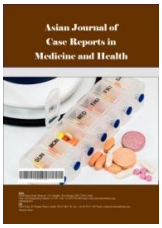

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Arsenicosis and Bronchiectasis: A Case Report

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Authors' contributions

This work was carried out in collaboration between both authors. Author RH collected patient data,drafting of manuscript and literatue searches. Author KR is the senior author who guided the patient care and approved manuscript for subimission. Both authors read and approved the final manuscript.

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Case Report

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ABSTRACT

Background: Arsenicosis is a multisystem disorder that may affect virtually every organs of the body. The lung involvement in arsenicosis includes bronchitis, obstructive airway disease, bronchiectasis, diffuse parenchymal lung disease and bronchogenic carcinoma.
Objective: To report a rare case of arsenicosis causing bilateral bronchiectasis in a nine year old girl.
Presentation of Case: We describe a 9-year-old girl, who developed bilateral bronchiectasis in association with chronic arsenicosis caused by long-term consumption of homeopathic medicines for drug-resistant epilepsy. To the best of our knowledge, only few cases of bronchiectasis in connection with confirmed arsenicosis has been reported.

Keywords: Arsenic; arsenicosis; bronchiectasis; pulmonary disease; toxicity.

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1. INTRODUCTION

Exposure to arsenic in humans can occur as an occupational hazard, environmental exposure, and through foods and drinks. Arsenic is a well-established cause of cancer of the lung, bladder, and skin [1]. Non-cancerous health effects of arsenic exposure involving different body systems have been described including the respiratory, cardiovascular, haematological, gastrointestinal, immune, dermal, reproductive and endocrine systems, as well as the central and peripheral nervous systems [2] .We describe a 9-year-old girl who developed bilateral lower lobe tubular bronchiectasis due to chronic arsenic exposure from homeopathic medicines prescribed for the treatment of epilepsy. To the best of our knowledge, no case of bronchiectasis in connection with confirmed arsenicosis has been reported.

2. CASE REPORT

A 9-year-old girl, a resident of Uttar Pradesh, northern India was referred to the Comprehensive Epilepsy Centre of Avitis Hospital, Palakkad, Kerala, southern India for evaluation for difficult to control epilepsy. The child was born by full term normal delivery. She has had epileptic seizures since early childhood for which she was receiving a combination of clobazam, oxcarbamazapine and levetiracetam. In addition to recurrent epileptic seizures, she had chronic cough during the previous one year, which prompted consultation at the Department of Pulmonology. The cough was associated with scanty mucoid expectoration. The cough was severe enough to disturb her night sleep and daily activities. There was associated exertional dyspnoea (MMRC grade 1). She denied symptoms of wheeze, allergic rhinitis, fever, joint pain, dry eye, or oral ulcers. There was no past history of pneumonia, tuberculosis or recurrent respiratory infections. No history of atopy. There was no history of any significant illness in her family members.

Upon clinical examination, she was alert, conscious, afebrile with a pulse rate of 88/min and regular, respiratory rate of 22/min, blood pressure of 100/60 mmHg, and SpO2 98% on room air. Auscultation of chest revealed bilateral coarse crepitations over the bilateral infrascapular areas. There was generalized darkening of skin, palmar and plantar keratosis and 'raindrop pigmentation' of the distal part of extremities (Fig. 1). Rest of the examination, including the neurological examination, was normal. On further inquiry, we came to know that she had been consuming homeopathic medicines as the treatment for epilepsy for the previous 6 years. The patient consumed following homeopathic medicines from 2014 to 2019 (Cuprumet, Kalibrometum, Constitutional, Gelsiminium, Beladona/ Brownia). When she developed the skin lesion during the previous one year, a dermatologist was consulted. The arsenic aetiology was suspected and she was advised to discontinue the homeopathic medicines which were discontinued in November 2019. The skin lesions have not progressed over the previous 6 months. She used to drink water from the household pipe water supply after using reverse osmosis filters. None of the family members or people in the neighbourhood suffered from similar dermatological problems.

Her chest radiograph did not reveal any abnormality. A high-resolution computed tomography (HRCT) of the chest revealed bilateral lower lobe tubular bronchiectasis with patchy collapse and consolidation of lingular segment with bronchial thickening in left lower lobe (Fig. 2). The spirometry showed mixed obstructive and restrictive pattern corroborating with bilateral bronchiectasis. The other laboratory reports including haemoglobin and total leucocyte and eosinophil counts, and renal, thyroid and hepatic functions were within normal range. Her serum IgE was in normal range, so were the serum globulins, and connective tissue serology. The sputum for acid-fast bacilli (AFB) and culture were non-contributory. The arsenic concentration in her hair was 5.91 microgram/gram, which is six times the upper permissible level (reference level <0.080 ug/g) and arsenic concentrations in her nails was 2.82 microgram/gram which is nearly three times the upper permissible level (reference level is <1ug/g). Additionally, she underwent a detailed evaluation for drug-resistant epilepsy with prolonged video-EEG monitoring and high-resolution brain magnetic resonance imaging (MRI). A diagnosis of MRI-negative focal epilepsy was made and the antiepileptic medicines were optimized.

For the pulmonary symptoms, she was treated with bronchodialators and chest physiotherapy. She had already discontinued the homeopathic medicines (the alleged source of arsenic). The patient and her family were counselled with regards to the hazards of chronic arsenic ingestion. After 3 months, her cough had ameliorated and the skin lesions also showed improvement (Fig. 3).

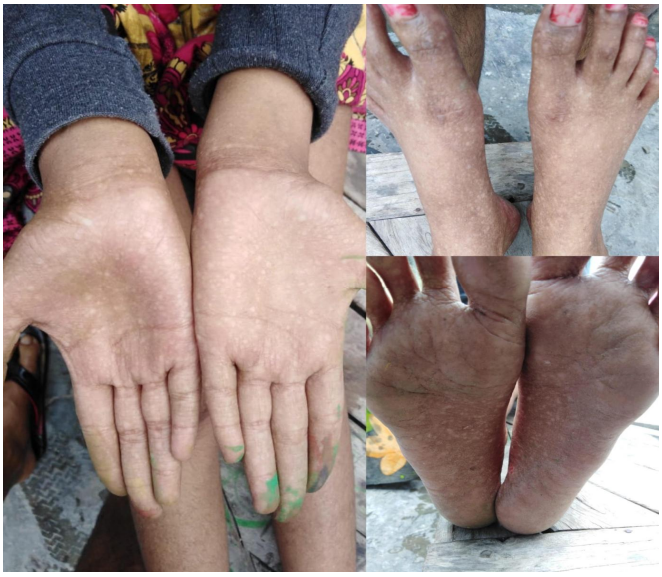

Fig. 1. Patient's photographs show plantar keratosis, and spotty pigmentation and depigmentation (rain-drop pigmentation) involving the palms, and dorsum and soles of the feet

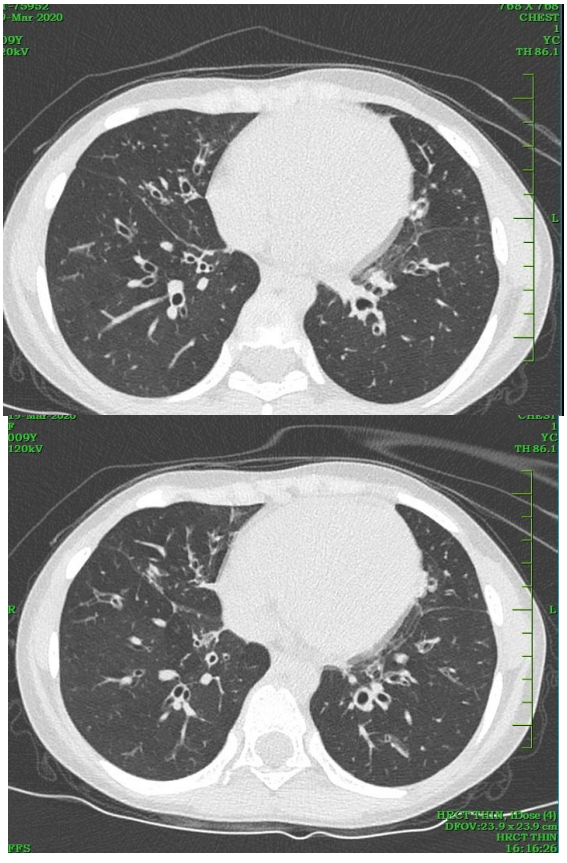

Fig. 2. The Chest HRCT of the patient showing bilateral lower lobe central tubular bronchiectasis with bronchial thickening in left lower lobe

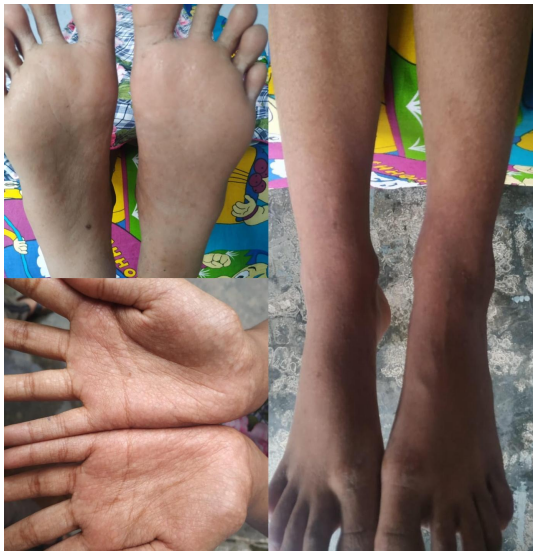

Fig. 3. Three months later, photograph shows decrease in the rain-drop pigmentation compared to Fig. 1

3. DISCUSSION

Our patient had the classical cutaneous manifestations of chronic arsenic intoxication such as hyperkeratosis of palms and soles, and spotty pigmentation and depigmentation affecting the whole body (rain-drop pigmentation), especially over the distal parts of extremities. The concentrations of arsenic well above the permissible levels in her hair and nail confirms the diagnosis of arsenicosis. The temporal relation between the evolution and resolution of her respiratory symptoms with the classical dermatological manifestations strongly supported arsenicosis associated bronchiectasis. Moreover,our patient did not have recurrent antecedent respiratory infections predisposing her for the development of bronchiectasis. In the absence of any history of environmental exposure, we consider the homeopathic medicines our patient took for several years for the treatment for epilepsy as the plausible source of the arsenic intoxication. This is further supported by the temporal relationship of the evolution and resolution of the dermatological manifestations to initiation and discontinuation of the homeopathic medicines.

Arsenic inactivates up to 200 enzymes, most notably those involved in cellular energy pathways and DNA replication and repair, and is substituted for phosphate in high energy compounds such as ATP [3]. Unbound arsenic also exerts its toxicity by generating reactive oxygen intermediates during their redox cycling and metabolic activation processes that cause lipid peroxidation and DNA damage [3]. In chronic arsenic ingestion, arsenic accumulates in the liver, kidneys, heart, and lungs and smaller amounts in the muscles, nervous system, gastrointestinal tract, and spleen [4].Though most arsenic is cleared from these sites, residual amounts remain in the keratin-rich tissues, nails, hair, and skin.

Most cases of acute arsenic poisoning occur from accidental ingestion of insecticides or pesticides. Prominent clinical manifestations of acute arsenic intoxication are nausea, vomiting, colicky abdominal pain, profuse watery diarrhoea, excessive salivation, acute psychosis, diffuse skin rash, toxic cardiomyopathy, and seizures. Haematological abnormalities, renal failure, respiratory failure, and pulmonary oedema may also occur [5]. Urinary arsenic concentration is the best indicator of acute arsenic poisoning [6,7]. Long-term arsenic toxicity leads to a multisystem disease and the most serious consequence of which is malignancy.

Pulmonary manifestations of chronic arsenicosis include bronchitis, malignancy, diffuse parenchymal lung disease, bronchiectasis, pulmonary artery hypertension and bronchial carcinoma [8]. Chronic bronchitis with or without obstruction are the common cause of mortality in many cases of chronic arsenic toxicity. Studies

from West Bengal, India drew attention to both restrictive and obstructive lung disease in arsenicosis [9]. The respiratory disease is more common in patients with the characteristic skin lesions of chronic arsenic toxicity [10]. The severity of respiratory involvement in arsenicosis is also found to be dose and duration related. The chronic obstructive pulmonary diseases accounted for the largest group of patients, followed by interstitial lung diseases, malignancies and bronchiectasis [11]. The pathogenesis of pulmonary involvement may be due to the activation of inflammatory mediators and the resultant lung injury [12].

Previous studies have shown that children were no less susceptible to the dermatological effects of arsenic than the adults living in the same communities although the incidence of arsenic-related skin manifestation vary depending on various factors, which include dose and duration of exposure, nutritional status of children, and ethnicity. The biotransformation of arsenic in humans occurs through the methylation process and the second methylation capacity in children is higher than adults, and hence children retain less arsenic in their body than adults does. But in reality, previous studies shows that other factors than age may also be related to methylation capacity in a child [13]. Chronic cough was complained of by 38.8% of children with skin lesions due to arsenicosis [14].

Many of the clinical manifestations of chronic arsenic toxicity are irreversible. There is no effective therapy for chronic arsenicosis; patients once affected may not recover even after further avoidance of exposure. Treatment for chronic arsenic intoxication is directed towards stoppage of arsenic exposure, averting disease progression, and general measures and symptomatic treatment [15]. Chelation therapy for chronic arsenic toxicity is thought to be the specific therapy for relief of systemic clinical manifestations and reduction of arsenic stores in the body, and in reducing subsequent cancer risk. Chelating agents like, dimercaptosuccinic acid, dimercaptopropane succinate and d-penicillamine have frequently been considered for treatment of chronic arsenic toxicity. However, their usefulness remains to be established. High protein diet possibly helps in clearance of inorganic arsenic by increased methylation. The various clinical manifestations should be treated symptomatically. Respiratory infection should be treated promptly because it aggravates breathlessness. Treatments with bronchodilators, mucolytics, smoking cessation, avoidance of dust are advised. Skin thickening of the sole and palm can be treated by local application of keratolytic ointment. Dyspeptic symptoms associated with chronic arsenicosis could be easily managed by use of H2 receptor blockers with/without prokinetic drugs. Stoppage of intake of arsenic contaminated water and intake of nutritious diets can reduce some of the symptoms of chronic arsenicosis. No specific drug for altering the natural history of the disease has yet been available. However, supportive and symptomatic treatment could help a lot to reduce the suffering of patients.

4. CONCLUSION

This case report illustrates a case of arsenic induced bronchiectasis in a pediatric patient, arsenicosis caused probably due to long term ingestion of homeopathic medicines and management of chronic arsenicosis.

CONSENT

As per international standard, parental written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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