ORIGINAL ARTICLE

RELATIONSHIP BETWEEN SILICA DUST EXPOSURE AND CHRONIC OBSTRUCTIVE PULMONARY DISEASE IN WORKERS OF DUST GENERATING INDUSTRIES OF DISTRICT PESHAWAR

Bushra Iftikhar, Muhammad Hussain Khan, Hamid Hussain, Mazhar Iqbal, Ghulam Sarwar Jadoon

Department of Community Medicine, Khyber Medical College Peshawar and

Gomal Medical College D.I.Khan, Pakistan

ABSTRACT

Background: Chronic Obstructive Pulmonary Disease is a common cause of morbidity worldwide. This study was conducted to assess the relationship between silica dust inhalation and COPD among workers of dust generating industries.

Materials & Methods: This cross sectional study was conducted in three main industrial regions of Peshawar i.e. Industrial Estate Hayatabad, Industrial area Ring Road and Industries of Warsak Road Peshawar. A preformed questionnaire was used to interview 160 workers, 40 each from stone-grinding, ceramics, pottery and brick industries respectively. Only workers having worked for more than five years were selected through convenient sampling.

Results: A total of 160 workers were interviewed out of which 56(35%) were symptomatic while 104(65%) were asymptomatic. Among symptomatic ones, 20(19.8%) were in 20-40years age group and 36(61%) in 41-60 years age group. In regard to exposure status, 7(26%) were symptomatic in those exposed for 5-10 years, 29(31.5%) amongst those exposed for 11-15 years and 20(49%) in those exposed for 15-20 years. With respect to working hours, none was symptomatic in those who had worked for 6 hours or less while 56(40%) were symptomatic in those who had worked for 8 or more hours. Among 128 smokers, 48(37.5%) were symptomatic and amongst 32 non-smokers 8(25%) were symptomatic. Amongst the symptomatic cases symptoms appeared within 5-10 years in 7(12.5%) of the cases, within 10-15 years in 29(51.8%) of the cases and within 15-20 years in 20(35.7%) of the cases. The major symptoms reported were cough in 56(100%) of the cases, dyspnea in 48(85.7%) and wheezing in 49(87.5%) of the symptomatic cases.

Conclusion: The study revealed that majority of respondents who were exposed to silica dust for ten years or more, had respiratory problems. The severity of the problems was directly proportional to the duration of exposure to silica dust, density of dust (maximum in stone crushing), hours of daily exposure and other contributory factors like tobacco smoking and increasing age.

Key words: Silica dust, Dust exposure, COPD.

INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is a global health concern and a major cause of chronic morbidity and mortality world-wide. According to the World Health Organization, it is currently the sixth leading cause of death in the world, and further increases in the prevalence and mortality of the disease is predicted for the coming decades. These increases are mainly linked to the epidemic of tobacco exposure and indoor and outdoor air pollution. The burden of COPD in Asia is currently greater than that in the developed western countries, both in terms of total number of deaths and the burden of disease,

as measured in years of life lost and years spent living with disability. 2

One of the causes of COPD is inhalation of crystalline silica dust particles less than 7 micrometer in size. The other form of silica that occurs in nature is amorphous which is not as dangerous as crystalline. Crystalline silica occurs in 3 forms. The most commonly occurring form is quartz, while less common are crystobalite and tridymite. But at temperatures >570 C alpha quartz is converted to beta quartz, at >870 C to beta tridymite and at >1470 C to beta crytobalite. Crystalline silica is more dangerous because of its structure which is in the form of sharp pointed crystals. These crys-

tals have highly reactive surfaces and are cytotoxic damaging the cell membrane. When it is freshly ground or fractured it produces highly reactive surface radicals, cleavage of Si-O bonds create sites for adsorption of biological material.³ The severity of disease depends upon the size, shape, concentration of particles and duration of exposure.⁴ Epidemiological and pathological evidence suggests that COPD is independent of silicosis and air flow obstruction can exist even in the absence of pathological effects of silica in lungs.⁵

Rapid industrialization, requiring heavy supplies of construction material like cement, bricks. sand and ground stones, is producing silica dust in health endangering amounts. Cigarette smoking, lack of protective measures against silica dust inhalation and long hours of daily exposure are all adding significantly to the problem.6 During the period 1991-1995, China recorded more than 500,000 cases of silicosis and more than 24,000 deaths occurring each year mostly among older workers while in India, a prevalence of 55% was found in a group of workers engaged in the quarrying of shale sedimentary rock and subsequent work in small, poorly ventilated sheds.7 Approximately 1.7 million US workers are exposed to crystalline silica each year and according to the American Lung Association, approximately 60,000 of these workers will suffer some degree of COPD.8

World COPD day is an annual event organised by the Global Initiative for Chronic Obstructive Lung Disease (GOLD) to improve awareness and care of COPD around the world and it is celebrated on the 19th November every year.⁹

In Asian countries, epidemiologic studies are scant, patchy and localized. Population-based studies are rare. According to WHO estimates the number of COPD cases in Asia exceeds by three times the total number of COPD cases for the rest of the world. Asia Pacific Round Table group consisting of a panel of regional respiratory experts, determined an overall pevalence rate of 6.3% for South Asian Region, which is considerably higher than that estimated by WHO for the region (3.8%).

Unprotected exposure to silica dust is a problem in the entire developing world. It is also a grave issue in Pakistan where majority of the population is poor and work as daily wage laborers in the factories and construction industry.

The purpose of this study was to establish a relationship between prolonged silica dust exposure and COPD.

MATERIAL AND METHODS

This was a cross sectional study. The sample population included 160 factory workers having

exposure to silica dust for 5 years or more. They were conveniently selected and interviewed through a pre-formed questionnaire, giving equal representation of 40 to four main types of industries producing silica dust i.e. stone-grinding, ceramics, pottery and brick industries. We controlled for the confounding effects of smoking by adjusting it since smoking exerts a synergistic effect with silica dust.

Inclusion criteria were adult males, age not less than 20 years who worked in silica related industry for 5 years or more. Workers suffering from chronic chest problems before entry into the silica related industry were excluded from the study.

The obtained data were processed by using SPSS version 10 and MS Excel. Results were presented by creating bar charts and tables. Frequencies and percentages were used in qualitative variables while quantitative variables were either presented as such or in categorical style by creating dummy variables, whichever was logically more plausible.

RESULTS

A total of 160 workers were interviewed out of which 56 (35%) were symptomatic while 104 (65%) asymptomatic.

With respect to age, the workers were divided into two groups. Between 20-40 years, 101 workers were interviewed, out of which 20 (19.8%) were symptomatic and 81(80.2%) asymptomatic. While between 41-60 years of age, 59 were interviewed, of which 36 (61%) were symptomatic and 23(39%) asymptomatic.

In regard to exposure status; 27(16.9%) of the workers had been exposed for 5-10 years and out of which 7 (26%) were symptomatic while 20 (74%) asymptomatic. 92(57.5%) had been exposed for 10-15 years out of which 29 (31.5%) were symptomatic and 63 (68.5%) asymptomatic. On the other hand 41 (25.6%) were exposed for 15-20 years and out of them 20 (49%) were symptomatic and 21(51%) were asymptomatic.

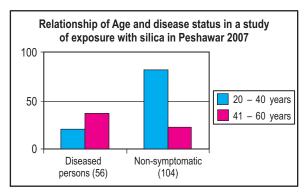
With respect to working hours, 20 (12.5%) had worked for 6 hours or less. 140 (87.5%) had worked for 8 hours or more. Out of these140 individuals, 56 (40%) were the symptomatic cases.

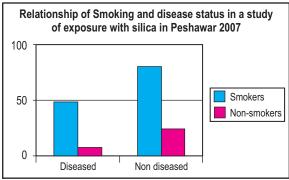
With respect to smoking status, 128 (80%) were smokers and 32 (20%) were non-smokers. Among 128 smokers, 48 (37.5%) were symptomatic and 80 (62.5%) asymptomatic. On the other hand, amongst 32 non smokers, 8 (25%) were symptomatic and 24 (75%) were asymptomatic.

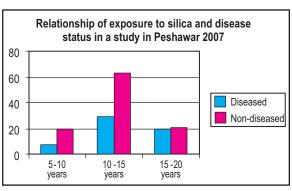
Amongst the symptomatic cases, symptoms appeared within 5-10 years in 7 workers which is

Table	4+	Logietic	ragrassion	Ωf	factors	associated	with	COPD
iable	1.	Logistic	regression	OI	Tactors	associated	witti	COPD.

Age years	Diseased persons (n=56)	Non-symptomatic (n=104)	O.R	p-value	
20 – 40	20	81	1	_	
41 – 60	36	23	6.34	.000	
Smoking status					
Smokers	48	80	1.8	.03	
Non-smokers	8	24	1	_	
Exposure to silica dust					
5-10 years	7	20	1	_	
10-15 years	29	63	1.32	.09	
15-20 years	20	21	2.72	.000	







12.5% of the cases, within 10-15 years in 29 workers which makes 51.8% of the cases and within 15-20 years in 20 workers which makes up 35.7% of the cases.

Regarding symptoms, cough was the major symptom reported by 56 (100%) of the cases. Out of these, 42 (75%) were having productive cough while 14 (25%) had dry cough. Dyspnea was reported by 48 (85.7%) and wheezing by 49 (87.5%) of the symptomatic cases. Weight loss was very uncommon and reported by only 15 (26.8%) of the cases.

DISCUSSION

This article evaluates the evidence for an increased risk of COPD in occupations and industries in which exposure to silica is the primary one, with a focus on the magnitude of risk and level of exposure according to the duration, causing disabling health effects. COPD is expected to become the 3rd leading cause of mortality and 5th leading cause of overall morbidity worldwide by 2020. 12 The effect of silica on lungs is so profound and specific that it has proved to be a confounding variable in gold and coal miners. It has been found that since gold is more mixed with crystalline silica than coal therefore the risk of COPD is more in gold miners as compared to coal miners. 13

All the 160 workers were broadly put into two age groups. Those of higher age group (40-60 years) were more prone (61%) to develop disease and this association was statistically significant (p<0.05). The effect of age on COPD might be due to prolonged exposure as the aged workers have spent more years in the trade.

COPD has a long latent period therefore the clinically disabling disease usually develops a number of years after first exposure and in many cases symptoms become manifest during mid-life or later. 14 In our study amongst the symptomatic ones, 17% had an exposure duration of 5-10 years, 31% 10-15 years while 49% were exposed for 15-20 years. It was found that the association was significant in those who were exposed for more than 15 years and they were about 3 times more prone to develop COPD.

Along with the number of years of exposure one important factor is the number of hours of work per day. In our study it was found that 92% of the workers were working for 8 hours or more and out of them 35% were symptomatic. This can happen by intensive exposure for a short duration regularly or moderate exposure for more hours on a daily basis. Longitudinal studies suggest that loss of lung function occurs with exposure to silica dust at concentrations of 0.1 to 0.2 mg/m³.15 Thus those who take more periods of rest or work for lesser hours are less exposed and are at a lesser risk.

Besides the exposure of silica the nature of silica is also important. This study was conducted on four industries where crystalline silica is the major component. The occurrence of silicosis, tuberculosis, COPD has been documented with crystalline silica and not much with amorphous silica which is the synthetic form used in various industries like rubber, tyre, powder materials, toothpaste additives, paints, silicon rubber, insulation material, liquid systems in coatings, adhesives, printing inks, plastisol car undercoats, and cosmetics.16 Crystalline silica, as mentioned earlier is more dangerous because of its needle like structure which is cytotoxic and produces highly reactive surface radicals after grinding which favor the adsorption of biological materials.3

The synergistic effect of tobacco smoke and occupational silica dust exposures on decreasing the lung function has been well documented by various researchers. It has been found that majority of the cases are attributable to cigarette smoking while cigar and pipe smoking have a lesser role.17-19 In this study 80% of the symptomatic workers were found to be smokers and the rest nonsmokers. Among the smokers, 2/3rd had smoked for 5-15 years and 1/3rd for more than 15 years. Smoking also showed a significant association with COPD (p<0.05). Occupational exposures to dust, vapors, and fumes can cause specific occupational respiratory diseases, collectively termed pneumoconiosis, or can add to the risk of COPD developing due to cigarette smoking. The relationship between smoking and various dust exposures like coal dust, silica, and asbestos, and a decline in lung function have been reported in Asia.20

Vast majority reported to have no symptoms before starting work in silica related industries as against symptomatic workers who formed only 6.9% of the study sample. Thus 93% of the total and 80% of the symptomatic cases developed symptoms after the job. This shows that most of them developed pulmonary symptoms in relation to dust exposure as a predominant causative factor while other factors like smoking and age were also acting concomitantly.

It is quite possible that COPD related symptoms may not appear till very late in the disease. In a survey in England most people with COPD did not report having significant symptoms leading to diagnosis. Similarly in the present study 87.5% reported chest symptoms during 10-20 year period while 12.5% developed symptoms earlier i.e. within 10 years of exposure.

The major symptoms of the disease were cough with or without sputum, dyspnea, and wheezing. ²² Cough was the major symptom present in 100% of the cases. More than 3/4th of the symptomatic persons reported dyspnea and wheezing while the remaining didn't experience these symptoms. Only 26.8% reported to have lost some of their body weight. Wheezing and dyspnea on exertion generally occur when the FEV1 is less than 50% of the predicted value, and significant physical disability usually occurs when it is less than 35-40%. ²³

CONCLUSION

The study revealed that majority of respondents who were exposed to silica dust for ten years or more, had respiratory problems. The severity of the problem was directly proportional to the duration of exposure to silica dust, density of dust (maximum in stone crushing), hours of daily exposure and other contributory factors like tobacco smoking and increasing age.

REFERENCES

- Aýt-Khaled N, Enarson D, Bousquet J. Chronic respiratory diseases in developing countries: the burden and strategies for prevention and management. Bulletin, World Health Organ 2001; 79: 971–9.
- 2. Tan WC, Ng TP. COPD in Asia, Where East meets West. CHEST **2008**; 133: **517-27**.
- Respirable Crystalline Silica: Occupational Exposures and Disease. 31st Annual Industrial Hygiene Conference. Presented by Connecticut River Valley Section. American Industrial Hygiene Association, Plantsvilles Connecticut.

- November 9 2006. Silicosis % 20rice % 202006.pdf
- Getex discuss control measures to prevent Silica dust exposure at workplace.htm/
- Hnizdo E and Vallyathan V. Chronic obstructive pulmonary disease due to occupational exposure to silica dust: a review of epidemiological and pathological evidence. Occupational and Environmental Medicine 2003; 60: 237–43.
- Rivera RM, Cosio MG, Ghezzo, Salazar, Pérez-Padilla. Comparison of lung morphology in COPD secondary to cigarette and biomass smoke. The International Journal of Tuberculosis and Lung Disease 2008; 12: 972-7
- 7. World Health Organization Silicosis.htm/
- Indecent Exposure Silica Dust MC Magazine November-December 2004 - Concrete Publications - NPCA.htm/
- World chronic obstructive pulmonary disease day.htm
- Murray, CJL, Lopez, AD Mortality by cause for eight regions of the world: global burden of disease study. Lancet 1997; 349: 1269-76.
- 11. Regional COPD Working Group.. COPD prevalence in 12 Asia-Pacific countries and regions: projections based on the COPD prevalence estimation model. Respirology 2003; 8: 192-8.
- Mannino DM, Homa DM, Akinbami LJ, Ford ES, Redd SC. Chronic obstructive pulmonary disease surveillance-United States, 1971-2000. Respiratory Care 2002; 47: 1184-99.
- Oxman AD, Muir DC, Shannon HS, Stock SR, Hnizdo E, Lange HJ. Occupational dust exposure and chronic obstructive pulmonary disease. A systematic overview of the evidence. Am Rev Respir Dis 1993; 148: 38-48.
- Review of literature on chronic bronchitis and emphysema and occupational exposure.MRC Institute for Environment and Health (2005) Leicester, UK.
- Rushton L. Chronic obstructive pulmonary disease and occupational exposure to silica. Re-

- views on Environmental Health. 2007; 22: 255-72.
- Merqet R, et al. Health hazards due to the inhalation of amorphous silica. Archives of Toxicology. 2002; 75: 625-34.
- Manfreda J, Johnston B, Cherniack RM. Longitudinal change of lung function: comparison of employees of hard rock mining industry and general population. Am Rev Respir Dis 1984; 129: 142.
- Hnizdo E. et al. Combined effect of silica dust exposure and tobacco smoking on the prevalence of respiratory impairments among gold miners. Scandinavian Journal of Work, Environment & Health 1990; 16: 411-22.
- Krzyzanowski M, Jedrychowski W, Wysocki M. Occupational exposures and changes in pulmonary function over 13 years among residents of Crakow. Brit J Ind Med 1988; 45: 747-54.
- Wang X, Yano E, Nonaka K, et al. Respiratory impairments due to dust exposure: a comparative study among workers exposed to silica, asbestos, and coalmine dust. Am J Ind Med 1997; 31: 495-502.
- Shahab L, Jarvis M, Britton J, West R. Prevalence, diagnosis and relation to tobacco dependence of chronic obstructive pulmonary disease in a nationally representative population sample. Thorax 2006; 61: 1043-7.
- 22. Chronic Obstructive Airway Disorders. In: Beers MH and Berkow R. (eds). *The Merck Manual of Diagnosis and Therapy*, 17th ed. (online). Available at:http://www.merck.com/mrkshared/mmanual/section6/chapter68/68c.jsp.
- 23. Petty TL. COPD in perspective. Chest 2002; 121 (Suppl 5): 116S-20S.

Address for Correspondence:

Dr. Bushra Iftikhar Assoc. Prof. Community Medicine Khyber Medical College Peshawar, Pakistan