Co-Existence of Tuberculous Meningitis and Pulmonary Tuberculosis in a Denim Sandblaster

Kot Taşlamaya Bağlı Silikozis Hastasında Tüberküloz Menenjit ve Akciğer Tüberkülozunun Birlikteliği

Kemalettin Ozden¹, Omer Araz², Elif Yılmazel Ucar², Fatih Alper¹, Metin Akgun²
¹Department of Infectious Diseases, Faculty of Medicine, Ataturk University, Erzurum, Turkey
²Department of Pulmonary Diseases, Faculty of Medicine, Ataturk University, Erzurum, Turkey
³Department of Radiology, Faculty of Medicine, Ataturk University, Erzurum, Turkey

Abstract

Silicosis is a well-known occupational lung disease that was discovered by the ancient Greeks and Romans. In 2001, it has emerged again in an unexpected occupation: denim sandblasting. Exposure to crystalline silica, with or without clinical disease, is one of the most important predisposing factors for the development of tuberculosis; however, there has been no previous report of tuberculosis among cases of silicosis due to denim sandblasting. Herein, we report the first case of a denim sandblaster with silicosis who developed both pulmonary tuberculosis and tuberculous meningitis.

Key Words: Denim sandblaster, Pulmonary tuberculosis, Tuberculous meningitis

Introduction

It is well known that silicosis, or even silica exposure without silicosis development, is one of the most important risk factors in the development of tuberculosis [1-3]. Silicosis due to denim (or jean) sandblasting in the textile industry has recently been reported as a new cause of silicosis [4-6]. In this process, sifted sea sand has been used as an abrasive to produce worn-out jeans. Because of high silica exposure and younger worker population, silicosis has developed in a relatively short period of time with fatal outcomes [6, 7]. Although some cases had been initially misdiagnosed as tuberculosis [4], there has been no previous report of tuberculosis among the cases of silicosis due to denim sandblasting. Herein, we report the first case of a denim sandblaster with silicosis who developed both pulmonary tuberculosis and tuberculous meningitis.

Case Report

A 21-year-old man was admitted to our hospital with a history of malaise, dry cough and fever with chills for the previous fifteen days. He was prescribed ceftriaxone 1 gram twice daily for one week with no benefit. On admission, the patient still had a fever (40 °C), although the rest of the physical examination was unremarkable. Based on his occupational history, five years ago, he had worked in a textile factory for approximately four months. His primary job was sandblasting of jeans to abrade them to produce a worn-out appearance. He had been diagnosed with silicosis two years ago. Laboratory investigations revealed a WBC of 13,000 cells/mm³ with increased neutrophils (85%), a CRP of 2.5 mg/dl and an erythrocyte sedimentation rate of 40 mm/h. Sputum analysis for tuberculosis and the tuberculin skin reaction were both negative. Other laboratory tests, including assays for Brucella because of its endemicity in our region, were unremarkable except for a real time polymerase chain reaction (RT-PCR) assay of the blood that revealed positivity for Mycobacterium tuberculosis. The patient’s chest x-ray showed a reticular pattern in the middle and lower part of both lungs, and high resolution computerized tomography revealed multiple micronodular opacities that were compatible with his previous diagnosis of silicosis (Figure 1a, 1b, 1c). A bron-
choscopic investigation was planned to obtain a lung tissue sample to confirm or rule out tuberculosis, but the patient refused all interventions. Then, he was given empirical antituberculosis treatment, but he decided leave the hospital against medical advice.

Five months later, the patient was re-admitted to our hospital with a history of fever, productive cough, headache, agitation and vomiting. On physical exam, he had ptosis of the right eyelid, neck stiffness, reduced cognitive function, and positive Kernig’s and Brudzinski’s signs. He had used his antituberculosis drugs for only twenty days after discharge. Magnetic resonance imaging of the brain showed multiple lesions indicative of tuberculomas (Figure 2). A lumbar puncture (LP) was performed, and 70 cells per mm$^3$ were counted with a lymphocyte predominance. Glucose level in the CSF was low (28 mg/dl) compared with the measurement of 113 mg/dl in the blood. The protein level in the CSF was 300 mg/dl. Gram stain and culture of the CSF were negative. An AFB stain of the CSF was negative, but RT-PCR showed positivity for *Mycobacterium tuberculosis*. Although there were lung cavitations on the second High Resolution Computed Tomography (HRCT) on his second admission, the sputum analysis for tuberculosis was still negative. Tuberculosis treatment was re-initiated, and both clinical and laboratory findings, including control LP, of the patient improved within ten days.

**Discussion**

Silicosis is a well-known fibrotic disease of the lungs caused by inhalation, retention and pulmonary reaction to crystalline silica. The clinical course of the disease is dependent on many factors, such as dust concentration, the exposure duration and the latency period. Silicosis has recently been described in denim sandblasters. In denim sandblasting, because of a combination of various factors, silicosis most commonly manifests as acute silicosis even in the cases with relatively short exposure duration [4-6]. Although silicosis mainly affects the lungs, it may have some direct or indirect extra-pulmonary effects [8-10].

In the acute forms of silicosis, the main problem is respiratory insufficiency; however, in the sub-acute and chronic forms of the disease, in addition to respiratory insufficiency, some other problems may affect the prognosis and survival of patients. Exposure to crystalline silica, with or without clinical disease, increases the risk of tuberculosis and other nonmalignant respiratory diseases and contributes to the development of renal or autoimmune respiratory diseases. In addition, crystalline silica has been designated as a known human carcinogen [11].

Although the prevalence of tuberculosis is decreasing in the general population, workers with silicosis are at risk for developing tuberculosis [12]. It is well documented that exposure to silica can lead to impaired cell-mediated immunity. Evidence from experimental studies suggests that silica impairs the metabolism and function of pulmonary macrophages and, with frequent exposure, causes macrophage apoptosis [13]. Alterations in lymphocyte subsets, i.e., a reduced number of T cells, and changes in serum immunoglobulin levels in patients with silicosis have been observed in workers with silicosis [14]. These findings are consistent
with observations that the incidence of tuberculosis is higher in dust-exposed workers, even in those without established silicosis, than in workers who are not exposed to dust.

Epidemiologic studies have confirmed that those with chronic silicosis have a 3-fold increased risk of developing tuberculosis (both pulmonary and extrapulmonary) compared with an aged-matched, silica-exposed group without silicosis [15]. The incidence of tuberculosis and non-tuberculous mycobacterial disease is highest in acute and accelerated silicosis [16].

A study among South African gold miners indicates that the silica dust that miners accumulate in their lungs during exposure is a lifelong risk for the development of pulmonary tuberculosis (PTB), even if silicosis is not present in the lungs. Furthermore, even after the exposure to dust ends, ex-miners continue to be at risk of developing silicosis, and the development of silicosis places them at even greater risk of developing PTB. Moreover, the data show that miners with very few silicotic nodules (negligible and slight degree of silicosis) have a significantly increased risk of developing PTB and that this degree of silicosis is seldom diagnosed radiologically.

This case was an accelerated silicosis case with a relatively short exposure and a latency period. In denim sandblasters, more than half of the workers had developed either acute or accelerated silicosis although they had very short periods of exposure, but possibly heavy exposure, to crystalline silica [6]. In the first cases, it is highly possible that they were misdiagnosed as having tuberculosis [4] because there are no previously reported silicosis cases among denim sandblasters and they were subjected to poor working conditions. To the best

Figure 2. Axial FLAIR brain images (A) shows periventricular hyperintensity and left frontoparietal hyperintensity due to vasogenic edema and isointense multiple tuberculomas. Note the widened occipital horns due to communicating hydrocephalus. Axial (B), coronal (C) and sagittal (D) gadolinium-enhanced T1-weighted MR images show multiple ring-enhancing tuberculoma lesions. The majority of these lesions are near the cortex or deep gray matter, with most being at gray matter-white matter junction.
of our knowledge, this is the first report of tuberculosis, both PTB and tuberculosis meningitis, among denim sandblasters; however, we can speculate that more cases will be reported given the numbers of denim sandblasters with silica exposure.

With this report, we aimed to present the first case of TB development in a silicosis cases due to denim sandblasting and to draw attention to this issue. Silicosis continues to cause deaths due to respiratory insufficiency. Although there are few new cases of silicosis due to recent silica exposure in denim sandblasting, there is a huge pool of these cases due to past exposure; therefore, the exact extent of this disease burden is unknown, but new tuberculosis cases among these workers are expected.

Conflict of interest statement: The authors declare that they have no conflict of interest to the publication of this article.

References