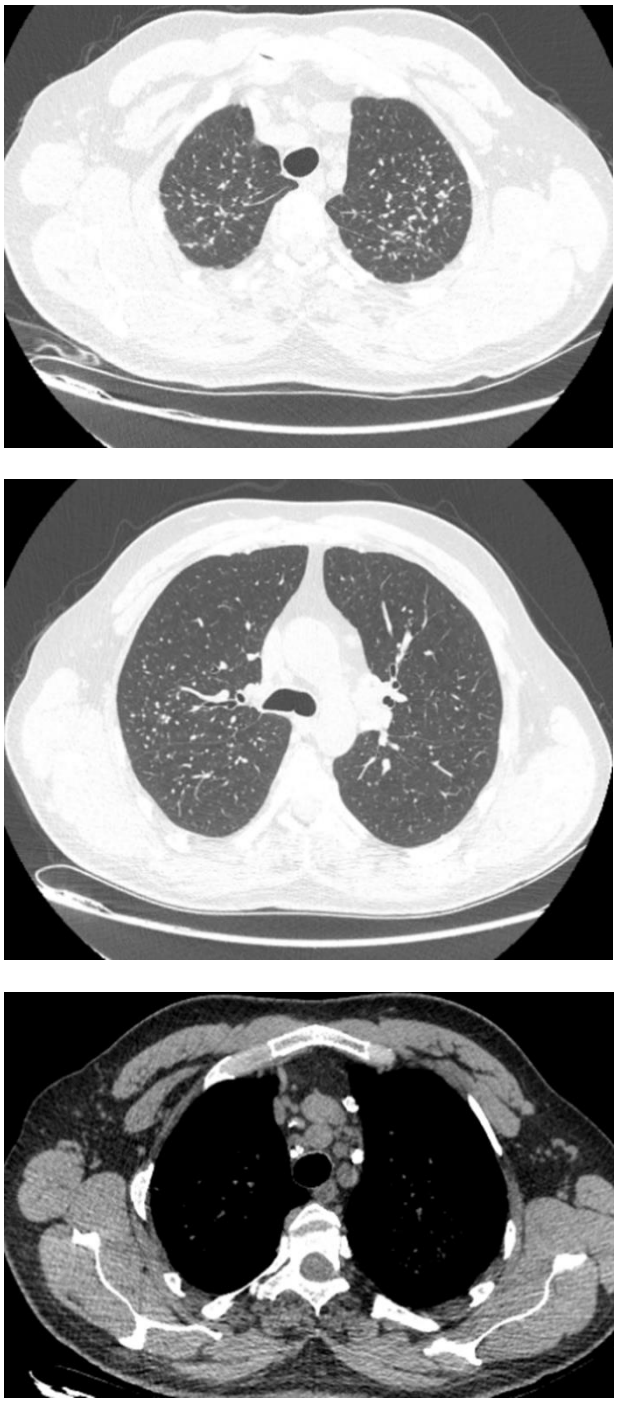

**Figure 2.** HRCT image of the patient

Chronic silicosis is the most common form of the disease. It develops after more than 10 years of exposure to relatively low-to-moderate concentrations of silicon dust. Patients may present with a wide range of clinical findings ranging from asymptomatic status to chronic cough, exertional dyspnea, fatigue, and weight loss. Classical radiological finding is diffuse nodular pattern mostly in bilateral upper lobe and posterior. Nodules are less than 10 mm in diameter. However, when the nodules merge, they form "choalescences" exceeding 10 mm, which is an indication of progress towards Progressive Massive Fibrosis (PMF). Hilar “eggshell” calcifications may be seen. The diagnosis is made according to a well-received occupational history, radiological imaging and differential diagnosis of other diseases. If necessary, bronchoalveolar lavage (BAL), lymph node sampling and lung biopsy can be performed. Today, invasive procedures are avoided unless necessary for the diagnosis of silicosis.[3,5]

Unlike many interstitial lung diseases, silicosis is selected because it is seen at a relatively younger age, progresses more slowly, and is seen in more than one person in the same environment. In an interstitial lung disease, the diagnosis of silicosis can easily be overlooked unless a comprehensive occupational history is taken. In a pathology series, occupational etiology was shown to be overlooked in 25% of lung biopsies with prediagnosis of Idiopathic Pulmonary Fibrosis (IPF), and the diagnosis was made by mineralogical analysis.[3]

In silicosis, the disease may develop or progress even after occupational exposure has ceased. Due to the long latent period (years-decades) from initial exposure to disease development, some cases of silicosis may not be diagnosed until the end of their lives.[6,7]

With this patient, we wanted to draw attention to what kind of picture the exposure to dust caused in a total of 5 years due to the nature of the work he did 20 years ago. As can be seen from the patient's history, he had no respiratory problems during the period he worked in the mine, and he had no emergency medical record (for acute or accelerated silicosis). This is a sign that chronic complicated silicosis can occur after less than 10 years of dust exposure.

In conclusion, it should not be forgotten that a good occupational history should be taken from every patient presenting with symptoms such as exertional dyspnea, sputum, cough, and if the patient's chest radiology findings indicate interstitial and parenchymal involvement. In addition, occupational exposures not only cause pneumoconiosis but may also increase the risk of idiopathic interstitial lung diseases. When evaluating a new case of interstitial lung disease (ILD), the occupational history should be questioned before calling it idiopathic, and in the absence of occupational exposure, the definition of idiopathic should be used.

**Compliance with Ethical Standards**

Informed consent was obtained from the patient.

**Conflict of Interest**

The authors declare no conflicts of interest.

**Author Contribution**

YSH: Design, concept, data collection, writing, literature search; MT: Design, concept, writing

**Financial Disclosure**

None

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