Case Study

Silicosis in Manufacture of Electric Cable: Report of Four Cases

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Free silica (silicon dioxide) is naturally found in amorphous and crystalline forms in many rocks in the Earth¹,². Inhaled silicon dioxide is usually crystalline and most often quartz which is the most important form. Silicosis is an irreversible disease³ that results from work-related exposures to dusts containing silica crystals, and is characterized by fibrotic tissue reaction caused by the presence of silica in the lungs⁴.

Crystalline silica exposure and silicosis have been associated with work in mining, quarrying, tunneling, sandblasting, masonry, foundry work, glass manufacture, ceramic and pottery production, cement and concrete production, and work with certain materials in dental laboratories⁵.

The diagnosis of silicosis is based on history of exposure to silica dust and multiple, bilateral pulmonary nodules and large opacities on chest radiography⁶. Several studies have demonstrated that high resolution computed tomography (HRCT) is superior to conventional radiography for detection of silicotic lesions, confluence of nodules, and presence of emphysema⁷.

Most pneumoconioses evolves slowly and changes in radiological findings take a long time, usually years, to occur. However, in rare instances, such as acute silicosis associated with exposure to a very large quantity of very fine silicaceous dust, radiological changes can show up within months¹-⁷.

We present clinical and radiological findings of four cases working in the same department producing 'silicone paste' by mixing quartz dust (a synonym for silicon dioxide (SiO₂), silica or silicon) with silicone (polysiloxane [(R₃SiO)₉], a class-name for various synthetic plastic substances made of silicon, oxygen, carbon, germanium, and hydrogen), in an electric cable factory, for 5 to 8 yr. Up to now, this branch of industry has not been reported in the English literature as a cause of silicosis.

Case 1

A 30-yr-old male patient was admitted to our clinic in April 2005 with complaints of progressive cough, sputum production, dyspnea, wheezing and malaise over the last ten months. He had smoked four cigarettes per day for four years. He had worked in the electricity cable factory for 8 yr. His physical examination revealed blood pressure (BP) of 105/75 mmHg, pulse rate (PR) of 88/min. The only abnormal physical examination finding was bilaterally decreased breath sounds. Pulmonary function tests revealed very severe obstruction (Table 1). His blood gas analysis results, leukocyte count, sedimentation rate, and CRP are shown in Table 1. Chest radiograph showed bilateral increased opacities in the upper zones representing progressive massive fibrosis (PMF) (Fig. 1A). Bronchodilator therapy was administered. A thorax tube was used for the treatment of spontaneous pneumothorax in August 2005 and February 2006 at an outside clinic (Fig. 1B). At the patient’s last admission to our clinic his blood gas analysis revealed PO₂ of 48.2, PCO₂ of 32.8, Ph of 7.47 and O₂ sat of % 86.8. Pulmonary artery pressure was measured as 55 mmHg in echocardiography. The patient had been receiving both oxygen and bronchodilator therapy since August 2005. The patient died due to respiratory failure in April 2006.

Case 2

A 27-yr-old male patient was referred to our clinic in April 2005 with complaints of progressive cough, sputum production, dyspnea, wheezing and malaise lasting for 10 months. He had a smoking history of 15 cigarettes per day for eight years. He had been working in the same department as Case 1 since 1997. BP was 115/80 mmHg, PR was 77/min, and respiratory system and other system examinations were normal. The patient’s leukocyte count, sedimentation rate, and CRP are shown in Table 1. Although within normal limits FEV1/FVC was slightly decreased in pulmonary function tests. There was a decrease in FVC and FEV1 values (Table 1). Chest radiograph showed bilateral hilar enlargement and bilateral, multiple milimetric nodules predominantly in the lateral parts of the upper and middle zones (Fig. 2A). Thorax CT showed multiple enlarged mediastinal and hilar lymph nodes some of which included peripheral calcifications (Figs. 2B and 2C). HRCT showed bilateral centrilobular and subpleural nodules, coalescence of some in the periphery, and interlobular septal thickening (Fig. 2D). The patient was still suffering from dyspnea and cough sixth months later, and there was no remarkable change on control CT scans. The patient is still being followed at our clinic.
Case 3

A 31-yr-old male patient was admitted to our clinic in June 2005 with complaints of progressive cough, dyspnea, chest pain, loss of appetite, malaise and weight loss. He had a smoking history of 15 cigarettes per day for 12 yr. He had been working in the same department, as Cases 1 and 2, for eight years. His physical examination revealed BP of 120/75 mmHg, PR of 81/min, and both respiratory system and other system examinations were normal. His leukocyte count, sedimentation rate, and CRP are shown in Table 1. Although there was a slight impairment in respiratory function test parameters, they were within normal limits. Chest radiograph showed right hilar enlargement (Fig. 3A), which was shown as enlarged lymph nodes with egg-shell calcifications on CT scans (Figs. 3B and 3C). HRCT demonstrated pleural- based fibrotic areas with irregular margins in the right upper lobe posterior segment (Fig. 3D). To exclude malignancy, CT guided fine needle biopsy was offered, but the patient refused it. His complaints regressed and his pulmonary function test parameters were normal at sixth months follow-up. There was no change in the control CT scan. The case is still being followed at our clinic and currently has no complaint.

Case 4

A 32-yr-old male patient was admitted to our clinic in June 2005 with complaints of cough and sputum production present for the last three months. He had a

| Table 1. Pulmonary function tests, blood gas analysis, CRP, sedimentation rate, and leukocyte counts of the patients |
|-------------------------------------------------|-------------------------------------------------|-------------------------------------------------|-------------------------------------------------|
| Case 1  | Case 2  | Case 3  | Case 4  |
| FVC (L)  | 2.42 (49%)  | 3.82 (77%)  | 3.99 (86%)  | 3.67 (74%)  |
| FEV1 (L)  | 1.02 (25%)  | 2.91 (69%)  | 3.28 (84%)  | 3.31 (80%)  |
| PEF (L/second)  | 1.73 (18%)  | 6.37 (66%)  | 5.50 (59%)  | 9.98 (104%)  |
| FEV1/FVC (%)  | 42 (51%)  | 76 (92%)  | 82 (101%)  | 90 (111%)  |
| pH  | 7.44  |  |  |  |
| O2 Saturation (%)  | 93  | 95  | 96  | 97  |
| PaO2 (mmHg)*  | 64.6  |  |  |  |
| PaCO2 (mmHg)*  | 35.1  |  |  |  |
| Leukocyte count (×10^3/µl)  | 8.9  | 7.8  | 4.9  | 5.2  |
| CRP  | 7.05  | 5.45  | 10.5  | 4.87  |
| Sedimentation rate (mm/h)  | 35  | 35  | 99  | 5  |

* Blood gas analyses were not performed for cases 2, 3 and 4 as their O2 saturations were within normal limits.

Fig. 1.  A: Chest radiograph shows large bilateral, symmetrical heterogenous opacities in the upper zones due to progressive massive fibrosis accompanying bilateral apical bullae as well as upward traction of both hila. B: Spontaneous pneumothorax on the right side is shown in the radiograph taken 4 months after first diagnosis and visceral pleura is adjacent to the inserted thorax tube (arrow).
Fig. 2. A: Chest radiograph shows multiple milimetric nodules in both lungs, predominantly in the peripheral regions of the upper and middle zones. There is also a bilateral hilar nodular enlargement (arrows). B: Hilar nodular enlargement represent lymph node enlargements which involve peripheral punctate calcifications (arrows) on contrast-enhanced axial CT scan with mediastinal window settings at the level of the pulmonary truncus. C: HRCT scan at the supra-aortic level demonstrates bilateral centrilobular and subpleural (perilymphatic distribution) small nodules, coalescence of some nodules in the lung periphery and interlobular septal thickening.

Fig. 3. A: Chest radiograph shows right hilar nodular enlargement. There is no distinguishable small nodules or large opacities. B: Hilar nodular enlargement in the chest radiograph represent lymph node enlargements involving peripheral punctate calcifications resembling ‘egg-shell’ (arrows) on contrast-enhanced axial CT scan with mediastinal window settings at the subcarinal level. There are additional non-calcified subcarinal and left tracheo-bronchial enlarged lymph nodes and atelectasis in the middle lobe of the right lung. C: Enlarged lymph nodes with ‘egg-shell’ calcifications at the right paratracheal area at the level of aortic arch (arrow). D: HRCT scan at the level of great vessels shows subpleural opacity greater than 2 cm diameter with irregular margins indicative of progressive massive fibrosis (black arrow), small nodules and traction bronchiectasis (white arrow) in the upper lobe of right lung. There is no calcification in the fibrotic area and no associated paracicatricial emphysema.
smoking history of 10 pack-years. He had been working in the same department as the other cases for 5 yr and 8 months. His physical examination was normal. BP was 110/70 mmHg and PR was 75/min. His leukocyte count, sedimentation rate, and CRP were unremarkable. Mild restriction was determined in pulmonary function tests (Table 1). Chest radiograph showed right paratracheal nodular opacities (Fig. 4A) represented as enlarged lymph nodes on CT (Figs. 4B and 4C). HRCT revealed centrilobular and subpleural nodules associated with ground-glass opacity following bronchiolovascular distribution. There was also interlobular septal thickening and fibrotic changes, and tractional bronchiactasis in the right lung apical region (Fig. 4D). The patient had no complaints at the sixth-month follow-up, although minimal impairment persisted in the pulmonary function tests. Minimal progression was detected in the lesions in a control CT scan. The case is still being followed at our clinic and has no complaint.

Discussion

The factory that employed our patients manufactures rubber materials and gaskets for household appliances and cars, such as silicone isolation for temperature resistant cables or silicone gaskets which go around car windshields. The factory had a total of 250 workers and 8 administrative personnel. The factory consists of several departments including silicone paste production, cable line production, cable grouping, cable cutting & enveloping, cable testing, gasket molding, gasket cutting and gasket press.

In the silicone paste production department, silicone (polysiloxane; \([R_2SiO]_x\), a class-name for various synthetic plastic substances made of silicon, oxygen, carbon, germanium, and hydrogen) is mixed with quartz dust (silicon dioxide, silica or silicon) at a ratio of one fifth to one half of pure silicone along with colorant and catalysts. Quartz powder is added as a reinforcing agent. The mixture is prepared by a machine called a ‘mixer’. As the mixer was not air-tight, quartz dust was heavily present in the room air and inhaled directly by the workers in this department. All four of the patients that had the diagnosis of silicosis were in the silicone paste production department. They had been working in the same department from the beginning of the manufacturing in 1997. These workers did not have masks until the Department of Labor fined the factory in 2004 for lack of precautions for protecting employees, for insufficient ventilation and for the use of a mixer which was not air-tight. As a result of this prosecution partial improvements were made in the silicone paste production department. Those improvements were: buying one air-tight mixer,
ventilation and providing protective breathing masks. Despite the lack of data of a numeric expression for the quartz concentration in the air, because of the still ongoing trial, the partial improvements do not seem to be sufficient to decrease the dust burden as there are still two non-airtight mixers in the same department adjacent to the airtight mixer and protective breathing masks are not sufficient for complete prevention of dust inhalation.

The usual chronic form of silicosis requires 20 yr or more exposure to high dust concentrations before radiographic abnormality becomes visible. With very high concentrations of dust, radiographic changes can appear in 4 to 8 yr\(^1\). In our study the time between the quartz dust exposure and the beginning of symptoms was 5 to 8 yr (mean 7 yr 5 months). As no control chest radiograph had been obtained before 2004 we do not know the exact time of initiation of radiological findings. When the time duration between beginnings of exposure and symptoms is taken into consideration our cases appear to be the accelerated form of silicosis. Development of accelerated silicosis in all patients working in the same department indicates the strong possibility of intensive exposure. Acute silicosis in jean sandblasting was reported in which the exposure time was less than five years in two patients\(^8\).

Our cases have two common demographic features: the age range of the four cases which was between 27 and 32 yr old (mean age 30 yr); and their cigarette consumption. Although no significant difference was reported in mean lung function parameters between those who had never smoked and smokers, who had a cigarette consumption of 20 pack-years or more the impact of smoking in accelerated silicosis might require further evaluation\(^9\).

Radiologically, nodular lesions were present in all cases. Progressive massive fibrosis (PMF) covered the upper zones bilaterally in Case 1 and was limited to the right upper lobe in Case 3. These two cases were the ones with the longest exposure times. Antao et al. reported a relationship between the intensity of the opacities seen in HRCT and functional impairment\(^10\). According to radiological findings and pulmonary function test parameters the most severe effect was seen in Case 1 who eventually died due to respiratory failure. In other cases there was no correlation between CT findings and pulmonary function tests. In several studies it was suggested that simple silicosis without superimposed emphysema has no influence on lung function. Another study observed a milder reduction in lung function associated with simple silicosis and a more severe reduction in lung function with increasing nodular profusion and PMF\(^9\). There was diffuse mediastinal lymphadenopathy in all cases with ‘egg shell’ calcification appearance in Case 2 and Case 3. It was hypothesized that the development of fibrotic nodules in the lymph nodes may influence the subsequent development of parenchymal silicosis due to obstruction of lymphatic drainage of the lung with consequent increase in lung dust burden\(^4\).

Exposure to quartz dust due to insufficient preventive precautions has led to the development of accelerated silicosis in our cases. After silicosis diagnoses of employees in the silicone paste production department, 20 other employees working in other departments were called in for check-up purposes. These 20 people were not directly exposed to quartz dust as in the silicone paste production department. Some of them exhibited small non-specific interstitial thickening in their HRCT exams. These people underwent regular examinations for almost a year and no changes on HRCT scans have been observed.

For prevention of silicosis in the future, precautions should be taken such as regular dust concentration measurements, particularly in the silicone paste production and also in other departments, providing appropriate protective breathing masks and controlling their usage, changing all old technology mixers for airtight systems, and improving or installing air-conditioning systems. In addition, employees should be checked regularly for silicosis and the ones who are suspicious for silicosis should be referred to hospitals having specialists on silicosis.

In conclusion, new cases of silicosis in new or previously unknown branches of industry still occur. If any kind of industrial manufacturing process consists of addition of quartz dust to silicone, such as in the production of rubber reinforcing agents in electric cable and gasket production, or as a filler in casting electrical components’ production, such as switch parts, ignition coils, and bell transformers, appropriate preventive measures should be present in the working environment to prevent or decrease the presence of quartz dust burden in the air.

We present clinical and radiological findings of accelerated silicosis of four young workers due to exposure to quartz while producing silicone paste by mixing quartz dust with silicone, and to the best of our knowledge this is the first report of silicosis in the manufacture of electric cable.

References

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