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Pathological study of the prevalence of silicosis among coal miners in Iran: A case history

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HIGHLIGHTS

- To present a seven years period case history for pathologic diagnosis of silicosis among selected coal miners.
- In more than half of silicosis cases the condition develops after discontinuation of dust exposure.
- Showing the need to further improve the health surveillance criteria for dust-exposed workers in Iran.

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ABSTRACT

One of the most hazardous diseases that is commonly associated with the coal mining industry is *Silicosis* which caused by dust inhalation. This disease occurs as a result of prolonged breathing of dust containing silica (quartz). The generation of coal mine dust during underground and surface coal mining is the most significant source of coal dust exposure. Silica dust develops scar tissue inside the lungs which reduces the lungs ability to extract oxygen from the air. All miners working in underground and surface coal mines are at risk of being exposed to mine dust containing silica. In this study, cases with pathologic diagnosis of silicosis during seven years period between 2000 and 2007 were retrieved, from the pathologic file of Department of Pathology, Massih Daneshvary Hospital in Iran. Results of this case study showed the great effects of dust exposure and inhalation from the viewpoint of symptoms especially between the miners.

1. Introduction

The present coal mining procedure in Iran was established about 30 years ago and is still carried out by labor-intensive processes, involving extensive manual work during activities such as drilling, loading and transport. This type of coal mining is common in developing countries where resources are scarce and is accompanied by high respirable dust and quartz dust exposure. Nevertheless, the dust exposure problem has been existed in developed countries. A study of coal mine workers in South Africa shows higher exposure, with a mean cumulative respirable dust exposure of 67.5 mg years m^{-3}, whereas quartz exposure was not reported (Naidoo et al., 2005). A study among coal miners in the United Kingdom showed that 10% of the coalface workers had cumulative respirable dust exposure higher than 260 mg years m^{-3}, which was associated with reductions in FEV_1 (Forced Expiratory Volume in 1 s) of 250 ml or more (Rogan et al., 1973). Hurley and Soutar (1986) showed that, across all ages of exposed coal mine workers, FEV_1 dropped for increasing levels of dust exposure. Coal mining in developing countries includes strenuous work, which might be reflected in high ventilatory volumes and possibly a relatively higher rate of dust inhalation and deposition among these workers than at similar exposure levels in more mechanized mines (Mamuya et al., 2007).

One of the most perilous diseases that is commonly associated with the coal mining industry is *Silicosis* which caused by dust inhalation. This disease occurs as a result of prolonged breathing of dust containing silica (quartz). Quartz is the main substance in materials associated with mining and drilling activities. Breathing quartz laden dust will yield the same effects as breathing fine glass particles. Silica dust develops scar tissue inside the lungs which reduces the lungs ability to extract oxygen from the air. All miners working in underground and surface coal mines are at risk of being exposed to mine dust containing silica.

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The relationship between silicosis and crystalline silica found in coal, as a fatal and destructive disease associated with coal mining has been known for decades. For example, silicosis has been on the list of occupational diseases for which compensation is paid in Germany since 1929. The clinical toxicity of crystalline silica and the pathogenesis of silicosis have been summarized in detail (Silicosis and Silicate Disease Committee, 1988). Since 1986 an epidemiological study on the relation of silica dust exposure, silicosis and lung cancer was jointly made by Tongji Medical College (in China), US National Cancer Institute and the US National Institute for Occupational Safety and Health. Many valuable results have been obtained from dust-exposed workers, including etiology of silicosis, exposure (dose)-response relation and risk assessment (NIOSH, 1995). Thereafter, Silica-containing dust and silicosis have been attracting more and more attention with numerous researches worldwide (Rice and Stayner, 1995; Chen et al., 2001). As a more acceptable research, Yang et al. (2006) established a natural course on dust-exposed workers and their affection to the silicosis. However, few studies have been conducted concerning the criteria of the period and content of medical examination of health surveillance for dust-exposed workers (Cocco et al., 2001). Particularly, up to the present time, little studies have been conducted concerning the pathology and diagnosis method for dust-exposed workers. The overall objective of our research was to describe more prevalent symptoms of preliminary probable patients affected to silicosis among coal miners exposed to respirable coal dust in Iran. Specifically, the aims included describing the outcomes of silicosis and radiologic features of the disease.

The rest of this paper is organized as follows. In the second section, the silicosis, its properties and the pathological mechanisms are thoroughly described. In the third section, relation between coal mine dust and affection to the silicosis is discussed. Then, in forth section, a case study is performed during a 7 years period between 2000 and 2007, in Department of Pathology, Massih Daneshvary Hospital in Iran and results of the application are presented. Eventually, fifth section concludes the paper.

The knowledge acquired through research has been most valuable in determining the cause and pathogenesis of the most important occupational disease associated with coal mining, silicosis and can lead to some conducting results for correct prevention of prevalence to this disease.

2. Silicosis

Silicosis is a potentially fatal, irreversible, fibrotic pulmonary disease that may develop subsequent to the inhalation of large amounts of silica dust over time. In most circumstances, silicosis only develops subsequent to substantial occupational exposures. The disease has a long latency period and may clinically present as an acute, accelerated, or chronic disease. The pathophysiology of chronic silicosis involves chronic inflammation arising as a result of the accumulation of various inflammatory mediators and fibrogenic factors. Under the influence of these factors, pulmonary silicoproteinosis develops as eosinophilic proteinaceous material accumulates in the pulmonary alveolar spaces. The rate of disease progression appears to depend upon the rate of silica deposition in the lungs, as well as the total amount of crystalline silica that is actually retained in the lung. In some cases, silicosis may be associated with the concomitant development of other diseases, including tuberculosis, cancer, or autoimmune disease. Currently, no cure or effective treatment is available for silicosis (Valiante et al., 2004). Due to the association between occupational exposure to silica and the subsequent development of silicosis, a variety of international societies have initiated strict regulations aimed at preventing the development of silicosis in certain workers. These regulations generally emphasize adequate ventilation on job sites and limiting the amount of time workers may spend in potentially exposing environments (Muetterties et al., 2003).

2.1. Chemical properties of silica

Silica refers to the chemical compound SiO_2 (silicon dioxide) that occurs in two specific and distinct forms: amorphous and crystalline. The word “crystalline” implies that the silicon and oxygen atoms are oriented and related to each other in a fixed pattern as opposed to the random fashion that predominates in the amorphous form of silica. Crystalline silica naturally exists in a polymerized tetrahedral framework producing several polymorphs. These polymorphs are a function of the temperature and pressure: alpha quartz (or quartz), the most common polymorph found on the earth’s surface, is stable over most temperatures and pressures found in the earth’s crust. In contrast, beta quartz is stable at high temperatures, whereas tridymite and cristobalite are stable only at high temperatures and low pressures. Other silica polymorphs include coesite and stishovite. These may be encountered at a wide range of temperatures but only in a high-pressure environment and therefore they may be created during a variety of industrial processes including ceramic manufacturing, foundry processes, and any other industrial operation wherein quartz may become heated to high temperatures at elevated pressures. The most common crystalline forms of silica involved in workplace exposures include quartz, tridymite, and cristobalite. Silica may also occur naturally and at varying concentrations in rocks such as sandstone (67% silica) and granite (25–40% silica) (NIOSH, 2003).

Silicates are structures composed of silicon dioxide bound to cations such as magnesium, aluminum, or iron. Examples of silicates include mica, soapstone, talc tremolite, Portland Cement, and others. Opal, diatomaceous earth (tripolite), silica-rich fiberglass, fume silica, mineral wool, and silica glass (vitreous silica) are common amorphous forms of silica (Castranova et al., 1996).

Dusts composed of amorphous silica, with the exception of fiberglass, are not generally considered to be harmful to humans (Mossman and Churg, 1998).

Quartz, cristobalite, and some forms of tridymite are inherently piezoelectric. Piezoelectricity is a property that produces opposite electric charges on opposite sides of the physical structure when pressure is applied directly to the crystal. This phenomenon occurs in crystalline silica because the chemical structure does not have a center, reflecting an inversion symmetry. In addition, the opposite sides of these crystals have dissimilar surfaces and carry opposite electrical charges. It is theorized that these piezoelectric characteristics may play a role in the pathophysiology of silica-related illness by the generation of oxygen free radicals produced on the cleaved surfaces of silica molecules and as a result of silica-damaged alveolar macrophages (Williamson et al., 2001; Castranova, 2004). Silanol (SiOH) groups present on the surface of silica particles are capable of forming hydrogen bonds with oxygen and nitrogen groups found in biologic cell membranes, which then may lead to a loss of membrane structure, lysosomal leakage, and tissue damage. These processes may all contribute to the development of lung scarring (Castranova, 2004). Experimental data suggest that there is a distinct fibrogenic order of potency for these materials as follows: tridymite _ cristobalite _ quartz (Wiessner et al., 1988; Zaidi et al., 1956; King et al., 1953).

2.2. Occupational exposure to silica

Silicosis is caused by inhalation of crystalline silica, mostly in occupational settings. It is most common among workers in underdeveloped countries. However, silicosis occurs frequently even