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Mini Review

Silicosis: Combating a No Cure but Preventable Occupational Disease

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Abstract

Silicosis is a preventable lung disease resulting from the chronic occupational exposure to free crystalline silicon dioxide or silica. Silica is major component of quartz and has been defined as human carcinogen by US government. This occupational exposure to respirable crystalline silica dust particles occurs in many industries including construction industry. Silicosis leads to impairment of lung function because of fibrosis of the lungs. Even though, numerous efforts have been made to prevent silicosis, numerous deaths have occurred due to long term exposure to silica dust in construction sites. There are no proven treatments yet to cure silicosis and medicines are mainly focused in curing symptoms. Few innovative treatment methods have not yielded sustained positive results. Proper diagnosis with detailed work history is vital in responding to silicosis. Chest radiography and high resolution CT has been found effective in determining silicosis, which sometimes can be confused with other lung diseases. Controls such as technical controls, water spraying, local exhaust ventilation works effectively in reducing crystalline silica in dust, thereby reducing chances of silicosis. Other method such as substitution of silica containing material with aluminum oxides, magnesium carbonate helps in reducing the exposure. Dust suppressant, which acts through wetting and encapsulation have been found with varying degree of success in construction sites. Personal protective equipment, proper housekeeping and training are effective in reducing the exposure to silica dust. Effective regulation at every level of governance is also very important in combating silicosis.

Keywords: *Crystalline Silica; Carcinogen; Construction Industry; Chest Radiography; High Resolution CT; Technical Controls; Substitution; Dust Suppressant; Personal Protective Equipment*

Introduction

Silicosis is a type of disabling, dust-related lung disease caused by inhalation of free crystalline silica [1]. The disease is mainly characterized by inflammation of respiratory tissues that leads to pulmonary fibrosis and hardening of lungs caused by accumulation of excess fibrous connective tissue, thereby reducing the ability to breathe efficiently [2]. One of the major factors responsible for development of silicosis is dust containing respirable silica and the percentage of respirable silica in total dust [3]. Crystalline silica is a basic component of quartz, which is the second most common mineral in the earth's crust. Some of the major occupational activities that can expose workers or employees to airborne silica are rock drilling, stone cutting, sandblasting, demolition and hammering procedure, chipping, and sweeping concrete or masonry [4]. While these sources have been known to cause silicosis for a very long time, workers are still suffering, and in many cases, are dying from this non-curable yet 100% preventable disease. Even though no curative treatment are currently available, implementation of comprehensive management strategies against silica hazards help to improve overall health standard of the workers and eliminate fatalities. Despite the fact that total mortality from silicosis has decreased significantly in recent years, deaths among young workers are still high [5] (Table 1). Due to huge repercussions in socioeconomic and health status of community, improved efforts to limit silica exposure by employers and employees should be enforced.

Number and rate* of silicosis deaths, by selected characteristics and year — United States, 2001 - 2010									
Characteristic	Age group (yrs)						Overall		
	15 - 44			≥45					
	No.	Rate	(95% CI)	No.	Rate	(95% CI)	No.	Rate	(95% CI)
Total	28	0.01	(0.01 - 0.01)	1,409	0.58	(0.55 - 0.61)	1,437	0.59	(0.56 - 0.62)
Sex									
Male	23	0.02	(0.01 - 0.03)	1,347	1.37	(1.30 - 1.44)	1,370	1.39	(1.32 - 1.46)
Female	5	0	—	62	0.04	(0.03 - 0.05)	67	0.05	(0.04 - 0.06)
Race									
White	22	0.01	(0.01 - 0.02)	1,214	0.57	(0.54 - 0.60)	1,236	0.59	(0.56 - 0.62)
Black	5	0.01	(0.01 - 0.05)	181	0.85	(0.72 - 0.98)	186	0.87	(0.74 - 1.00)
Other	1	0.01	(0.00 - 0.06)	14	0.15	(0.08 - 0.25)	15	0.16	(0.09 - 0.26)
Year									
2001	1	0	—	163	0.74	(0.63 - 0.85)	164	0.74	(0.63 - 0.85)
2002	5	0.02	(0.01 - 0.05)	143	0.64	(0.54 - 0.74)	148	0.66	(0.55 - 0.77)
2003	6	0.02	(0.01 - 0.07)	173	0.76	(0.65 - 0.87)	179	0.78	(0.67 - 0.89)
2004	3	0.01	(0.00 - 0.03)	163	0.7	(0.59 - 0.81)	166	0.71	(0.60 - 0.82)
2005	2	0.01	(0.00 - 0.04)	159	0.67	(0.57 - 0.77)	161	0.68	(0.57 - 0.79)
2006	6	0.02	(0.01 - 0.07)	120	0.49	(0.40 - 0.58)	126	0.52	(0.43 - 0.61)
2007	1	0	—	122	0.49	(0.40 - 0.58)	123	0.5	(0.41 - 0.59)
2008	2	0.01	(0.00 - 0.04)	146	0.58	(0.49 - 0.67)	148	0.58	(0.49 - 0.67)
2009	1	0	—	120	0.47	(0.39 - 0.55)	121	0.48	(0.39 - 0.57)
2010	1	0.01	(0.00 - 0.06)	100	0.38	(0.30 - 0.46)	101	0.39	(0.31 - 0.47)
p-value†	—§	—§		0.012	0.002		0.01	0.002	

Table 1: *Number of silicosis deaths by age group (Source: CDC).*

The main aim of this mini-review is to provide an overview on the impact of silicosis with focus on management, prevention, and control strategies. The overall objective will be achieved by addressing the following questions:

- Historical events reigniting silicosis attention, current prevalence and exposure limits.
- Brief description on major types of silicosis, and its signs and symptoms.
- Identifying steps for silicosis management and their effectiveness including diagnosis.

Historical Events

One of the consequential industrial disasters of silica hazard that caught eye of the public happened in Hawk's Nest, West Virginia in 1930s. About 2,500 workers were involved in drilling through a tunnel for power station containing high concentration of silica with negligible protection from dust [6]. Due to likelihood of profitable production of ferrosilicon, a key component in manufacturing steel, the drilling and blasting was expanded without any prevention or mitigating procedure in place. Ultimately, 764 died from acute silicosis and additional 1,500 workers developed the disease [7]. Even though congressional hearings were critical of the mining operations, the disease faded into oblivion and only few of the workers were compensated. With sandblasting jobs booming in oil rigs and refineries in 1970s, Steven Weisenfeld, a physician from West Texas, diagnosed silicosis in Mexican-American workers, and held oil companies responsible for the preventable disease. He was swiftly booted out from medical society but collaborated with his friend to unravel sili-

cosis epidemic among sandblasters [8]. During 2003 - 2005, "the Mississippi cases" brought national attention when the judge ruled that thousands of silica claims were fabricated for money and dismissed the plaintiffs [9]. After these major national events, interests have reignited health hazards imposed by silica exposure.

Past trends and current scenarios

According to CDC, almost 2 million workers are exposed to respirable crystalline silica in their occupations, which mainly includes construction, sandblasting and mining. A surveillance report by Attfield., *et al.* (2009) [10] found that deaths due to silicosis in construction industry has decreased significantly across all age groups (Figure 1). According to the report, 12% of the total deaths from silicosis during 1985 - 1999 occurred in the construction industry. From 1999 to 2013, number of deaths due to silicosis declined significantly by 40%. However, the number of deaths increased from 88 to 111 during 2011-2013 (Table 1). During the period of 1999 - 2013, a total of 2,065 people's death certificate listed silicosis as underlying or contributing cause of death with more than 50% of them listing it as the underlying cause of deaths [11].

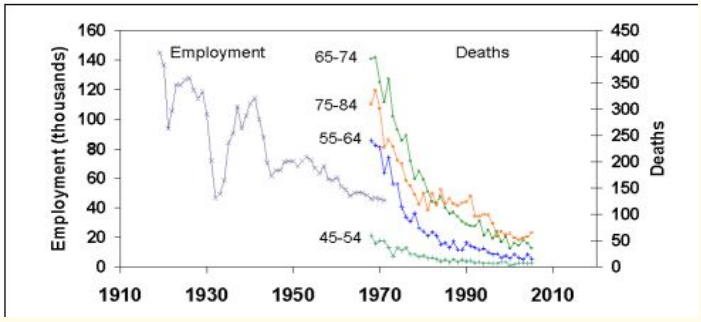

Figure 1: *Silicosis deaths by Age (Adopted from Attfield., et al. 2009).*

A surveillance program in 5 states including Michigan found 9 cases of silicosis among sandblasting workers in dental laboratories (CDC). In another surveillance program (1987 -1996) in Michigan, 77% of deaths that mentioned silicosis were confirmed to be silicosis-related death with the ratio of living to deceased as 6.44 [12]. A study conducted in 1999 - 2000 investigating exposure of silica to various construction industries in USA found that workers are overexposed relative to current exposure limit. The study clearly depicted the need to reduce exposures by applying various control methods [13].

Exposure Limits

The current Occupational Safety and Health Administration (OSHA) permissible exposure limit (PEL) for dust containing respirable silica for the construction as well as general industry is measured by millions of particles per cubic foot (mppcf) and is determined by equation below:

- PEL = 250 mppcf / % silica +5 8-hr time weighted average (TWA).

The National Institute for Occupational Safety and Health [3] recommended exposure limit (REL) for respirable crystalline silica is 0.05 mg/m^3 (50 µg/m^3) as a TWA up to 10 hours/day during 40-hr workday.

Types of Silicosis

Adverse pulmonary responses, such as acute silicosis, accelerated silicosis, chronic silicosis and conglomerate silicosis are the major types of silicosis resulting from exposure to crystalline silica [14]. For the objective of this review, the three major silicosis types: acute, accelerated and chronic silicosis will be briefly discussed with major signs and symptoms, and the associated physiological changes.

Acute Silicosis

Acute silicosis usually occurs when workers are exposed to high concentrations of freshly fractured silica in relatively short period. The symptoms may develop within a few weeks to 4 - 5 years after the exposure. Patients suffer from labored breathing, fatigue, cough, weight loss, decreased pulmonary function, and compromised gas exchange [15]. The major morphological feature in this type of silicosis is pulmonary edema, interstitial inflammation and accumulation of proteinaceous fluid [16].

Acute silicosis patients may also develop cyanosis and respiratory failure that is often complicated by mycobacterial infections. Large number of infectious diseases can be attributed to soil borne to some extent. However, the risk of transmission of disease is quite low even if theoretically possible.

Accelerated Silicosis

Accelerated silicosis is commonly associated with exposure to high concentrations of crystalline silica and normally develops within 5 - 10 years after the initial exposure [3]. While its symptoms are similar to acute silicosis in many ways, patients with accelerated silicosis develop fibrotic granulomas containing collagen, reticulin, and a large number of silica particles [15]. The major symptoms include inflamed and scarred lungs, shortness of breath, fever, bluish skin, fatigue, and rapid and shallow breathing. These symptoms are also seen in chronic silicosis (discussed in next section); however, the disease progression is much faster in accelerated silicosis.

Chronic Silicosis

Chronic silicosis develops due to prolonged exposure to crystalline silica at relatively low concentration and usually occurs after 10 or more years of initial exposure [3]. Silica dust promotes the formation of fibrotic nodules exhibiting typical histologic appearance of concentric arrangements of collagen fibers [16]. This type generally features breathlessness and might resemble chronic obstructive pulmonary disease (COPD). Two types of chronic silicosis are generally described in literature.

- **Simple Silicosis:** It can be asymptomatic or present with exertional dyspnea and cough with sputum production. Differentiating simple silicosis with chronic bronchitis and emphysema in a smoker is sometimes difficult and confusing.
- **Complicated Silicosis:** Dyspnea and cough are accompanied with symptoms of malaise and weight loss in complicates silicosis.

Workers or patients with silicosis are also at risk for other disorders, such as tuberculosis, lung cancer, progressive systemic sclerosis, rheumatoid complications, and nocardiosis [1].

Management

No proven treatment for silicosis are currently available and existing treatments are basically focused on curing symptoms. In addition to proper diagnostic approach, the major focus has therefore been on prevention and control of silicosis, adequate training, and raising awareness among employees and workers [1].

Diagnosis

Chest radiography is the primary method used to diagnose silicosis (Figure 2). As degree of silicosis increases, efficiency in radiography improves. High resolution CT scan has been found to be more sensitive in detecting features like nodular changes, progressive massive fibrosis (PMF) and emphysema [17]. However, patients with moderate or severe degree of silicosis already identified through histology (Figure 3) may not be diagnosed by radiography [18]. The major component in silicosis diagnosis relies on the detailed description of past and current jobs where workers might have been exposed to silica dusts. Great care should be given while contemplating on radiological features as they might be confused with tuberculosis, fungal infections, carcinomatosis and other interstitial lung diseases [1]. Sometimes, without thorough examination on occupational history, silicosis diagnosis might be missed in absence of nodular lesions. Due to long latency period of silicosis (esp. in chronic silicosis), careful review of all jobs held, job practices, and exposure estimates along with potential environmental and domestic exposures should be considered.

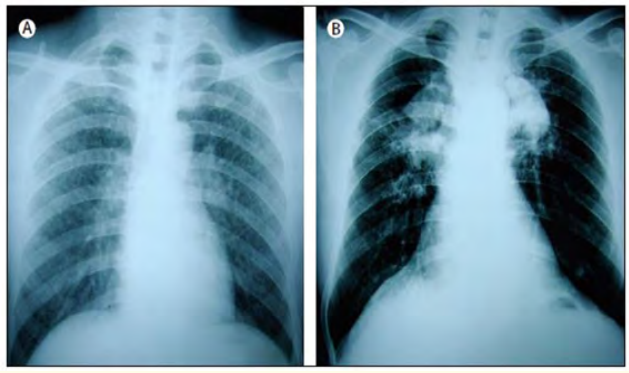

Figure 2: *Patient with Silicosis. A-Simple Nodular Silcosis, B- Progressive massive fibrosis (Adopted from Leung., et al. 2012).*

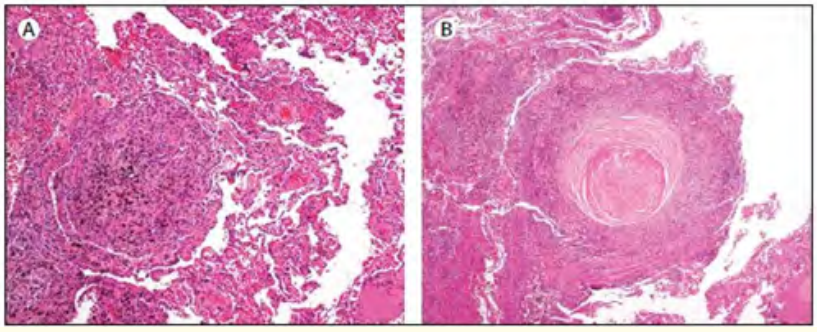

Figure 3: *Histological sections of lung with silicotic lesions (Adopted from Leung., et al. 2012).*

Treatments

Until now, no treatment has been proven effective in curing silicosis. One earlier study found that particles coated with aluminum didn't produce fibrosis in the lungs of rabbit [19]. A trial conducted few years later did not have any distinguished effect either on progression of the disease or on the symptoms [20]. Other coating compounds, such as aluminum citrate and herbal substance like tetrandrine had no significant and sustained benefits on silicosis. Further, curative treatments like lung lavage had few benefits of relieving some of the symptoms but objective parameters such as pulmonary functions were not achieved consistently [21]. Other treatments, such as corticosteroid and prednisolone have helped to improve lung function but were ineffective in the long run.

Silicosis treatments are generally focused on relieving the symptoms. For example, bronchodilators are useful for the patients with airflow obstructions [1]. Cough suppressants are used for relieving the symptoms. Additionally, antibiotics are provided as a necessary step to check chest infections. People with silicosis are prohibited from smoking, and encouraged to receive vaccines for influenza and pneumococcal in order to avoid further complications. As a result of having no effective treatment yet, prevention from silica exposure is the most useful way to avoid silicosis. Next sections will describe effective control strategies and discuss their effectiveness for silicosis prevention.

Controls

Employees in the construction industry are at high risk for exposure to respirable crystalline silica. Therefore, in addition to personal protective measures, preventive strategies like technical controls, substitution, dust suppressants, and good housekeeping play major roles in decreasing the risk of silicosis.

Technical controls

The two major technical controls are spraying and local exhaust ventilation, particularly applied to control dust originated from tools.

Spraying

Water spraying helps in controlling dust mainly created by grinding concrete, masonry, brick and block cutting, and using jackhammer for breaking concrete. The implementation of wet methods such as water spraying at construction sites have shown to drastically reduce respirable crystalline silica. A review of various studies has found that silica dust was reduced by 86% in construction industry and 80% in manufacturing industry as the result of spraying [22]. Another study has shown that atomizing spray nozzles effectively reduces respirable dust in brick cutting operation as much as by 79% [23]. Enough consideration should also be given to components, such as water source, hose, flow, and frequent checking for clogging to make the system work efficiently.

Local Exhaust Ventilation (LEV)

LEV, also known as dust collection, uses ventilation system to carry dust filled air stream through a collection and isolation point. The major components of the system are: exhaust hood to collect dust from their source, a system of ducts to transport collected dust to collector, dust collector, and suction hose and filtration system for collecting dust. Proper consideration like systemic cleaning of vacuum, verification of effectiveness of particle exhaust, and filtration should be given for effective performance. A study conducted about the effectiveness of LEV on dust exposures during concrete cutting and grinding activities found that the exposure was reduced significantly by 80 - 95% [24]. Another study to gauge dust exposure control during concrete surface grinding found out overall mean exposure reduction to be about 92% [24]. Even though both studies stated effectiveness of LEV, personal respirable exposure of quartz remained high [24,25]. Therefore, it is advised to use minimum respiratory protection when LEV is used.

Substitution

Substitution is basically replacing a hazardous substance with another less hazardous substance. Crystalline silica, which is constituent of sandstone grinding wheels, can be replaced with corundum (aluminum oxide) and magnesium carbonate bricks instead of siliceous bricks [26]. Also, preventing silicosis in drywall finishers can be achieved through silica-free joint compounds. Silica sand can also be substituted with limestone in the production of concrete.

Dust Suppressants

Dust suppressants are mainly used during maintenance on construction sites. These dust suppressants mainly work by involving two techniques: wetting and encapsulation. Different types of dust suppressants include water (described earlier in spraying), acrylic polymers, asphalt, chloride compounds, lignin compounds, natural oil resins, organic resin, emulsions, and petroleum-based oils and waste products. However, petroleum-based oils and waste products are prohibited to use due to EPA regulations [27]. Acrylic polymers are plastic adhesives that work as a suppressant by chemically binding during curing, thereby creating a surface crust. Using acrylic polymers on unpaved roads gave varying degree of success [28]. Additionally, recycled asphalt from roads or roofing materials can be used to suppress dust when crushing rock. This method is environmental friendly as well because it reduces disposal or storage cost for the materials. Liquid asphalts have been banned to be used as a dust suppressant due to EPA regulation [29].

Calcium chloride and magnesium chloride are hygroscopic, and therefore work as dust suppressants by attracting moisture in the air. They help bind smaller particles into heavier ones, thereby helping to negate their airborne capability. Likewise, lignin compounds such as lignin sulfate chemically binds soil by reducing surface tension between clay in the soil and water. They are water soluble and effective in dry climates. Soybean oil is an example of natural oil resins suppressant that is used for road stabilization and a dust suppressant. When sprayed, oil makes dust heavier and makes them stick together like an adhesive through the process called agglomeration. However, they have varied success rate [30] as researchers have claimed inconsistency as major hurdle. Also, precipitation is major obstacle for their use as they leech away with water. Organic resins emulsions also bind and adhere to dust particles as they cure and create a surface crust. They are environmentally safe, noncorrosive, non-leaching, and waterproof.

Personal Prevention and Site Housekeeping

Site housekeeping and personal prevention techniques are also important to negate the respirable silica spread. Few housekeeping methods, such as avoiding resuspension of deposited dust by cleaning activities, prohibiting use of air jet to remove or eliminate dust, and workers standing upwind from dust emission sources goes a long way in keeping protected from silicosis. Personal prevention measures, including respiratory protection, wearing disposable or washable garments, taking shower, and leaving in clean clothes helps in reducing exposure from crystalline silica. Workers can also demand regular air monitoring of the sites, periodic medical examinations and training to handle equipment, and use of personal protective equipment (PPE) to safeguard against silicosis.

Conclusion

Silicosis is a preventable lung disease that has continued to cause deaths to construction workers. Despite its long-known history and well established etiology, this disease has not been handled properly throughout US history. Continued emergence of silicosis, specially among construction workers, has reignited debate on silicosis at a wider national level. As of yet, there is are no proven effective treatment of silicosis, which makes control of silica exposure at the source the primary method of silicosis prevention. Source control can be achieved through various methods from technical controls, substitution, and suppressants to personal prevention and effective housekeeping. Research conducted to determine the efficacy of such methods has provided ample evidence supporting their effectiveness in reducing crystalline silica, and thereby preventing silicosis development in construction workers. To further reduce the susceptibility to silica exposure, there also should be effective regulation from federal to local levels to eliminate this non-curable but entirely preventable disease.

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