Pulmonary Cavity due to Chronic Eosinophilic Pneumonia Associated with Arsenicosis.

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ABSTRACT

In developing country like India arsenic poisoning is a major public health problem. Association of chronic eosinophilic pneumonia and chronic diarrhea with arsenicosis is rare. Also pulmonary cavity formation in chronic eosinophilic pneumonia is very uncommon. A 44-year-old male patient, resident of an arsenic affected area was admitted for evaluation of chronic diarrhoea, persistent peripheral eosinophilia along with radiologically visible cavity in right upper zone. There were dermatological manifestations of arsenicism along with presence of noncirrhotic portal fibrosis and peripheral eosinophilia. On bronchoalveolar lavage study, eosinophil comprised 40% of total cellularity making the diagnosis of chronic eosinophilic pneumonia. After ruling out all possible causes of diarrhoea and chronic eosinophilic pneumonia, we came to conclusion that arsenic could be implicated as causative agent.

Keywords: Arsenicism, chronic diarrhoea, chronic eosinophilic pneumonia, noncirrhotic portal fibrosis

INTRODUCTION

Arsenicosis has become an important health hazard in countries like India due to ingestion of contaminated drinking water. Apart from skin, chronic ingestion of arsenic can affect various organs of body like liver, lung, nervous system, heart etc. There was conflicting reports regarding association of chronic eosinophilic pneumonia in patients of arsenicism. Chronic eosinophilic pneumonia presents classically radiologically with “photographic negative of pulmonary oedema”. We are reporting a patient of arsenicism presented to us with right sided pulmonary cavity due to chronic eosinophilic pneumonia.

CASE REPORT

A 44-year-old male from Eastern India was admitted with complaints of dry cough & low grade fever for seven months along with recurrent diarrhoea since childhood. Seven years ago, he had two episodes of upper GI bleeding due to variceal haemorrhage and underwent endoscopic sclerotherapy. At that time, liver biopsy was also done and it revealed histopathological features suggestive of noncirrhotic portal fibrosis (NCPF). There was no suggestive drug history causing eosinophilia in this patient. Clinical examination revealed moderate splenomegaly with absence of hepatomegaly and ascites. On dermatological examination, there

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was raindrop pigmentation over trunk and punctuate keratoderma over palm and sole (Figure 1).

Figure 1. Raindrop pigmentation visible over back with bowenoid keratosis.

Sputum for acid fast bacilli for consecutive 6 days, Gram stain and culture for pyogeny organisms and mycobacterium tuberculosis was negative. Blood for ANCA (Antineutrophil Cytoplasmic Antibody) and p-ANCA was negative. CT scan guided fine needle aspiration cytology from wall of radiologically visible cavity revealed marked eosinophilic infiltration of lung tissue without any evidence of malignancy & granuloma (Figure 3).

Figure 3. CT guided fine needle aspiration cytology showing eosinophilic infiltration of lung parenchyma.

Fibreoptic bronchoscopy was done and bronchoalveolar lavage (BAL) was sent for investigation. Cell count in BAL fluid was 650/mm³ & eosinophil comprised 40% of total cellularity. BAL fluid culture revealed no mycobacterial tuberculosis complex or fungal elements. Pulmonary function test was suggestive of mixed (both obstructive & restrictive) pattern. No ova, parasite & cysts were found in stool examinations on repeated occasions. Blood for antifilarial antibody was negative. Serum IgE was 1320 IU/ml which was above the normal range. There was no history suggestive of arthropathy, haematuria or mononeuritis multiplex or in other words that of Churg-Strauss syndrome. So, a diagnosis of chronic eosinophilic pneumonia was established. On evaluation of etiology of diarrhoea, a detailed history was taken. Stool frequency was three-four times per day, devoid of any blood or offensive smell without any episodes of constipation alternating with diarrhoea. Stool culture was done and no growth was found. Anti-tissue transglutaminase antibody and anti-endomysial antibody both were negative. Colonoscopic biopsy did not add any clue to diagnosis. Ultrasonography of abdomen showed normal sized liver, normal echo texture, mildly irregular margins. Gall bladder was anechoic, but walls were thickened 7.3 mm; showing multiple anechoic tortuous structures. Portal vein was 14 mm and there was marked thickening of wall of portal vein especially intrahepatic branches along with moderate splenomegaly with multiple echogenic specks noted specially at the poles. Overall, it was suggestive of NCPF. Upper G.I Endoscopy revealed grade-2 oesophageal varices along with multiple post-EST mucosal tags suggestive of mild portal hypertensive
gastropathy. Duodenal biopsies were also done which showed chronic duodenitis with no increase in intraepithelial lymphocytes. We thought that arsenic could be regarded as the agent responsible for chronic eosinophilic pneumonia, chronic diarrhoea and NCPF. For confirmation of diagnosis of arsenicosis, hair & nail sample for arsenic was sent which was found to be 0.78 mg/g & 1.34 mg/g respectively. So, a diagnosis of arsenicosis was made which was probably the causative factor behind the skin manifestations, NCPF, chronic diarrhoea and persistent peripheral eosinophilia. Patient was diagnosed as a case of arsenicosis associated with chronic eosinophilic pneumonia.

DISCUSSION

Arsenic contamination in drinking water has become an important public health hazard in developing countries like India especially in states like West Bengal. The normal amount of arsenic in hair is about 0.08 to 0.25 mg kg⁻¹ with 1 mg kg⁻¹ indicating the presence of excess arsenic and in nail is 0.43 to 1.08 mg kg⁻¹. Arsenicosis usually comes into notice after a careful dermatological examination because skin is early & commonly affected organ. The pigmentation of chronic arsenic poisoning usually is bilaterally symmetrical, finely flecked, “raindrop” pattern of hyperpigmentation or hypopigmentation that is particularly pronounced on the trunk and extremities. Keratoses is predominant on palms and plantar aspect of feet. As dermatological findings and the catchment area of patient were suggestive of arsenicosis, we measured the arsenic content of hair & nail which was 0.78 mg/g & 1.34 mg/g respectively (reported mean concentration of arsenic in hair & nail of affected persons in Chapra block are 0.73 mg/g & 1.28 mg/g respectively). Hence our patient satisfied the criteria required for diagnosis of arsenicosis.

Our patient was a previously diagnosed case of NCPF and it was supported by various examinations on present admission. Nearly 25-30% of all patients with portal hypertension in India who undergo surgery or sclerotherapy, have NCPF. In fact, the largest number of cases of NCPF (or a similar disease elsewhere) in the world has been reported from India. History of arsenic ingestion or residence in arsenic affected area should be actively searched or enquired in patients presenting with noncirrhotic portal fibrosis especially in countries like India.

In a study conducted in West Bengal about systemic manifestations of arsenic in absence of skin lesions, it was found that among males, the overall prevalence of diarrhoea in lowest and highest exposure group were 0.5 per 100 & 4 per 100 respectively. After ruling out all the possible causes of chronic diarrhoea in this patient, a conclusion was drawn that the arsenic might be the agent responsible for causation of chronic diarrhoea.

Peripheral eosinophilia may be observed in a significant percentage of arsenic affected area. In a study done in West Bengal, there was significant increase in eosinophil count (121.3%) of arsenic exposed subjects, as compared to controls. The increase in eosinophil count may be due to chronic respiratory symptoms encountered frequently in arsenic victims of West Bengal as that of other places. Another study in West Bengal showed that 33 out of 107 cases of chronic arsenic poisoning had evidence of pulmonary involvement. Out of 29 cases with non-malignant lung disease, 20 (68.9%) cases had obstructive pattern of lung involvement, 8 (27.0%) cases had mixed obstructive-restrictive pattern and only one (3.5%) had pure restrictive involvement. Mixed pattern detected in pulmonary function test in our patient was nothing unusual as this is often seen in person with arsenicosis. But the unusual thing was the detection of chronic eosinophilic pneumonia in this patient. The radiological abnormality in chronic eosinophilic pneumonia is usually symmetrical, affecting both lungs classically described as ‘photographic negative of pulmonary edema’. It can rarely present radiologically as presence of cavitations, consolidations, atelectasis or pleural effusions. Presence of cavity in this patient made us to think first about the possibility of common causes of cavity such as tuberculosis, malignancy, infections and Wegener’s granulomatosis. After excluding all common causes of cavity formation in this patient, presence of peripheral eosinophilia made us think about chronic eosinophilic pneumonia which may rarely present with cavity formation. No aetiology of chronic eosinophilic pneumonia could be established in this patient confirmatively. In the background of arsenicosis, arsenic could be regarded as the agent responsible for chronic eosinophilic pneumonia in this patient. There are reports of arsenic as a drug causing acute and chronic eosinophilic pneumonia. Conflicting reports are also available which suggest arsenic reduces eosinophilic infiltration of peripheral airway in patients of asthma. So, it might be a case of chronic eosinophilic pneumonia associated with arsenicosis. All the features of the patient like dermatological manifestations, chronic diarrhoea, non cirrhotic portal fibrosis and pulmonary cavity formation due to chronic eosinophilic pneumonia can be attributed to arsenicosis.

In conclusion, pulmonary cavity is a rare manifestation of chronic eosinophilic pneumonia. Chronic eosinophilic pneumonia can be associated with arsenicosis but whether it is implicated in its causation or not needs further research.
REFERENCES


