Mine Dust and Pulmonary Health

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Abbreviations: PM: Particulate Matter; CMDLD: Coal Mine Dust Lung Disease; DDF: Dust-Related Diffuse Fibrosis; PMF: Progressive Massive Fibrosis; HRCT: High-Resolution Computed Tomography; COPD: Chronic Obstructive Pulmonary Disease; PDS: Personal Dust Sampler; ABGs: Arterial Blood Gases.

Introduction

Mining activities mainly deal with the excavation of raw material from earth’s crust. The sources of particulate matter (PM) emission in opencast coal mines include blasting and drilling operations, coal handling plant operation, loading and transport of overburden and coal by shovel-dumper combination, crushing, conveying and handling of overburden by draglines, and vehicular emission from unpaved roads [1]. Dust is termed as particulate matter (PM) and are of two types as respirable (≤ PM10) and non-respirable (>PM10). Mining dust is considered as the most dangerous health hazards for miners and nearby people. Different factors such as distance from the source, wind speed and direction affect the transportation of dust to the nearby area. Dust generated through mining activities consists of mostly coarse particles and particles larger than PM10. The dust generated from vehicular exhausts and mobile equipment are fine particles and only account for about 5 per cent of the total particles emitted [2]. A number of defense mechanisms working in the human respiratory system protect people from the harmfulness of particulate matter (PM). The sticky wall of the breathing airways traps the PM. Then the PM with the mucus is removed by swallowing or coughing. Finer particles (≤PM2.5) reach deeper into the lungs and are most dangerous and severely affect them.

Dust from the mining activity contains trace elements such as Ca, Al, Mg, Ti, Mn, Fe, Zn, Ni, Cr, Sn, V, Sb, Cu, Co, Cu, Pb, Cd, etc. The toxic trace elements and minerals present in dust are major threat to human health [3]. Microorganisms such as Acinetobacter schindleri, Aeromonas cavernicola, Alternaria alternata, Aspergillus penicillioides, Cladosporium cladosporioides, and Penicillium brevicompactum etc. may be present in coal dust which may affect the miner's health through inhalation [4]. The health risk from dust hazards depend on size, concentration, mineralogical composition and duration of exposure.

Miner’s lung diseases are the most important health issues from historic time. Miners are exposed to a number of hazardous chemicals, silica (quartz), radon and diesel exhaust during their exposure and are known carcinogens for lungs related diseases. The abundance of silica is about 27.7% and the second most common element found in the earth’s crust. The three most common crystalline forms of free silica encountered are quartz, tridymite, and cristobalite. The predominant form is quartz. Freshly-fractured siliceous rocks have been found to be more fibrogenic than aged particles [5].

The development of pneumoconiosis in a worker mainly depends on the concentration of dust in the breathing zone, the shape, size and buoyancy of the particles, and physio-chemical reactivity. The amount of dust retained in the lungs is determined by dust concentration in surrounding air, duration of exposure and effectiveness of clearance mechanisms of the body. The most dangerous particles range from 1 to 5 μm in diameter because they may reach the terminal small airways and air sacs and settle in their linings. Smaller particles tend to cause acute lung injury. Larger particles
resist dissolution and so may persist within the lung parenchyma for years.

Pulmonary occupational diseases are the result of inhalation of dust and it may take years to become noticeable. Pulmonary diseases or coal mine dust lung disease (CMDLD) such as dust-related diffuse fibrosis (DDF), coal worker’s pneumoconiosis, mixed dust pneumoconiosis, silicosis, chronic airway diseases including emphysema and chronic bronchitis are associated with exposure of dust. Recent investigation shows that coal miners may show excess lung cancer mortality as there is no specific treatment [6].

Generally pneumoconiosis takes about 10 years after initial exposure to develop and the exposure to quartz enhances the effect. Other type of pneumoconiosis are coal worker’ pneumoconiosis (coal dust), Asbestosis (asbestos), Berylliosis (beryllium), Kaolin pneumoconiosis (china clay), Siderosis (iron oxide), Silicosis (silica dust), Talc pneumoconiosis (talc dust) and Metallic pneumoconiosis( barium, cobalt, tin, tungsten dust). Some more types of pneumoconiosis include graphite pneumoconiosis, carbon black pneumoconiosis, mica pneumoconiosis, aluminosis etc. Pneumoconiosis is broadly classified as non-fibrogenic pneumoconiosis and fibrogenic pneumoconiosis or true pneumoconiosis.

Classic coal worker’s pneumoconiosis and silicosis are most familiar to coal mine workers. Small (<1cm) rounded opacities formed in upper lung zone in milder forms in the case of Classic coal worker’s pneumoconiosis. Progressive massive fibrosis (PMF) is characterized by coalescence of small opacities into large (>1cm) opacities. Rheumatoid pneumoconiosis (Caplan syndrome) may also affect the coal miners with rheumatoid arthritis. Rapidly progressive pneumoconiosis with progressive massive fibrosis (complicated coal workers’ pneumoconiosis) is being observed in US coal miners [7].

Silicosis is generally divided into three types: acute silicosis, chronic silicosis and accelerated silicosis. The cumulative dose of silica (respirable dust concentration x crystalline silica content x exposure duration) is probably the most important factor for the development of silicosis. Generally, chronic silicosis develops between 15 to 45 years after first exposure. In some instances even, single heavy dose/ exposures to very high concentration of silica for a short span of time may develop silicosis.

Diagnosis includes taking data on exposure history of the worker and radiological or pathological findings. The exposure history is important especially if the worker is exposed to face in the underground mine or near the drilling in surface mining. Silica crystals in lung tissue can be observed under polarized light microscopy. Lung tissue changes in progressive silicosis are often detected by chest x-ray before they can cause any symptoms. Pulmonary function tests are used to evaluate lung function and confirm the presence of lung disorders. These may include lung volume measurement and spirometry to detect any abnormalities of normal lung expansion or obstruction of air flow, peak flow measurement to detect narrowing of the airways, and diffusing capacity to assess the efficiency of gas absorption into the blood. The efficiency of gas exchange in the lungs can be assessed by Arterial blood gases (ABGs) techniques, by measuring oxygen and carbon dioxide (CO2) in arterial blood. CT scanning and high-resolution computed tomography (HRCT) has been the major diagnostic techniques used. Chronic obstructive pulmonary disease (COPD) is a disease categorized by breathing restriction and is not fully treatable. Lung transplantation may be performed with very advanced stage of lung diseases, but it is quite difficult Silicosis mortality declined between 1968 and 2002, but new cases of silicosis continue to occur, even in young workers. High risk of silicosis also reported in different part of the globe. The proportions of silicosis for black gold miners increased from 0.03 to 0.32 and from 0.18 to 0.22 for white gold miners in South Africa [8]. WHO (2015) [9] fact sheet revealed that more than 3 million people died of COPD in 2012, and at present 65 million people are affected by moderate to severe COPD. During 2000-2005, in United States about 718,077 numbers of people (aging> 25 years) died due to COPD (Min et al., 2015) [4]. Silicosis occurs everywhere but is especially prevalent in low- and middle-income countries. China is the country with the largest number of silicosis patients, with more than 500,000 cases in records from 1949 to 2010. During 1991 to 2010, more than 6,000 new cases and more than 24,000 deaths occurred annually [10,8].

To prevent the workers from dust related pulmonary diseases medical methods such as periodic health examinations, pre placement health examinations, health education, working environment supervision and periodic personal dust monitoring can be performed. Notification, maintenance and analysis of records, and counseling are also steps towards prevention occupational pulmonary diseases. NIOSH (2002) [11] recommended respirable coal mine dust exposures limit as 1 mg/m³, and respirable crystalline silica level as 0.05 mg/m³. [TWA -10 hours, time weighted average concentrations/day and 40-hours work week]. The dust level in the work environment should be reduced to meet the prescribed limit. It is also
important to frequently monitoring of worker exposure of dust by personal dust sampler (PDS) in the mine. The personal dust monitoring allows measuring the concentration of respirable dust in their breathing zone and the feedback will be helpful to take control measures if that exceeds the legally permissible limits. The risk of pulmonary diseases increases with the increase in the exposure time and years of work [12]. In addition to recommending a reduction in the exposure limit for coal mine dust, NIOSH also recommended a change in the exposure limit for crystalline silica dust. It recommended increase in the frequency of medical examinations of workers and extending it to surface coal mine workers. NIOSH has been investigating the health implications of possible excessive crystalline silica exposure arising from the cutting of the rock adjacent to the coal seams [2]. This analysis used the presence of r-type pneumoconiosis. There is the clear need to minimize exposure to silica dust, especially for those jobs involving drilling or cutting sandstone and other siliceous rock. Job rotation in the working area will help in the reduction of total exposure time for a particular worker. There is a need for reduction in the silica level in the working area by taking engineering measures such as proper design of plant, ventilation, dust isolation through enclosure and isolation, and provision of dust masks for the workers. Efforts should be undertaken to reduce dust generation at source, prevent it from getting airborne besides reduction of duration exposure of workers to higher dust concentration can reduce the health impacts and pulmonary diseases in miners.

References


2. NSW Mining Council (2000) Mine Dust and You.


