Case Report

Diffuse parenchymal lung disease in a case of chronic arsenic exposure

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ABSTRACT

A 42-year-old housewife, the resident of rural part of West Bengal, presented with gradually progressive exertional dyspnea associated with a dry cough for last 3 years clinical features were suggestive of diffuse parenchymal lung disease (DPLD). Her chest X-ray posteroanterior view and high resolution computed tomography scan of the thorax showed bilateral patchy ground glass opacities and reticulonodular pattern. Search for the etiology revealed classical skin findings of chronic arsenic exposure in the form of generalized darkening and thickening of skin and keratotic lesions over the palms and soles and classical raindrop pigmentation over leg which was present for last 7 years subsequently her bronchoalveolar lavage fluid, hair, nail, and drinking water showed significant amount of arsenic contamination. By exclusion of all known causes of DPLD, we concluded that it was a case of DPLD due to chronic arsenic exposure. To the best of our knowledge, only few case report of DPLD in chronic arsenicosis has been reported till date.

KEY WORDS: Arsenic, bronchoalveolar lavage fluid, diffuse parenchymal lung disease

INTRODUCTION

Arsenic exposure with drinking water and the food is very common in West Bengal particularly in rural parts of north and south 24 Parganas district. Around 150 million people are at risk from arsenic contaminated ground water in the combined areas of West Bengal and its neighboring country Bangladesh.[1] While toxicological mechanisms for pulmonary effects of inorganic arsenic are not known, some reports have demonstrated that arsenic can accumulate in human lung tissue thus enhancing the possibility that the metal can produce respiratory effects.[2] Arsenic-induced malignant lung diseases are well known. Various nonmalignant lung diseases such as obstructive airway disease, bronchiectasis have also been reported.[3] However, arsenic-induced diffuse parenchymal lung disease (DPLD) is a very rare presentation of arsenosis which has been reported here.

CASE REPORT

A 42-year-old nondiabetic nonhypertensive housewife was admitted to the chest department of a tertiary care hospital with gradually progressive exertional dyspnea (modified medical research council grading 2) associated with a dry cough for last 3 years clinical features were suggestive of diffuse parenchymal lung disease (DPLD). Her chest X-ray posteroanterior view and high resolution computed tomography scan of the thorax showed bilateral patchy ground glass opacities and reticulonodular pattern. Search for the etiology revealed classical skin findings of chronic arsenic exposure in the form of generalized darkening and thickening of skin and keratotic lesions over the palms and soles and classical raindrop pigmentation over leg which was present for last 7 years subsequently her bronchoalveolar lavage fluid, hair, nail, and drinking water showed significant amount of arsenic contamination. By exclusion of all known causes of DPLD, we concluded that it was a case of DPLD due to chronic arsenic exposure. To the best of our knowledge, only few case report of DPLD in chronic arsenicosis has been reported till date.

General examination revealed grade 1 digital clubbing. There was no peripheral lymphadenopathy. Systemic examination revealed bilateral scattered end-inspiratory

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crepitations not altered by coughing and posture change. Examination of other systems was essentially normal. Routine blood analysis including complete hemogram, glycemic status, renal hepatic, and thyroid profile were all within normal range. Blood for HIV I and II was negative, and there was no evidence of any immunosuppression. Work up for connective tissue disorders namely serum anti-nuclear antibody by Hep2 cell line, rheumatoid factor, anti-CCP, anti-ds-DNA antibody, anti-phospholipid antibody, anticientromere antibody, anti-Ro, and anti U1 RNP antibody all were negative and serum angiotensin converting enzyme was normal. Two sputum samples for acid-fast Bacilli (AFB) were negative. Her chest X-ray (posteroanterior) showed reticular opacities of both lung fields [Figure 1]. We did a high-resolution computed tomography scan of thorax which revealed bilateral interstitial septal thickening and ground glass opacities suggestive of DPLD [Figures 2 and 3].

Her spirometry showed a restrictive pattern (forced expiratory volume in 1 s/forced vital capacity [FVC]: 82.03, FVC: 58%) and 6-min walk distance (6MWD) was 250 m with 2% desaturation in resting O2 saturation (SpO2) (pretest value 98% whereas posttest value was 96%). The arterial blood gas (ABG) analysis was normal with pH 7.43, pCO2 39 mmHg, and pO2 78 mmHg. At that stage, patient was diagnosed as a case of DPLD of unknown cause. As the patient complaining of progressively increasing distress, we went for a close look up for the etiology.

We noticed generalized darkening of skin and keratotic lesions over the palms and fissures and cracks over the soles which was present for last 7 years and progressively increasing. On further enquiry, we came to know that many other villagers of her village were also having similar skin lesions. She resides in a village of North-24-parganas and used to drink water from a local tube well where arsenic contamination of drinking water is known. Skin of hands showed hyperpigmentation, palmar keratosis [Figure 4] and skin over leg showed “raindrop pigmentation” [Figure 5]. We asked for consultation from our dermatological department who confirmed it as arsenic-induced skin lesions.

Then we did fibreoptic bronchoscopy of this patient which showed normal appearing bronchial tree. Bronchoalveolar lavage (BAL) fluid was taken after wedging the bronchoscope into the subsegmental bronchi both in right and left side and sent for routine examinations along with arsenic level determination. The BAL fluid report showed 22% lymphocyte, 76% macrophage, and neutrophils 2%; Gram stain and AFB stain negative with mycobacterial culture being negative. However, the arsenic level in the BAL fluid was 0.09 µg/g in both samples (normally lung may contain up to 0.01 µg/g of arsenic). [4]

We also sent the local drinking water used by the patient, hair, and nail of the patient for arsenic level determination. The drinking water, hair, and nail contained 0.18 mg/L (normal = up to 0.05 mg/L), 5.34 µg/g, 8.93 µg/g (normal up to 0.5 µg/g) of arsenic, respectively.
The patient again later presented to us around 7 months later with severe respiratory distress with bilateral pitting pedal edema. The patient had cyanosis, SpO$_2$ 86% at room air, tachycardia, angorged and pulsatile internal jugular vein. Systemic examination showed left parasternal heave and a loud pulmonary component of 2$^{nd}$ heart sound ($P_2$) with a closed split. The clinical profile was suggestive of right heart failure with pulmonary hypertension. Subsequent echocardiography revealed right atrial dilatation and right ventricular hypertrophy, grade 2 tricuspid regurgitation and mean pulmonary artery pressure of 42 mmHg. The patient was put on high flow oxygen, inhaled bronchodilators, and diuretic, by which she improved symptomatically. She is on regular follow-up in our OPD and no further exacerbations occurred till now over last 6 months. On follow-up, spirometry, 6 min walk test, and ABG did not reveal any significant improvement; but echocardiography showed favorable response in terms of mean pulmonary artery pressure (comparison done with reports at initial visit and reports at 14 months later i.e., around 6 months after starting treatment for pulmonary hypertension). FVC decreased from 1.34 l to 1.26 l; 6MWD almost remaining same with 1% desaturation; ABG showed pH 7.36, pCO$_2$ 48 mm Hg, pO$_2$ 80 mmHg.

Thus, we concluded that it was a case of DPLD with cor pulmonale in a case of chronic arsenic exposure.

Though lung biopsy is imperative to confirm the diagnosis but in our case it was not possible due to the poor functional status of the patient due to Pulmonary artery hypertension.

**DISCUSSION**

DPLDs represent a large number of conditions that involve the parenchyma of lung – the alveoli, alveolar epithelium, capillary endothelium, and spaces between those structures as well as the perivascular and lymphatic tissue.[6] Chronic arsenic poisoning or arsenicosis is a major health problem in eastern India and Bangladesh. Arsenic, a metalloid occurring naturally, is the 20$^{th}$ most abundant element in the earth’s crust,[7] and a component of more than 245 minerals. Humans are exposed to this toxic arsenic primarily from air, food, and water. Drinking water and natural groundwater may be contaminated with arsenic from arsenical pesticide, natural mineral deposits or improperly disposed arsenical chemicals, industrial effluent, and drainage problems.

Arsenosis is a multisystem disorder that may involve the skin, lung, gastrointestinal tract, cardiovascular, nervous, hematological, and endocrine system. Symptoms include weakness, paresthesia, cough, burning eye, pain abdomen, etc., However, skin manifestations are the most diagnostic. Hyperkeratosis of palms and soles, rain-drop like spotty pigmentation and depigmentation or diffuse melanosis affecting the whole body are the characteristic features. Diffuse thickening of palms and soles with or without nodular elevations are diagnostic of arsenic-induced keratosis.

Arsenosis may result in both malignant and nonmalignant lung diseases including obstructive airway disease, bronchiectasis, and bronchitis.[8] In a hospital-based study carried out on 29 cases of chronic arsenic toxicity with nonmalignant lung disease in Kolkata, West Bengal, obstructive lung disease was most prevalent being diagnosed in 17 (58.6%) cases.[9] However, still no confirmed case of DPLD due to arsenic, substantiated by demonstration of significantly high level of arsenic in BAL fluid as well as in hair, nail, and drinking water source has been reported in the literatures. Henceforth, it should be a learning curve for physicians particularly belonging to the highly endemic belt of arsenic to consider arsenicosis as a potential etiology in cases of DPLD with no identifiable cause. Careful attention to such cases may enlighten more evidence of association between these two conditions in near future.
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**Conflicts of interest**
There are no conflicts of interest.

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