Prevalence of Silicosis and Respiratory Impairment among the Miners of Rajasthan State – A Pilot Study Report

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The Greeks first reported prevalence of silicosis among miners exposed to hazardous silica dust in different mines. In the present study 2203 miners working in different mines of Rajasthan state in Western India underwent health status evaluation on anterior posterior chest X-ray as per ILO guidelines and pulmonary function test. Chest X-ray radiograph of 154 miners revealed that they were suffering from silicosis, silico-tuberculosis and pulmonary tuberculosis. Results of pulmonary function test of 379 miners showed that they were suffering from restrictive, obstructive or combined impairment. The rest of the miners had normal chest radiograph X-ray and pulmonary function test.

Key words: Silicosis, pulmonary impairment, silica, and pulmonary function test.

The potential dust hazard of silica particles on workers dates back to thousands of years. Hippocrates and Pliny mentioned silica's ability to cause disease among workers[10]. Lanza AJ in 1938 rferred the work of Hippocrates and Pliny in his book silicosis and asbestosis. Agricola in 1556 described in De Re Metallica that men who worked in local mines of Carpathian Mountains died of pulmonary disease at a very young age and their women had to marry as many as seven times[2]. The ancient Greeks first reported silicosis. Ramazzini in 1713 discussed an observation by Diemerbroeck, stating the extremely graphic description of the effects of exposure of silica on stonecutters[13]. While dissecting the cadavers of stonecutters who died of asthma, Diemerbroeck found that the lungs of stonecutters were filled with sand[13].

The prevalence of silicosis as an illness peaked in the last half of the 19th century and in the early part of the 20th century during the time of Industrial revolution[14]. Johnstone in 1796 reported the first study of silicosis on needle pointer workers at Redditch, England[8]. Knights reported the cause of silica-induced disease in Sheffield in early 1800s. Thackrah, who acknowledged the work of Knights described the danger of sandstone dust in mining leading to the death of the mineworkers at an early age[17].

Alice Hamilton in 1917 described the stone cutters of Barre Vermont as dreadful men who were carving tombstone for others and preparing themselves for their own graves. The death rate from tuberculosis in Vermont area increased to 60.6% per 1000 due to dust exposure after the introduction of air hammer to the stonecutters of granite mines in Barre[7].

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In 1918 English workers received compensation for disability as a result of Silicosis[1]. The National Institute for Occupational Safety and Health reported 13,744 deaths due to Silicosis from 1968 to 1990[14].

Silica is a natural mineral composed of silicon dioxide, which occurs-either in crystalline or amorphous form-making upto 21% of the earth's crust. Davis and Tepordei in 1983 estimated the world production of silica as 182 million tons[11]. According to the Occupational Safety and Health Administration, permissible exposure limit (PEL) for Silica is 100µg/m^3 for an 8 hours exposure[14]. Hazardous respirable size silica causing silicosis is about 0.5 to 5.0µm in diameter[4,6,14]. The silica particle size of 0.5 to 5.0µm in diameter can enter the alveoli of human lungs. As per the International Labour Office (ILO) description, Silicosis is an occupational lung disease attributed to the inhalation of Silicon dioxide in a crystalline form usually as quartz, cristobalite and tridymite6. Silicosis is classified under the following types:

Chronic or classical silicosis : Occurs due to moderate exposure of respirable dust with less than 30% quartz for a period over 20 to 45 years, causing progressive disease of pulmonary parenchyma. The lesions are nodular and more prominent in the upper lobes. Silica particles induce the formation of fibrotic nodules with typical appearance. The nodules are 5mm or less in diameter and impairment of pulmonary function is uncommon. Chronic silicosis may progress to massive fibrosis even after the exposure to silica containing dust has ceased. The silica particle size less than 1.0µm in diameter are most fibrogenic and can penetrate the interstitium initiating the fibrogenic process[6,11,14,18]. The pathophysiology of silicosis involves deposition of particles into alveoli where they cannot be cleared. Ingestion of these particles by alveolar macrophages initiates an inflammatory response, which stimulates fibroblasts to proliferate and produce collagen. The silica particles are enveloped by collagen leading to fibrosis and nodular lesions characteristic of the disease. Pollard, 2016.

Progressive massive fibrosis (PMF): It can be characterized by nodular opacities greater than 1cm on chest radiograph and it involves reduced arterial oxygen tension at rest or with exercise and marked restriction on spirometry or lung volume measurement. Weight loss and cavitation of large opacities may prompt concern for tuberculosis or other mycobacterial infection. PMF is composed of confluent silicotic nodules. There is a distortion of the bronchial tree leading to airway obstruction and productive cough[6,11,18].

Accelerated silicosis : It is caused due to intense exposure of 5 to 10 years duration. The deterioration in lung function is more rapid and many workers with accelerated disease may develop mycobacterial infection. The progression of radiographic abnormalities and functional impairment is very rapid [6,14].

Acute silicosis : It develops within few months to 2 years of massive silica exposure causing dyspnoea, weakness and weight loss. Rapid progression to severe hypoxaemic ventilatory failure is the usual course. Miners developed "Chalicosis pulmonum" after three months of employment in Nevada quartzite mill in 1900[14]. Death due to this disease occurred within a year among the miners. In 1929 Middleton reported that the packers of abrasive soap powder in London plant had a febrile illness associated with progressive respiratory failure after an exposure to overwhelming amount of free crystalline silica[14]. Chapman in 1932 reported, death of three silica abrasive packers only after 8 to 29 months of initial exposure[14.] Buechner in 1969 reported four sandblasters with acute silicosis and coined the term silicoproteinosis[14.]

The survival rate of British sandblaster suffering from acute silicosis was ten years in 1936[9]. In 1929 Sutherland and Bryson detected silicosis by X-ray in 25% of sandstone workers. Sutherland et. al, detected silicosis on 53% granite workers11. US Bureau of Mines and Public Health Service documented the occurrence of pulmonary disease in various workers exposed to silica in their case report. Outbreak of acute silicosis was reported among Ganley Bridge tunnelers working on pure quartz. This incident led to the formation of Walsh-Heaby legislation in US in 1937 for dust control standards[11]. In 2004 Rajnarayan R. Tiwari stated that subjects who were exposed to silica dust for ≥2 years in quartz crushing units have lower peak expiratory flow rate (PEFR) compared to those workers who were exposed for<2years duration. The low PEFR is due to the irritation of the upper respiratory tract mucosa among the exposed subjects[16.]

Hnizdo and Vallyathan (2003) suggested that chronic lower level of silica exposure may lead to the development of emphysema, chronic bronchitis that can lead to airflow obstruction.

The present health survey was conducted among the miners working in different mines of Rajasthan state in western India. Rajasthan is geographically the largest state having second highest mineral deposit in India. Three-fifth of 3,42,239 square kilometers of land in Rajasthan is desert[12]. The number of mines reporting to Indian Bureau of Mines (IBM) in the organized sector was 425 mines in 2003-04. The value of mineral produced from these mines in 2003-04 was Rs 27,596,556/-[3.]

As per the report of Mine Labour Protection Campaign (MLPC) there are 1324 mining leases of major minerals, 10851 leases of minor minerals and 19251 license quarries in Rajasthan[12.] The population of this state is 49.7 million out of which 2.5 million people are mine workers engaged in 64 kinds of metallic and non-metallic mineral extraction activities[12.] 97% of mining activities are in the unorganized sector. 37% of miners are women and 15% are children[12.] Recent study on steel foundy workers showed that thre is deterioration of respiratory status due to exposure of silica Paine *et al* (2017). The mortality rate among miners due to occupational diseases like silicosis and tuberculosis is around three persons per day[12.] The literacy level of mine workers is 0.8% and their average working hours is 8 to 10 hrs/day[18.] The purpose of this study was to estimate the percentage population of mine workers in the organized sector of Rajasthan suffering from silicosis and pulmonary impairment.

MATERIALS AND METHODS

The present study was undertaken by making a preliminary survey of various mines located in different regions of Rajasthan. The miners who participated in this health survey were working in phosphate mine, fluorite mine, lignite mine, limestone mine and gypsum mines. In this study posterior anterior chest X-ray radiographs and pulmonary function test (PFT) were carried out on 2203 miners working in the above mines of Rajasthan. Out of 2203 miners 1403(55.24%) miners were working in phosphate mines, 27(1.06%) were in fluorite mine, 265(10.43%) of them were working in lignite mine, 260(10.24%) were working in limestone mines and the remaining 248(9.76%) miners were working in gypsum mines.

The miners were briefed on the posterior anterior chest X-ray radiograph (CXR) and pulmonary function test (PFT). Radiographic chest X-ray of posterior anterior view was taken on each miner as per ILO guidelines on a 300 mA X-ray machine with 125KV. All CXRs were interpreted as per standard set of ILO classification of CXR for pneumoconiosis. In silicosis CXR depicts presence of 0/1 or 1/0 small regular (p, q, r) or irregular (s, t, u) opacities. In silicotuberculosis CXR shows presence of silicosis and pulmonary tuberculosis.

The PFT was done by using Cosmed Pony Graphic 4.0 Spirometer. The parameters measured by the Spirometer were FVC, FEV1, FEV1/FVC %, PEFR, FEF 200-1200, and FEF 25-75%. Parameters studied in this survey were FVC, FEV1, FEV1/FVC %. Subjects were told to take a deep forceful inspiration followed by a deep forceful expiration through the mouthpiece of the spirometer. Three readings were obtained for each miner and the best reading was taken for reporting and analysis of data. All PFT data were expressed in BTPS (body temperature and pressure saturated). The results were interpreted as normal (N) spirometry, obstructive impairment (OI), restrictive impairment (RI) and combined impairment (CI) that is having both restrictive and obstructive impairment on the basis of subject's performance in the PFT. As normal PFT values changes in relation to age, sex, body height and race of a subject, the predictive FVC and FEV1 were calculated by using the predictive equation of Kamat *et al.,* (1982)[9]. To determine OI the predictive FEV1/FVC % was calculated for each subject. If the predicted FEV1/FVC % value <75% the subject is considered having OI. For RI predictive FVC was determined from Kamat's equation. The observed FVC was divided by predictive FVC and multiplied by 100%. If the percentage value <80% than the person is having RI. If the subject is having the signs and symptoms of both OI & RI than he is classified under the category of CI. Figure 1 depicts the level of respirable dust generation in an open cast mine of Rajasthan state.

Figure 1 respirable dust generation during pneumatic drilling operation in Open cast mine

RESULTS

A) Chest X-ray

Table 1 depicts the chest X-ray radiographic result showing the prevalence of silicosis, silicotuberculosis, pulmonary tuberculosis and normal cases among the miners of Rajasthan. CXR was conducted on 2203 miners of different mines out of whom 110 (4.99%) of them were confirmed having silicosis, 17 (0.77%) had silicotuberculosis and 27 (1.23%) miners had pulmonary tuberculosis. 2054 (93.24%) miners had normal CXR.

Table 1: Chest X-ray radiographic result depicting prevalence of silicosis, silicotuberculosis and pulmonary tuberculosis among the miners of Rajasthan

Mine Name	Total No. of miners	Silicosis	Silicosis Tuberculosis	Pulmonary Tuberculosis	Normal
Phosphate Mine	1403	54 (3.85%)	16 (1.14%)	21 (1.49%)	1312 (93.53%)
Limestone Mine	260	20 (7.69%)	0 (0%)	0 (0%)	240 (92.30%)
Lignite Mine	265	11 (4.15%)	0 (0%)	2 (0.75%)	254 (95.91%)
Gypsum Mine	248	25 (10.08%)	1 (0.4%)	3 (1.2%)	222 (88.33%)
Fluorite Mine	27	0 (0%)	0 (0%)	1 (3.7%)	26 (96.3%)
Total	2203	110 (4.99%)	17 (0.77%)	27 (1.23%)	2054 (93.24%)

As per the table 1 silicosis was highest (10.08%) in gypsum mine and lowest (3.85%) in phosphate mine. The percentage of silicotuberculosis was highest (1.14%) in phosphate mine and lowest (0.4%) in gypsum mines. Fluorite mine had highest (3.7%) pulmonary tuberculosis whereas lignite mine had lowest (0.75%) of same disease. Silicosis and silicotuberculosis was nil among the miners working in fluorite mine. Silicotuberculosis was absent among the miners of limestone and lignite mines. Pulmonary tuberculosis was nil in limestone mine miners.

B) Pulmonary function test

Table 2 depicts the pulmonary function test on 2203 miners of Rajasthan. 1834 (82.80%) miners were having normal respiratory function. Among the remaining 379 miners, 290 (76.52%) were having restrictive impairment, 64 (16.89%) have obstructive impairment and the remaining 27 (7.12%) have combined impairment.

RI was more common than OI and CI. As per table 2 RI was 12.97% in phosphate mine, 12.3% in limestone mines, 17.35% in lignite mine, 9.67% in gypsum mines and 22.22% in fluorite mine respectively. OI was 2.56% in phosphate mine, 0.76% in limestone mines, 6.79% in lignite mine, and 3.21% in gypsum mine. CI was 1.21% in phosphate, 1.15% in limestone, 0.75% in lignite and 1.2% in gypsum mines. RI was highest (22.22%) in fluorite

Table 2: Prevalence of pulmonary impairment among the miners of Rajasthan

Mine Name	Total No. of miners	Silicosis	Silicosis Tuberculosis	Pulmonary Tuberculosis	Normal
Phosphate Mine	1403	182 (12.97%)	36 (2.56%)	17 (1.21%)	1168 (83.26%)
Limestone Mine	260	32 (12.3%)	2 (0.76%)	3 (1.15%)	223 (85.79%)
Lignite Mine	265	46 (17.35%)	18 (6.79%)	2 (0.75%)	199 (75.11%)
Gypsum Mine	248	24 (9.67%)	8 (3.21%)	3 (1.2%)	213 (85.92%)
Fluorite Mine	27	6 (22.22%)	0 0	0 0	21 (77.78%)
Total	2203	290 (13.16%)	64 (2.91%)	27 (1.23%)	1824 (82.80%)

mine and lowest (9.67%) in gypsum mines. OI was highest (6.79%) in lignite mine and lowest (0.76%) in limestone mines. CI was highest (1.21%) in phosphate mine and lowest (0.75%) in lignite mine. Obstructive and combined impairment was nil in fluorite mine.

DISCUSSION

In medical literature many studies were available which describe the prevalence of silicosis and pulmonary impairment and changes occurring in lung function of workers exposed to dust in industries. But studies on silicosis and pulmonary impairment of mine workers in the desert region of western India are very few.

The present study depicts the percentage of workers affected by silicosis and pulmonary impairment in the organized mining sector in different mines of Rajasthan state of western India. The cause of silicosis and pulmonary impairment in workers might be due to uncontrolled exposure to dust during mining operation without using personal protective device and sprinkling adequate amount of water for dust suppression as per the directives of Director General of Mine Safety (DGMS). The deprivation of mine workers in this state is obvious from various social indictors like occupational disease, hazardous and unsafe working conditions, violation of labour and mining laws etc.

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