SILICOSIS MISDIAGNOSED AS SEVERE ASTHMA IN A GOLD MINER FROM ZAMFARA, NORTH-WESTERN NIGERIA.

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ABSTRACT

Background: Silicosis is an incurable occupational fibrotic lung disease caused by inhalation of dust that contains crystalline silica. Illegal small-scale gold mining, one of the major occupations associated with the development of this disabling disease is carried out by artisans in Zamfara state, North-Western Nigeria. Miners uses local tools with little or no means of protection.

Case summary: We present H.B, a 35-year-old gold miner who presented to our unit on account of recurrent cough and progressive shortness of breath of 1.5- and 1-year duration respectively. He received treatment for bronchial asthma from a private hospital for 2 months with no significant improvement. He has been mining gold at one of the gold mines in Maru, Zamfara state for 20 years but stopped a year ago on account of worsening breathlessness. On general physical examination, he was found to be in respiratory distress and centrally cyanosed with an SPO_2 of 86% on room air. Examination of the chest revealed right apical flattening with tracheal deviation to the right and widespread wheezes and crackles all over the lung fields. Spirometric measurement revealed restrictive abnormality. Radiological examination by way of chest x-ray showed bilateral upper and mid zones homogenous opacities and 'tenting' of the left hemidiaphragm. Chest CT scan showed dense soft tissue masses in the upper lobes bilaterally with areas of calcifications including an 'egg shell' calcification on the right hilar region. A diagnosis of silicosis with progressive massive fibrosis was made. He was placed on nebulized levosalbutamol/ipratropium bromide and steroids and was optimized on oxygen therapy. He was subsequently counseled on the need to change occupation.

Conclusion: Silicosis as one of the many causes of progressive fibrosis exists in our unregulated mining communities. The need for vigilance by clinicians and regulatory authorities is hereby emphasized.

INTRODUCTION

Silicosis is an incurable fibrotic lung disease caused by inhalation of dust that contains crystalline silica. Silica is known to exists in rock in the form of quartz, cristobalite and tridymite and prolonged occupational exposure to any form of these types of silica could result in silicosis. Some occupations at risk of silica exposure including construction work, surface and underground rock drilling, sandblasting, foundries and mining are found world over. In 2019, the national institute of occupational safety and health (NIOSH) reported deaths from silicosis to constitute 7% of all pneumoconiosis deaths in United States of America[1]. A growing number of studies performed in Nigeria to identify the effect of occupational exposures on lung function of industrial workers shows significant decline in lung function among workers exposed to high level of dust[2,3,4,5,6]. A study among grind stone cutters in Northern Nigeria found significant number of workers who develops silicosis including progressive massive fibrosis[7]. Those with silicosis in the study, have worked in the stone quarries for longer period and are slightly older than those with no silicosis.

Though mining started in Nigeria during the colonial era with discovery of coal in Udi ridge in Enugu in 1906 and gold in Northern Nigeria in 1913, there is no reported case of silicosis in the history of Nigerian mining industry[8]. The index case is to our knowledge the first case of silicosis to be reported in a Nigerian miner.

CASE REPORT

We report a 35-year-old gold miner from Maru, Zamfara State, Nigeria who was referred to our clinic on account of recurrent cough and progressive shortness of breath of 1.5- and 1-year duration respectively. The cough was productive of yellowish sputum with no hemoptysis. Shortness of breath has been progressive since onset and now occur at rest. He has no orthopnea or paroxysmal nocturnal dyspnea and no body swelling. He received treatment for bronchial asthma for 2 months before referral with no appreciable improvement. He has no history of cigarette

smoking. He has been mining gold at one of the gold mines in Maru, Zamfara state for 20 years but stopped 6 months ago on account of breathlessness. His work at the mining site included rock blasting and subsequent preparation to extract gold.

On general physical examination, he was found to be in respiratory distress and centrally cyanosed with an SPO_2 of 86% on room air. Examination of the chest revealed right apical flattening with tracheal deviation to the right and widespread wheezes and crackles all over lung fields.

Chest radiograph (figure 1) showed homogenous opacity with air bronchograms in upper and middle zones bilaterally with 'tenting' of the left hemidiaphragm. The patient had a computed tomography of the chest (figure 2 and 3) which revealed irregular iso-dense soft tissue masses in the upper lobes bilaterally and areas of calcification including an 'egg-shell' calcification in the right hilar region. There are widespread areas of emphysematous changes in the middle and lower lobes bilaterally. Pleural thickening is also noted in the posterior aspect of left upper lobe. Spirometry revealed restrictive abnormality with Forced Expiratory Volume in 1 second (FEV_1) (% predicted), Forced Vital Capacity (FVC) (% predicted) and FEV_1/FVC (% predicted) of 0.35 (11.55%), 0.35 (9.67%), and 100.00 (124.98%) respectively. Evaluation for pulmonary tuberculosis using sputum for Ziehl-Neelsen stain and Xpert MTB/RIF were negative. Sputum cytology showed inflammatory cells.

A diagnosis of silicosis with progressive massive fibrosis was made. He was placed on antibiotics, bronchodilators and intranasal oxygen therapy. He was subsequently counselled to stop mining and change occupation.

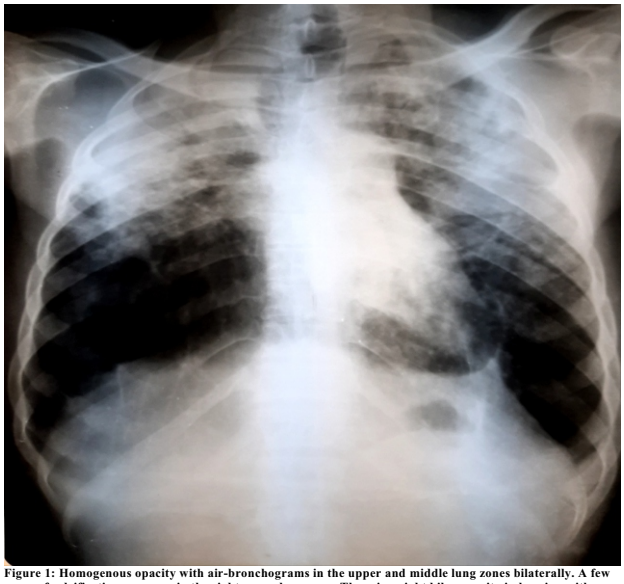

Figure 1: Homogenous opacity with air-bronchograms in the upper and middle lung zones bilaterally. A few areas of calcifications are seen in the right upper lung zone. There is a right hilar opacity in keeping with a hilar nodal mass. There is 'tenting" of the left hemidiaphragm.

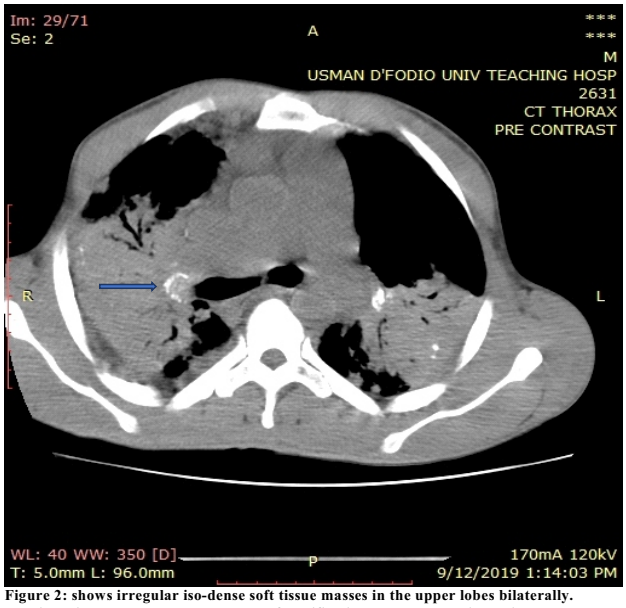

Figure 2: shows irregular iso-dense soft tissue masses in the upper lobes bilaterally. Multiple air-bronchograms and areas of calcifications are also seen including an "egg-shell" calcification in the right hilar region (arrow).

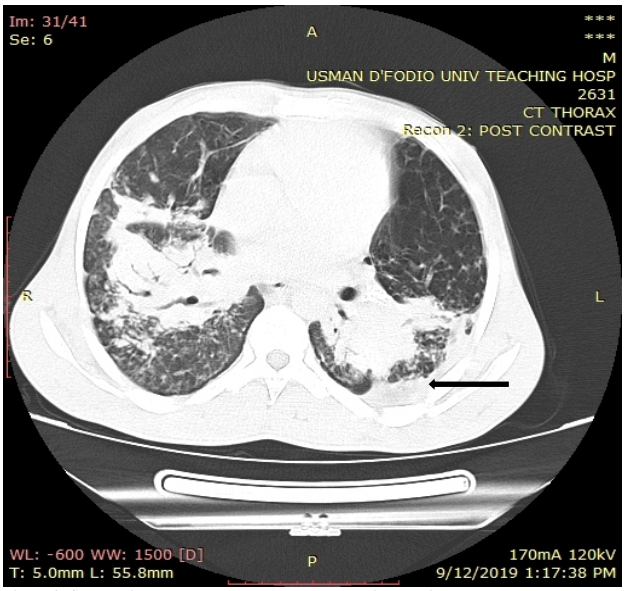

Figure 3: Shows widespread emphysematous changes in the middle and lower lobes. Pleural thickening is seen in the posterior aspect of the left upper lobe (arrow)

DISCUSSION

Many effects have been described in humans exposed to rock and mineral dust generated during mining. One of such effects is silicosis. In 1995, the International Labor Organization (ILO) and World Health Organization (WHO) set a target to eliminate silicosis by the year 2030[1]. The measures taken resulted in a declining trend in the incidence of silicosis in the developed mining countries, however cases of silicosis are rising in countries where small scale artisanal mining activities predominate[9]. Gold mining in Nigeria is carried out in small scale by artisans mainly in the North-Western part of the country. The activity is unregulated with workers mainly children and young adults exposed to hazardous levels of dust with no protective equipments[10].

Three clinical forms of silicosis have been described[11]. chronic silicosis, which occur over a period of twenty to forty-five years of exposure to relatively small amount of quartz (30% or less). Lesions are usually nodular and are likely to be prominent in the upper lobes. In this simple stage of silicosis, nodules are usually small (5mm or less). Normally this stage has little effect on pulmonary function. The complicated form of chronic silicosis also develops in the upper lobes. In this form, the fibrotic nodules coalesce and encompass blood vessels and airways. Pulmonary function maybe severely compromised under these conditions. Acute and accelerated silicosis are the other clinical forms of silicosis which results from very high exposure to silica. This form may appear one to three years after onset of exposure. The distinguishing feature of acute silicosis is intra-alveolar deposits (similar to those seen in alveolar proteinosis), appropriately termed 'silico proteinosis'. Additionally, in contrast to the nodular fibrosis seen in the chronic form, diffuse interstitial fibrosis is found. Silicosis developing in less than 10 years has been described in sandblasters[11]. In these cases, massive fibrosis is likely to develop and locate in the middle lobe and later lower lobes.

The main symptom of chronic silicosis is breathlessness which is more pronounced in patients who have developed progressive massive fibrosis. Cough is also a feature which is mostly dry but may be productive of sputum as the disease progresses. Hemoptysis is not common. Wheezing is not common unless the disease is accompanied by asthma, or if there is significant large airway distortion (such as distortion seen occasionally in progressive massive fibrosis). Chest pain is not a feature of silicosis, nor are systemic symptoms such as weight loss and fever which should be attributed to tuberculosis or lung cancer until proved otherwise. Clubbing is also not a feature of silicosis and should raise concern about lung cancer[12]. In acute silicosis, breathlessness may become disabling within months, followed by impaired gas exchange and respiratory failure.

The ILO international classification of pneumoconiosis[13], is used for the descriptive interpretation of the radiologic appearance of silicosis. Small parenchymal opacities are classified by shape and size: p, q or r for rounded opacities (diameter <1.5mm, 1.5-3mm, or >3mm respectively). Profusion (concentration) is read on a 12-point scale (0/-, 0/1, up to 3/2, 3/3, and 3/+) in comparison with standard radiographs. Large opacities are classified as category A (for one or more such opacities with a diameter of 1cm but not exceeding a combined diameter of 5cm. category B (one or more opacities >10cm in diameter and who's combined area does not exceed one upper zone) and category C (>B). Provision is made to grade pleural thickening for width (a <5mm, b >5mm but <10mm and c ≥10mm) and extent (1= up to one quarter, 2= one quarter to one half, and 3= over half of the lateral chest wall)[13].

Silicosis commonly present with upper lobe lung fibrosis, though, can also present with nodular opacities which can affect any part of the lung. This and other clinical presentations present a diagnostic challenge with pulmonary tuberculosis. Moreover, silica exposure and silicosis are risk factors associated with developing tuberculosis[14]. The diagnosis of silicosis in the index case was based on two decades of exposure to dusty underground gold mining activity, radiological features in keeping with ILO category C opacities involving both upper lung zones, reduced oxygen saturation at rest and marked restriction on spirometry. This is in keeping with ILO criteria for diagnosis of silicosis with progressive massive fibrosis[13]. Microbiologic investigations were also used to exclude pulmonary tuberculosis.

Silicosis is an incurable disease and management mainly target complications once the disease has set in. Experimental treatments used in the past have all been discarded due to lack of effectiveness[1,15]. Complications of silicosis including airway obstruction, respiratory infections, pneumothorax, hypoxemia and respiratory failure are managed as in other respiratory diseases. The presentation of the index case with shortness of breath and wheezes in the chest prompted the misdiagnosis of bronchial asthma. Wheezes in silicosis are as a result of airway disease caused by silica dust. Treatment of the patient with bronchodilators resulted in some minimal improvement.

CONCLUSION

As one of the many causes of occupational lung disease, silicosis exists in our unregulated mining communities. Workplace evaluation is therefore necessary to protect other workers. The need for vigilance by clinicians and regulatory authorities is hereby emphasized.

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