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CASE REPORT

A Case Report of Silicosis: Culprit of Progressive Massive Fibrosis

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ABSTRACT

Silicosis is a fibronodular lung disease secondary to the inhalation of crystalline silica dust. It had continued to cause significant morbidity and mortality worldwide. Here is a case of a 63-year-old woman, a lifelong non-smoker who complained of intermittent wheezing since retired 8 years ago. She had worked in the clay and piping industry for more than a decade. She wore only a simple 3-ply face mask at work without other protective devices. She had multiple hospital admissions for acute exacerbation of chronic lung disease for the past several years. Respiratory examinations revealed bilateral fine crepitations and occasional rhonchi on auscultation. Chest radiograph revealed ground glass changes. Pulmonary function testing showed an irreversible severe obstruction picture with an FEV1 of 45%. High resolution computed tomography thorax demonstrated hyperinflated lungs with emphysematous changes and multiple nodules over subpleural region, conglomerate mass with calcifications over bilateral lungs. Her imaging findings combined with a significant occupational history were suggestive of progressive massive fibrosis (PMF) due to silicosis. She is currently being treated with several inhalers and does not require home oxygen therapy. She is suffering from accelerated silicosis which may potentially progress to radiological deterioration, altered respiratory function and premature death. Therefore, it is essential to avoid any potential hazards that may predispose to silicosis.

INTRODUCTION

Silicosis is a disabling and irreversible lung disease causing progressive massive fibrosis. It takes a toll on occupational health diseases.

The causative agent is the crystalline silica dust presents in concrete, sandstone, rock and other abrasives. Workers at risk are those who use machines to remove paint or rust, who grind mortar, crush, haul, chip and drill concrete or rock, and who perform a variety of construction jobs. In high-risk industries, the high biological reactivity of inhaled freshly crushed silica dust (concentration as high as 99%) can cause accelerated progression of the disease because of its greater respiratory burst and cytotoxic effects on alveolar cellular membrane integrity (Bhattacharya et al., 2016). Prevention is better than cure. For this reason, National Institute for Occupational Safety and Health has advocated routine chest radiographs to United States coal miners aiming for early detection of coal workers' pneumoconiosis (National Safety Council, 2015). Despite this, silicosis still can occur in the absence of further exposure and this has increased the incidence of Tuberculosis or atypical mycobacterium diseases, resulting in an increasing healthcare burden. This case report aims to highlight the significance of relating chronic progressive lung disease with occupational hazards and the importance of exploring a detailed occupational history.

CASE PRESENTATION

A 63-year-old woman, who is a lifelong non-smoker, had been complaining of wheezing and dyspnoea since retiring from her work 8 years ago. She was diagnosed to have hypertension for the past 10 years. She has no childhood asthma and has no significant family history of asthma or atopy. She had worked in the clay and piping industry for more than a decade without any personal protective device. On further questioning, she admitted that she wore only a simple 3-ply face mask at work without any other protective respirator, gown or clothing. She did not separate her working attire from normal clothing during laundry. Ever since she has retired 8 years ago, she experienced progressive worsening dyspnoea and wheezing leading to multiple hospital admissions. Respiratory examinations revealed bilateral fine crepitations and occasional rhonchi on auscultation, cardiovascular examination did not show any evidence of pulmonary hypertension, while other systemic examinations were unremarkable. Chest radiograph (Figure 1) showed ground-glass appearance over her bilateral lung mid-zones which mimics the typical 'angel's wings' appearance.

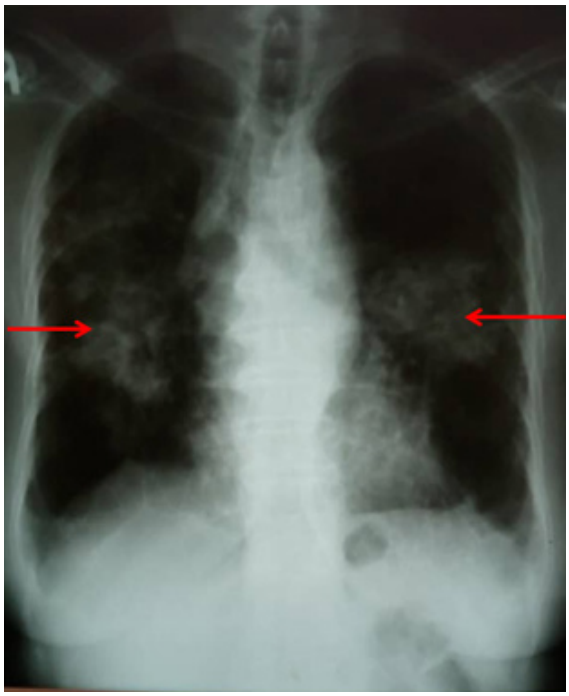

Figure 1 Chest X-ray P/A view shows reticulonodular shadows and multiple mass-like lesions with irregular margins over right upper to mid-zones and left mid-zone with background ground glass appearance in both hyperinflated lung fields causing an 'angel's wings' appearance (indicated by red arrows). The lateral interfaces of the mass lesions parallel with the lateral chest wall.

High-Resolution Computed Tomography (HRCT) of the thorax (Figure 2 and Figure 3) demonstrated hyperinflated lungs with emphysematous changes with multiple nodules over the subpleural region and conglomerate mass with calcifications over bilateral lungs (both upper and lower lobes) with a fibrotic band and adjacent pleural thickening.

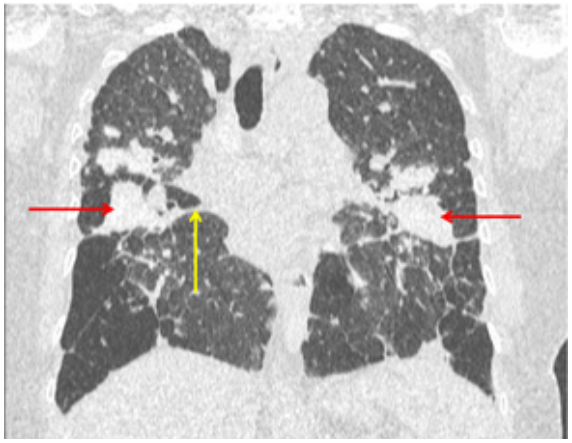

Figure 2 HRCT thorax axial view shows hyper-inflated lungs with multiple calcified mass (conglomerate mass indicated by red arrows) in both perihilar regions with surrounding fibrosis and adjacent pleural thickening (indicated by the yellow arrow)

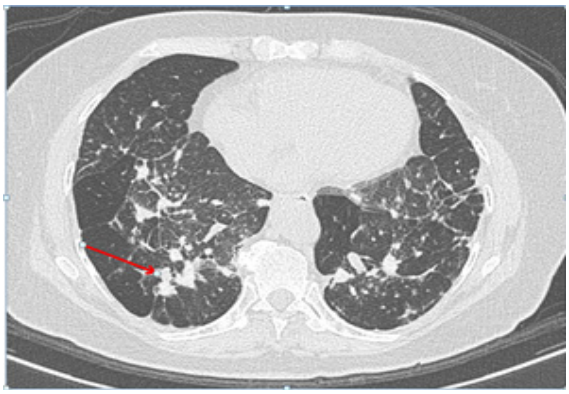

Figure 3 HRCT thorax coronal view shows scattered multiple small nodules (indicated by the red arrow)

These findings were in keeping with progressive massive fibrosis. Pulmonary function testing was suggestive of irreversible severe obstruction with an FEV1 of 45% and FEV1/FVC of 49% which is shown in Figure 4. Initially, she was discharged with MDI Berodual (short-acting muscarinic agonist), MDI Budesonide (inhaled corticosteroids) and MDI Salbutamol (short-acting bronchodilator). In view of persistent cough with wheezing, Ultibro Breezhaler (long-acting beta-agonist/ long-acting muscarinic agonist combination) was added to her current therapy. Her condition is currently controlled with much-reduced exacerbation's frequency and saturating well without the need for home oxygen therapy. She was referred to the occupational health division for potential compensation for her silicosis under the Employee's Social Security Act 1969.

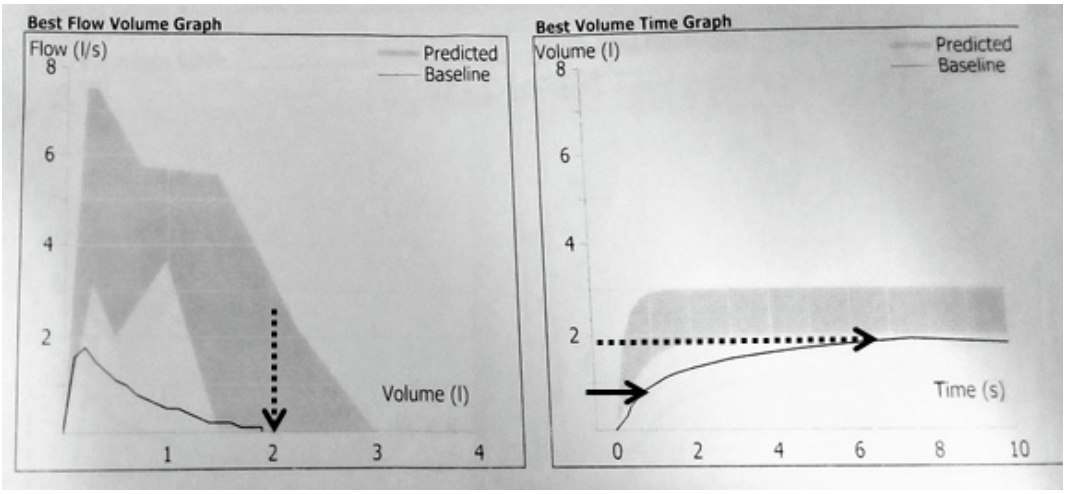

Figure 4 Flow volume graph and volume time graph show obstructive pattern with FEV1 of 45% (indicated by the solid arrow) and FEV1/FVC of 49% (FVC indicated by the dotted arrow).

DISCUSSIONS

There are 3 types of silicosis that existed. Type 1 (chronic silicosis) is the most common and generally develops after 10 years exposing to low levels of crystalline silica. Type 2 is accelerated silicosis developed 5 to 10 years after exposure to higher levels of crystalline silica. Type 3 is acute silicosis which occurs months or weeks after exposure to a very high concentration of crystalline silica, usually leading to death in months (National Safety Council, 2015). Silicosis is associated with multiple lung comorbidities including lung cancer, autoimmune diseases and tuberculosis which are also known as 'silicotuberculosis' (Ali et al., 2010).

The formation of silicotic nodules is the primary pathology of silicosis. Govindaraj et al. (2016) described continuous exposure results in continuous release of inflammatory cytokines which promotes collagen synthesis and produces anti-collagen antibodies, stimulating the production of more collagen which eventually leading to silicotic nodule formation. A silicotic nodule has a central zone of hyalinized fibrous tissue, a middle zone of concentrical collagen fibres and an outer zone of random collagen fibres combining with macrophages and lymphoid cells (Mossman et al., 1998).

Ali et al. (2010) outlined the approach to diagnose silicosis which encompassed exposure to sufficient silica dust, radiographic features of silicosis and exclusion of other illnesses causing similar abnormalities. Pulmonary Function Tests may be normal or obstructive and/ or restrictive abnormalities may be present. A chest radiograph reveals nodules, especially in upper lung zones. In 5 – 10% of cases, calcified hilar nodes which are also known as 'eggshell' calcification, is strongly suggestive, although not pathognomonic of silicosis**.** Progressive massive fibrosis is characterized by mass lesions typically over the lateral interfaces which are parallel to the lateral chest wall (Gera et al., 2014).

In this case, the patient is a lifelong non-smoker presenting with persistent cough associated with progressive worsening dyspnoea and wheezing. Occupational hazards shall ring a bell in this case. The occupational history of exposure to silica dust, progressive nature of breathlessness and the classical radiological findings are the main clues for the diagnosis supported by pulmonary function testing with an obstructive pattern. Her clinical condition fits into accelerated silicosis complicated with progressive massive fibrosis. Although the current lung condition is stable, there is a high chance of further deterioration in terms of symptoms, radiological findings and pulmonary function testing. The mode of silica dust exposure, in this case, was mainly inhalation. Goodman et al. (1992) mentioned that there is no definitive treatment for silicosis while Sharma et al. (1991) stated that only symptomatic treatment in which bronchodilators and corticosteroids may be useful. National Safety Council advocated water spray and dry air filtering to control dust as preventive measures (National Safety Council, 2015).

Silicosis may also be caused by silicone injection as discussed in a case report from Texas which reported a transgender female suffered from interstitial lung disease secondary to silicosis 2 years after underwent a silicone breast implant, together with silicone injections unto thigh and gluteal (Mattox et al., 2018). Otherwise, most of the silicosis is caused by inhalation of silica dust for a long period as reported in other case reports.

CONCLUSIONS

Silicosis is an occupational disease that may present even after the cessation of silica exposure. As there is no cure, prevention is undoubtedly important. The best way of prevention is to identify high-risk workplaces and take necessary precautions as advised by the National Institute of Occupational

Safety and Health (NIOSH). Workers who are exposed to a high level of silica dust should be using standard protective respirators and monitored by occupational physicians with chest radiography regularly. If any early signs of silicosis are detected, the worker should be stopped from further exposure to prevent further complications**.** Exposure to high concentrations of dust is a significant risk factor for the development of progressive massive fibrosis as in this case.

CONFLICT OF INTEREST

The authors declare that they have no competing interests in publishing this case.

CONSENTS

Written consent was obtained from the patient to publish the case. A copy of the written consent is available for review by the Chief Editor.

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