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CASE REPORT

Acute silicosis in a patient with Tubercular Lymphadenitis

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ABSTRACT

Background: Excessive exposure to airborne crystalline Silica can result in irreversible lung inflammation, and fibrosis. **Case Report**: A 22-year man presented with shortness of breath and persistent cough for 3 days. He was taking oral anti-tuberculous therapy for tuberculous lymphadenitis diagnosed by cervical lymph node biopsy for the last 9 months. He had right sided spontaneous pneumothorax 3 months ago, chest tube thoracostomy and pleurodesis with doxycycline were done. For the last 1 year, he had been working in a cement factory but previously was employed in rice shelling. His chest X-ray demonstrated a right sided pneumothorax, non-homogenous opacifications in middle and lower zones bilaterally. HRCT chest revealed bilateral ground-glass opacifications with nodularities and infiltrates more pronounced peripherally. He was diagnosed as having Acute Silicosis based on radiographic findings and occupational history. He was managed with oxygen inhalation therapy and intravenous steroids. **Conclusion**: Counseling regarding occupational rehabilitation was done.

Keywords: Oxygen Inhalation Therapy, Silicosis, Silica, Tuberculosis

This article may be cited as: Samad A, Butt NI, Naz F, Zahid A, Ghoauri MSA, Ashraf T. Acute silicosis in a patient with Tubercular Lymphadenitis. Int J Pathol;22(4):250-4. https://doi.org/10.59736/IJP.22.04.907

Introduction

Excessive exposure to airborne crystalline Silica can result in a serious health hazard leading to lung inflammation resulting in irreversible fibrosis and respiratory abnormalities.(1)Based on clinical, radiological and functional data, Silicosis may be classified into different types: acute, accelerated and chronic.(2) The most common presentation is in the form of chronic silicosis which usually develops after a long latency of 10-30 years after exposure to respirable crystalline silica dust.(3) Acute silicosis occurs within weeks to a few years of silica dust exposure.(4) Accelerated silicosis occurs within 3 to 10 years of silica dust exposure and is associated with higher risk to develop progressive massive lung fibrosis.(3,4) Silicosis is an important occupational health illness having the potential to cause progressive physical disability. Its prevalence varies from 3.5%to 54.6% depending on silica concentration at the work environment, exposure duration and job demands.(1,5)Occupations where crystalline silica exposure occurs include quarrying, mining, glass work including fiberglass industry, construction work including cement blasting, agriculture work including rice shelling and metal product manufacturing industries.(5,6)

Due to improvements in occupational health standards in recent years, there has been a reduction in pneumoconiosis incidence worldwide but pneumoconiosis including silicosis is still a problem in the developing

world including Pakistan.(6) Furthermore numerous difficulties in diagnosis and management of these cases include low awareness, limited resources and co-existence of tuberculosis which is epidemic in most developing countries. Here, we report a case of acute silicosis in a young man with occupational exposure to cement factory and rice shelling. His disease course was complicated by secondary spontaneous pneumothorax recurred twice. Furthermore, he had a history of tubercular lymphadenitis for which he was taking anti-tubercular therapy.

Case Presentation

A 22-year-old gentleman presented with worsening shortness of breath for 3 days along with persistent cough producing scant white sputum. For the last 9 months the patient was taking oral anti-tubercular therapy for tubercular lymphadenitis which was diagnosed by excisional cervical lymph node biopsy. At the time of diagnosis of tubercular lymphadenitis he was having low grade fever, weight loss and palpable cervical and axillary lymph nodes for the last 9 months which had gradually resolved within 2 months of starting anti-tubercular therapy. There was history of a right sided spontaneous pneumothorax 3 months ago for which chest tube thoracostomy and pleurodesis with doxycycline were done. For the last 1 year, he had been working in a cement factory but previously was employed in rice shelling. He was a non-smoker and did not use alcohol or illicit drugs.

Figure 1: Chest X-ray of the patient

On chest examination, there were reduced movements, hyper-resonant percussion and decreased breath sounds on right lower zone but normal vesicular breathing with crepitations were present elsewhere. As shown in Figure 1, his chest X-ray demonstrated a right sided pneumothorax and non-homogenous opacifications in middle and lower zones bilaterally. HRCT chest revealed bilateral ground-glass opacifications with nodularities and diffuse infiltrates more pronounced peripherally as shown in Figure 2. His sputum examination was negative for gram and AFB stain, pyogenic culture and GeneXpert PCR-MTB. He was diagnosed as having Acute Silicosis based on his radiographic findings and occupational exposure history. He was managed conservatively with oxygen inhalation therapy and intravenous steroids. Counseling regarding occupational rehabilitation was done.

Figure 2: HRCT Chest of the patient

Discussion

Pleural involvement in silicosis is uncommon and may occur in form of pleural effusion, rigidity or pneumothorax. (7) Pneumothorax is usually seen unilaterally in pleura-related silicosis and can be potentially life-threatening. Increased risk of secondary spontaneous pneumothorax is more associated with chronic silicosis but can rarely be seen in acute silicosis as well.(7,8) Acute crystalline silica has a size of 10 microns and reaches the lower respiratory system easily where it accumulates to cause disease.(9) Studies show association of secondary spontaneous pneumothorax with bullae presence in silicosis.(7,8) Lack of activation of Type 2 alveolar cells play a role in pathogenesis of pneumothorax as the silica particles induce inequities between inflammatory reaction generation. Inflammatory products formed as a result of silica presence in the lungs lead to damage of elastic fibers in alveolar walls which result in decreased alveolar wall elasticity, bleb and bullae creation which rupture leading to pneumothorax in addition to lung fibrosis. (10,11) Co-existence of tuberculosis with silicosis also expedites development of pulmonary fibrosis and in endemic regions such as South Asia, up to 25% silicosis patients develop tuberculosis as well. (7) Other important complications of silicosis include cor pulmonale and esophageal stress.

The major methods used to diagnose and monitor respiratory surveillance in silica exposed workers include chest X-rays, HRCT chest, pulmonary function tests and health & exposure questionnaires but there may still be a delay in diagnosis.(12,13) Even though the gold standard for diagnosis is lung biopsy but is avoided unless absolutely necessary for another reason because surgical manipulation in silicosis is associated with unfavorable repercussions.(14) The treatment of silicosis is usually conservative due to delay in diagnosis and therapeutic interventions are still limited. For end-category silicosis, lung transplantation is the main modality as it prolongs survival of patients with end-category fibrosis.(15) However facilities of lung transplantation are not readily available in developing countries. Furthermore it is risky, difficult and expensive procedure requiring immunosuppressants after surgery and the post-transplantation median

survival is relatively short.(15) Other treatment options being tried for silicosis include large volume whole lung lavage, pirfenidone, nintedanib, tetrandrine and certain traditional herbal medicines.(11)

In conclusion, workers of certain occupations have silica exposure and are at risk of developing lung dysfunction and fibrosis. It should be noted that large exposures of silica may possibly remain unnoticed as silica is odorless, colorless and may not cause lung irritation immediately. Silicosis may be a long latency period spanning years after exposure with clinical symptoms occurring late in the disease. Crystalline silica is more fibro genic as compared to amorphous silica, highlighting their role and significance in pathogenesis of silicosis.

References

1. Rajavel S, Raghav P, Gupta MK, Muralidhar V. Silico-tuberculosis, silicosis and other respiratory morbidities among sandstone mine workers in Rajasthan- a cross-sectional study. PLoS One. 2020; 15(4):e0230574. Doi: 10.1371/journal.pone.0230574.
2. Fernández-Álvarez R, Martínez-González C, Quero-Martínez A, Blanco-Pérez JJ, Carazo-Fernández L, Prieto-Fernández A. Guidelines for the diagnosis and monitoring of silicosis. Arch Bronconeumol. 2015; 51(2):8693.doi:10.1016/j.arbres.2014.07.010.
3. Krefft S, Wolff J, Rose C. Silicosis: An Update and Guide for Clinicians. Clin Chest Med.2020;41(4):709722.doi:10.1016/j.ccm.2020.08.012.
4. Khemakhem R, Moussa N, Kotti A, Feki W, Mnif Z, Feki W, et al. Accelerated silicosis and silico-tuberculosis: A difficult diagnosis. Clin Case Rep. 2022; 10(2):e05482. Doi: 10.1002/ccr3.5482.
5. Shamim M, Alharbi WD, Pasha ST, Nour OMM. Silicosis, A Monumental Occupational Health Crisis In Rajasthan-An Epidemiological Survey. 2017; 5(7):554-583. Doi: 10.5281/zenodo.841120.
6. Iga N, Nishi H, Fujimoto N, Kishimoto T. Clinical features of secondary spontaneous pneumothorax complicated with silicosis. Respir Investig. 2018; 56(2):144-9. Doi: 10.1016/j.resinv.2017.11.007.
7. Meena MK, Singh R, Joshi N, Rathore SS, Chadalawada S, Abubakar M, et al. Silicosis with Secondary Spontaneous Pneumothorax intheWesternRajasthan.Cureus.2020;12(11):e11811. Doi: 10.7759/cureus.11811.
8. Fotedar S, Chaudhary D, Singhla V, Narang R. Silicosis with bilateral spontaneous pneumothorax. Lung India. 2010; 27(3):173-5. Doi: 10.4103/0970-2113.68325.
9. Saiyed HN, Ghodasara NB, Sathwara NG, Patel GC, Parikh DJ, Kashyap SK. Dustiness, silicosis & tuberculosis in small scale pottery workers. Indian J Med Res. 1995; 102:138-42.
10. Mishra P, Jacob SE, Basu D, Panigrahi MK, Govindaraj V. Bilateral spontaneous pneumothorax in chronic silicosis: a case report. Case Rep Pathol. 2014; 2014:561861. Doi: 10.1155/2014/561861.
11. Li T, Yang X, Xu H, Liu H. Early Identification, Accurate Diagnosis, and Treatment of Silicosis. Can Respir J. 2022; 2022:3769134. Doi: 10.1155/2022/3769134.
12. Austin EK, James C, Tessier J. Early Detection Methods for Silicosis in Australia and Internationally: A Review of the Literature. Int J Environ Res Public Health. 2021Jul;18(15):8123.doi:0.3390/ijerph18158123.
13. Şener MU, Şimşek C, Özkara Ş, Evran H, Bursali İ, Gökçek A. Comparison of the International Classification of High-resolution Computed Tomography for occupational and environmental respiratory diseases with the International Labor Organization International Classification of

Radiographs of Pneumoconiosis. Ind Health.2019;57(4):495502.doi:10.2486/indhealth.2018-0068.
14. Akgun M, Ucar EY, Araz O, Parker JE. Prognosis of patients with silicosis due to denim sandblasting. Chest. 2012;141(3):831. Doi: 10.1378/chest.11-2558.
15. Hoy RF, Chambers DC. Silica-related diseases in the modern world. Allergy. 2020; 75(11):2805-17.doi: 10.1111/all.14202.

HISTORY	
Date received:	20-09-2024
Date sent for review:	23-09-2024
Date received reviewers comments:	23-12-2024
Date received revised manuscript:	26-12-2024
Date accepted:	28-12-2024

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