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Arsenic and non-malignant lung disease

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Many aquifers in various parts of the world have been found to be contaminated with arsenic at concentration above 0.05 mg/L. However reports of large number of affected people in India and Bangladesh are unprecedented. Characteristic skin lesions (pigmentation, depigmentation and keratosis) are the hallmark signs of chronic arsenic toxicity. Emerging evidences show that ingestion of arsenic through drinking water may also lead to non-malignant respiratory effects. Early report of non-malignant pulmonary effect of chronic ingestion of arsenic was available from studies in children in Chile as early as 1970. However on the basis of case studies, respiratory effect of chronic arsenic toxicity in adults following dinking of arsenic contaminated water in West Bengal was first reported in 1997. Epidemiological studies carried out in West Bengal on a population of 7683 showed that the prevalence odds ratio (POR) estimates were markedly increased for participants with arsenic induced skin lesions who also had high levels of arsenic in their current drinking water source (≥0.5 mg/L) compared with individuals who had normal skin and were exposed to low levels of arsenic (<0.05 mg/L). In participants with skin lesions, age-adjusted POR estimates for chronic cough were 7.8 for females (95% CI:3.1–19.5) and 5.0 for males (95% CI:2.6–9.9). In Bangladesh, similar study carried out on a population of 218 showed that the crude prevalence ratio for chronic bronchitis was found to be 10.3 (95% CI:2.4–43.1) for females and 1.6 (95% CI:0.8–3.1) for males. Reports of lung function tests were available from both hospital and population based studies. Results show evidences of restrictive, obstructive and combined obstructive and restrictive lung disease in different people having chronic lung disease associated with chronic arsenic toxicity. On the basis of clinical study, chest X-ray and HRCT done in Arsenicosis patients with features of chronic lung disease, the abnormalities observed were varied. Evidences of obstructive pulmonary disease (COPD), interstitial lung disease (ILD) and bronchiectasis were found in some of the cases. Results of studies carried out on people showing features of Arsenicosis due to drinking arsenic contaminated water provide evidence that arsenic is a potent respiratory toxicant, even following ingestion.

Keywords: Arsenic, lung disease, lung function tests, chronic bronchitis, COPD, ILD, bronchiectasis, West Bengal.

Introduction

Many aquifers in various parts of the world have been found to be contaminated with arsenic (As). Of these the most noteworthy occurrences are in large areas of India,[1–5] Bangladesh,[6–8] Taiwan[9,10] and Northern China.[11,12] Other Asian countries affected are Lao PDR, Cambodia, Mayanmar, Pakistan,[13] Nepal,[14,15] Vietnam.[16] Other countries having reports of significant arsenic contamination of ground water are, Hungary, Mexico, USA, Chile and Argentina.[17] In India over and above West Bengal, other states affected are Bihar,[18] Uttar Pradesh, Jharkhand and Assam.[19] Though chronic arsenic toxicity due to drinking of arsenic contaminated water has been reported from many countries, but reports of large number of affected people in West Bengal, India and Bangladesh are unprecedented. In West Bengal, arsenic contamination of ground water has been reported in 777 villages of 8 districts. It is suspected that about 6 million people are exposed to arsenic-contaminated drinking water (As level >0.05 mg/L) in 79 blocks of those 8 districts.[20]

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Non-malignant health effects of arsenic

Most reports of chronic arsenic toxicity focus attention on skin manifestations such as pigmentation and/or depigmentation affecting trunks and limbs and keratosis affecting hands and feet. Diffuse or spotty pigmentation, the initial nonmalignant dermatological effect of chronic arsenic intake can first appear within 6 months to 3 years of chronic arsenic ingestion at concentrations in excess of approximately 0.04 mg/kg per day. Lower exposure rates can also result in pigmentation after an interval of 5 to 15 years. Palmer-planter keratosis, the other principal non malignant cutaneous manifestation of chronic arsenic exposure, usually follows the appearance of pigmentation within a period of years.[21]

Reports from population based studies and clinical case reports relating to ingestion of inorganic arsenic in drinking

water suggest that arsenic also adversely affect multiple organ systems of human body. Systemic manifestations like peripheral neuropathy, hepatomegaly with non-cirrhotic portal fibrosis and peripheral vascular disease have frequently been reported in cases of chronic exposure to arsenic. Other systemic manifestations include non-pitting edema of hands, feet or leg, dyspepsia, diarrhea, anaemia, genaralised weakness and peripheral vascular disease like Blackfoot disease. Exposure to arsenic has also been associated with increased risk of diabetes mellitus, cardiovascular and cerebrovascular disease. Except for only report of affection of lung in children from Chile, not much information on pulmonary effect of chronic arsenic toxicity was available in the literature till the late 1990s.[17,21]

Arsenicosis and lung disease

Initial report of non-malignant pulmonary effect of chronic ingestion of arsenic was available from studies in children in Chile as early as 1970. Rosenberg conducted autopsies on five children manifesting characteristic features of chronic arsenic toxicity, including pigmentation and/or keratosis. Lung tissue was examined in 4 of the 5 children, with abnormalities found in each and 2 having pulmonary interstitial fibrosis with mild bronchiectasis.[22] A 1976 cross-sectional survey in Antofagasta examined 144 school children with arsenical skin lesions, and reported that chronic cough was complained of by 38.8% of children compared to 3.1% of children with normal skin.[23] In survey data collected between 1968 and 1972 in Antofagasta, Chile, Zaldiver reported that prevalence of cough and/or dyspnoea among 398 children correlated with mean drinking water arsenic concentrations.[24] In addition, they found the prevalence of bronchiectasis was 23 times higher and recurrent bronchopneumonia was 3.44 times higher in children with chronic arsenical dermatosis than in general population of Chilean children.[25]

Clinical case studies

A respiratory effect of chronic arsenic toxicity in adults following dinking of arsenic contaminated water in West Bengal was first reported by Guha Mazumder *et al.* in 1997. A total of 156 patients with arsenical skin lesion were investigated in a hospital at Kolkata. The arsenic contaminated water (0.05–3.2 mg/L) which they were drinking was drawn from subsoil water by hand pump from varying depths. The patient population also included 20 cases from south Calcutta who took water containing higher quantities of arsenic (5.05–14.2 mg/L) due to contamination of subsoil water by dumped waste of factory manufacturing Paris green (Copper acetoarsenate). Duration of contaminated water usually varied from 1–15 years, but in some cases it was lifelong. Symptoms of chronic cough, dyspnoea and heamoptysis were present in 57%, 37% and 8% of cases respectively. Evidences of chest signs such as rals and rhonchi

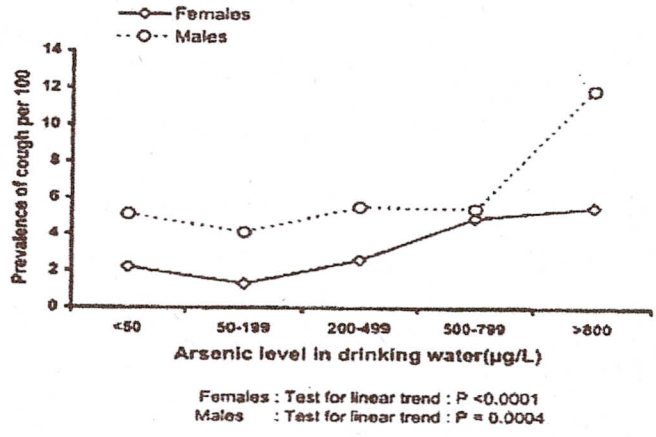

Fig. 1. Prevalence of cough per 100 in relation to arsenic exposure among nonsmokers.[28]

were present in 28.8% of cases.[26] In a study of 1774 cases of chronic arsenic toxicity living in 627 villages of the Inner Mongolia Autonomous Region of China, drinking arsenic-contaminated water (As concentrations 0.05–1.82 mg/L), 22.63% of cases showed respiratory system involvement.[27]

Epidemiological studies

A cross-sectional survey involving 7683 participants of all ages was carried out in South 24 Parganas, one of the severely arsenic-affected population, in West Bengal. The study was conducted between April 1995 and March 1996. The focus of the study was to ascertain prevalence of respiratory signs and symptoms over and above determination of the prevalence of dermatological effects of Arsenicosis like pigmentation and keratosis, the most common arsenic-related health effects. Participants were clinically examined and interviewed, and the arsenic content in their current

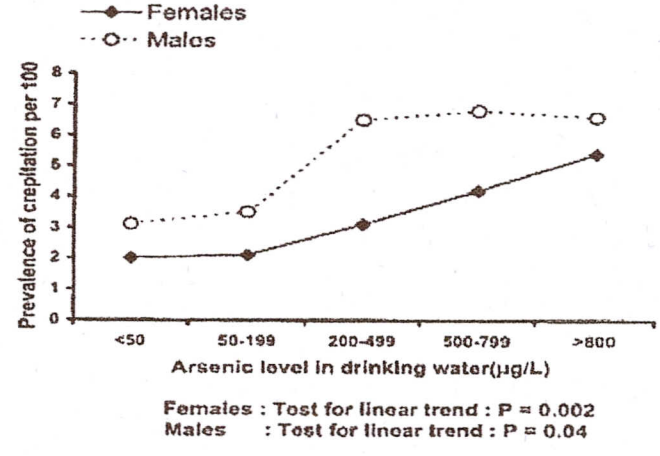

Fig. 2. Prevalence of crepitation per 100 in relation of arsenic exposure among nonsmokers.[28]

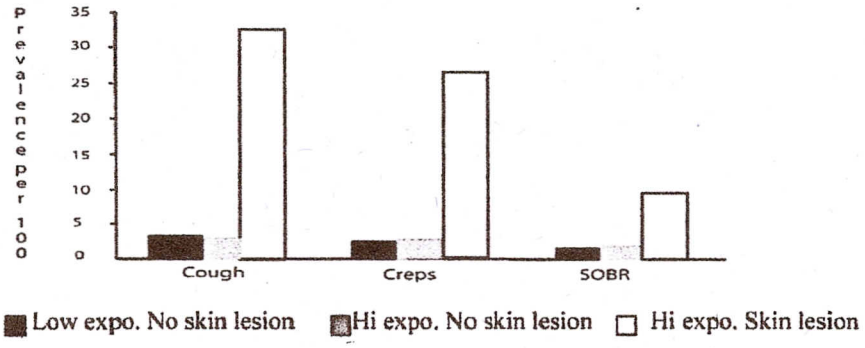

Fig. 3. Prevalence of respiratory diseases by exposure level and skin lesions status men. SOBR—Shortness of Breath.[29]

primary drinking water source was measured. There were few smokers and the analyses were confined to non-smoker (N = 6864 participants).

Although water concentration in the drinking water of the exposed population ranged up to 3.4 mg/L of arsenic, over 80% of participants were consuming water containing <0.5 mg/L. Strong exposure-response gradients were found for both pigmentation and keratosis in both sexes. Among both males and females, the prevalence of cough, (Fig. 1) shortness of breath, and chest sounds (crepitations and/or rhonchi) (Fig. 2) in the lungs rose with increasing arsenic content in drinking water.[28] These respiratory effects were most pronounced in individuals with high arsenic water concentrations who also had skin lesions.

Prevalence odds ratio (POR) estimates were markedly increased for participants with arsenic induced skin lesions who also had high levels of arsenic in their current drinking water source (>0.5 mg/L) compared with individuals who had normal skin and were exposed to low levels of arsenic (<0.05 mg/L). In participants with skin lesions, age-adjusted POR estimates for cough were 7.8 for females (95% CI:3.1–19.5) and 5.0 for males (95% CI: 2.6–9.9); for chest sounds POR for females was 9.6 (95% CI:4.0–22.9) and for males 6.9 (95% CI:3.1–15.0). The POR for shortness of breath in females was 23.2(95% CI:5.8–92.8) and males 3.7 (95% CI;1.3–10.6). The results of this epidemiological study provided evidence that long-term drinking of arsenic contaminated water can cause respirator effects (Figs. 3 and 4).[29]

A prevalence comparison study of respiratory effects among subjects with and without arsenic exposure through drinking water was conducted in Bangladesh. Exposed participants were recruited through health awareness campaign program. Unexposed participants were randomly selected, where tube wells were not contaminated with arsenic. A total of 218 individuals participated (94 exposed individuals exhibiting skin lesions; 124 unexposed individuals). The arsenic concentrations ranged from 0.136–1.0 mg/L. Only non-smokers without any history of asthma or tuberculosis were recruited and data on respiratory symptoms and signs were collected. The crude prevalence ratios for chronic bronchitis were found to be 1.6 (95% CI: 0.8–3.1) and 10.3(95% CI:2.4–3.1) for males and females, respectively. These results show that long-term arsenic exposure can cause respiratory effects.[30]

In a black-foot disease-prone area of Taiwan an ecological study of mortality showed that the Standardized Mortality Ratio (SMR) for "bronchitis" increased significantly relative to a nearby reference population (SMR = 1.53; 95% CI = 1.30–1.80).The authors observed that it was un-

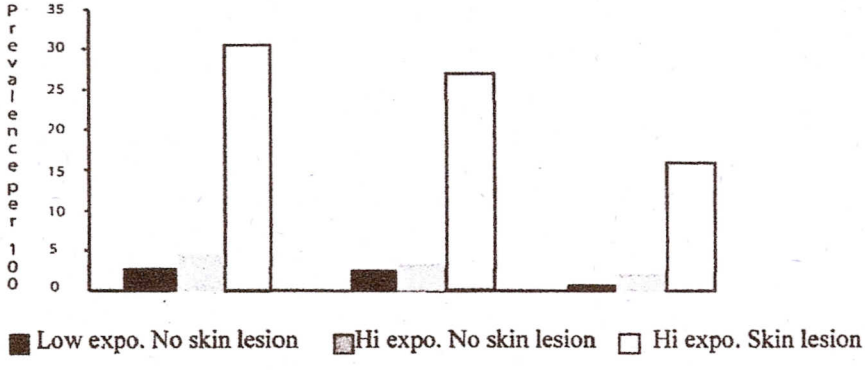

Fig. 4. Prevalence of respiratory diseases by exposure level and skin lesions status women. SOBR—Shortness of Breath.[29]

Table 1. Arsenic exposure and pulmonary function profiles compared between patients of chronic arsenic toxicity with and without pulmonary involvement[34]

	Lung change (n = 33)	*No Lung change (n = 74)*	*P value*
Age (years)	39.2 ± 9.81	32.2 ± 10.40	0.001
M:F	22:11	59:15	
Water As content (mg/L) (n)	0.77 ± 0.47 (30)	0.42 ± 0.42(54)	0.0009
FEVI (n)	39.9 ± 20.43(31)	82.2 ± 15.77	<0.0001
FVC (n)	54.8 ± 20.34(31)	87.0 ± 15.99	<0.0001
FEVI/FVC (n)	60.1 ± 15.78(31)	79.7 ± 6.05	<0.0001
PEFR (n)	34.6 ± 19.48(31)	62.5 ± 16.01(70)	<0.0001

likely that the differences in the rate of smoking account for the increased bronchitis mortality.[31] In a retrospective study carried out in Chile, though the COPD (Chronic Obstructive Pulmonary Disease) mortality was not increased overall, the Standardized Mortality Ratios (SMRs) for the years 1989–1993 were increased for both men and women aged 30–39 years (combining men and women, 10 deaths observed, 0.9 expected, SMR = 11.1, 95% CI:5.3–20.4, $P < 0.001$). It was also noticed that those in this age group were likely to have had their highest exposure to arsenic as children in the period 1955–1970, when arsenic levels in water in this Region particularly in the city of Antofagasta were in their peak.[32]

Further study compared mortality rates in Antofagasta in the period 1989–2000 with those of the rest of Chile, focusing on subjects who were 30–49 years of age at the time of their death. For the birth cohort born just before the high-exposure period (1950–1957) and exposed in early childhood, the SMR for bronchiectasis was 12.4 (95% CI:3.3–31.7; $P < 0.001$). For those born during the high-exposure period (1958–1979) with probable exposure in utero and early childhood, the corresponding SMR was 46.2 (95% CI:21.1–87.7; $P < 0.001$).[33] All these data suggest that exposure of arsenic through drinking water during infancy or in utero has pronounced effect on lung, greatly increasing subsequent mortality in young adults from non-malignant lung disease.

Lung function studies in arsenicosis

First report of lung function tests in patients suffering from chronic arsenic toxicity was available in the literature from studies carried out in 17 out of 89 patients having features of chronic lung disease and investigated in a hospital at Kolkata. Evidences of restrictive lung disease was found in 9 (53%) and combined obstructive and restrictive lung disease in 7 (41%) patients.[26]

In another study carried out in the same hospital where 107 subjects with (Cases) and 52 subjects without (Control) features of chronic arsenic toxicity were examined by spirometry. Thirty-three cases (30.8%) and 4 controls (7.6%) had features of chronic lung disease. Mean arsenic level in drinking water consumed by the cases was 0.77 ± 0.47 mg/L. Evidences of obstructive lung disease were found in 20 (68.9%), restrictive lung disease in 1 (3.5%) and combined obstructive and restrictive lung disease in 8 (27.6%) cases. Results of lung function tests of cases of chronic arsenic toxicity with and without clinical evidence of lung disease are given in Table 1.[34]

Lung function tests were carried out in a population-based case control study of arsenic-related skin lesions that were selected from the source population of 7,638 people as described in detail earlier.[28] This case-control study was carried out during 2000–2003 in which cases were selected having skin lesions and primary drinking water sources contained arsenic 0.05 mg/L–0.5 mg/L during the 1995–1996 study. Controls were selected from survey participants who did not have arsenic-related skin lesions when seen during the 1995–1996 survey, and whose main tube well-water source, contained an arsenic concentration of <0.05 mg /L. For each case, one control matched on age (within 5 years) and sex was randomly identified from all eligible non-cases. The study was confined to those participants of at least 20 years of age who completed the pulmonary function testing and for whom information on smoking was available. In this study 132 and 155 subjects fulfilled the criteria of cases, and controls.[35]

In this study pronounced decrements in lung function were observed in males with skin lesions, both non-smokers and smokers, as compared to those without skin lesions. Male smokers had, on average, lower mean residual values for spirometric parameters than male nonsmokers. The decreases in FEV_1 and forced vital capacity (FVC) in male nonsmokers with skin lesions as compared with nonsmoking men without skin lesions were 157.3 mL (95% CI: −24.7, 339.2) for FEV_1 and 188.5 mL (95% CI: 0.6, 376.3) for FVC. In male smokers, the decreases were 271.1 mL (95% CI: 158.0, 384.2) for FEV_1 and 304.1 mL (95% CI: 180.1, 428.1) for FVC. Among women, the respective reductions were 63.2 mL (95% CI: −31.8, 158.2) for FEV_1 and 101.5 mL (95 percent CI: −8.8, 211.8) for FVC. Decreases in FEV_1 and FVC related to increased water arsenic concentration were observed among men; reduction in mean values from low exposure (arsenic level < 100 μg/L) to high exposure

Table 2. Results from multivariate linear regression analysis of lung function and arsenic-related skin lesions in men (n = 178), with adjustment# for age, height, and smoking, West Bengal, India, 1998–2000.[35]

Variables	FEV1+ (mL)	95% CI+	*P* value*	FVC+ (mL)	95% CI	*P* value*	FEV_1/ FVC	95% CI	*P* value*	FEF^{+}_{25-75} (mL/sec)	95% CI	*P* value*
Skin lesion	−256.2	−398.4, 113.9	<0.001	−287.8	−440.8, 134.9	<0.001	−0.025	−0.05, −0.002	0.03	−259.1	−510.6, −7.6	0.04
Age$^{\$\$}$ (years)	−23.6	−28.8, −18.5	<0.001	−21.3	−26.9, −15.8	<0.001	−0.003	−0.004,−0.002	<0.001	−40.4	−49.7,−31.2	<0.001
Height (cm)	34.4	23.6, 45.2	<0.001	43.3	31.7, 54.9	<0.001	0.09	−0.09, 0.26	0.3	24.6	5.5, 43.7	0.012
Smoking$^{\$}$	−156.4	−316, 3.2	0.055	−119.7	−291.4, 52.0	0.2	−0.026	−0.05, 0.0006	0.045	−231.2	−513, 50.9	0.12

#The following variables were added to the model one by one and were not found to confound the association with skin lesions: weight, occupation (service, farmer, other), education (no formal education, primary, secondary or higher), and type of house (mud, mixed materials, brick).

+FEV_1, forced expiratory volume in 1 second, CI, confidence interval FVC, forced vital capacity; FEF_{25-75}, forced expiratory flow between 25% and 75% of forced vital capacity.

*Two-tailed.

$^{\$\$}$ Continuous variable.

$^{\$}$Smoking was defined as ever smoking versus never smoking. Different smoking variables, including pact-years of smoking, were also incorporated into the models but had no effect on the skin lesion result.

(arsenic level ≥400 μg/L) were 194.7 mL (95% CI: 35.5, 353.9) for FEV_1 and 83.8 mL (95% CI: −93.8, 261.5) for FVC in non-smokers and 226.1 mL (95% CI: 45.2, 407.0) for FEV_1 and 247.6 mL (95% CI: 58.3, 436.9) for FVC in smokers. Among women, the respective reductions were 28.5 mL (95% CI: −71.3, 128.2) for FEV_1 and 7.5 mL (95% CI: −122.4, 137.5) for FCV.

In the multivariate linear regression analyses stratified by sex and adjusted for age, height, and smoking, lung function was significantly decreased for signs of arsenic-related skin lesions among men, with a reduction in FEV_1 of 256.2 mL (95% CI : 113.9, 398.4, *P* value: <0.001) and in FVC of 287.8 mL (95% CI : 134.9, 440.8, *P* value: <0.001) (Table 2). To further investigate the effects of ingested arsenic on flows the FEV_1/FVC ratio and the forced expiratory flow between 25% and 75% of forced vital capacity (FEF_{25-75}) were investigated and significant reductions related to the presence of skin lesions consistent with the findings for FEV_1 and FVC in men were found.

Reductions were also observed related to smoking in FEV_1 (156.4 mL; 95% CI: −3.2, 316.0, *P* value: 0.055) and FVC (119.7 mL: −52.0, 291.4, *P* value: 0.2) but the effect size was smaller than for presence of skin lesions. Using arsenic levels in water as a measure of exposure instead of skin lesions, significant decreases were found in FEV_1 of 45.0 mL (95% CI : 6.2, 83.9, *P* value: 0.02) and in FVC of 41.4 mL (95% CI: −0.7, 83.5, *P* value: 0.054) in men per 0.1 mg/L increase of arsenic. Potential confounders such as weight, type of house, education and occupation were assessed in the multivariate models but did not change the estimates for skin lesions or arsenic in water. Interestingly, in women estimates for skin lesions or arsenic in water did not indicate a strong relation with lung function.[35]

The pathophysiologic mechanism by which ingested arsenic leads to impairments of lung function and increased respiratory symptoms is yet to be understood and needs further investigation. The decreases in FEV_1 and FVC that were observed in men were of similar magnitude suggesting a restrictive (e.g., lung fibrosis) disease.

Radiological studies in arsenicosis

In a hospital-based study carried out in Kolkata 29 cases of chronic arsenic toxicity with non-malignant lung disease were investigated with chest X-ray in all.[34] Four of the cases with restrictive/mixed pulmonary changes, and one with a clinical suggestion of bronchiectasis, were also investigated with HRCT (high resolution computerized tomography). Only those patients who fulfilled at least 2 of the following 3 criteria for chronic arsenic toxicity were included: (a) evidences of drinking water with arsenic level >0.05 mg/L for at least 3 years; (b) presence of raindrop mottled skin pigmentation in the trunk and limb with or without palmoplanter keratosis; and (c) hair or nail arsenic level above control values.

On the basis of clinical evaluation, chest X-ray and HRCT (done in 5), the patients were diagnosed to have obstructive pulmonary disease (COPD) in 17 (58.62%), bronchiectasis in 3 (10%) and interstitial lung disease (ILD) in 9 (31.2%). ILD was diagnosed by HRCT in 4 and by chest radiography in 5.[34]

A population based study was carried out to evaluate the association between arsenic ingestion and the presence of radiographic abnormalities of the lung using HRCT. The subjects in this study were selected from the same individuals who participated in a 1995–1996 population-based cross-sectional survey of arsenic-caused skin lesion.[28] All participants in the previous cross-sectional study who had arsenic-caused keratosis or pigmentation and who were thought to be consuming water with arsenic concentrations above 0.4 mg/L at the time of the cross-sectional

found

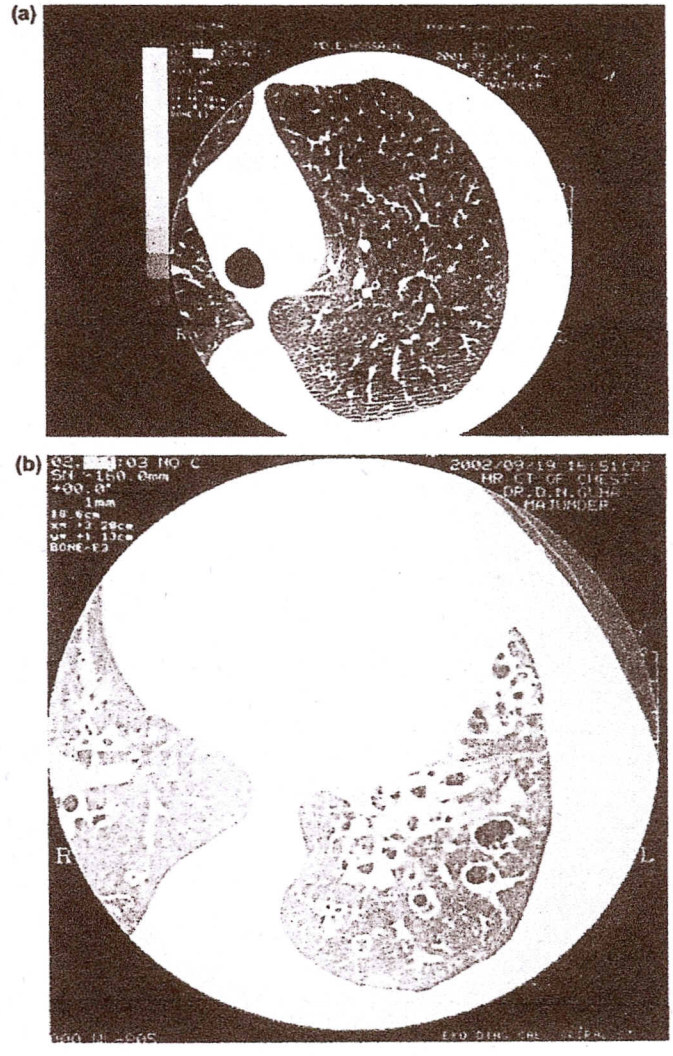

Fig. 5. HRCT picture of (a) a control subject showing normal lung architecture, and of (b) a patient suffering from chronic arsenic toxicity showing bronchiectasis.

survey were selected for this study. A randomly selected group of subjects without skin lesions and with recorded arsenic exposures less than 0.05 mg/L at the time of the cross-sectional survey, matched to skin lesion cases by age and gender, were chosen as a comparison group.

Subsets of both the groups who reported chronic cough (more than 3 months per year for at least 2 years) were referred to a tertiary referral center in Kolkata for HRCT. Initial review of the CT scans suggested that bronchiectasis was a frequent finding (Fig. 5). The severity of bronchiectasis in each lobe was ranked on a scale of 0 to 4 using a modification of the system described by Lynch *et al.*[36] Briefly, a 5-point grading system was employed : 0 = no bronchiectasis; 1 = mild bronchiectasis (non-tapering cylindrical internal bronchial diameter 1.5–3 times the diameter of the accompanying artery); 2 = moderate bronchiectasis (non-tapering cylindrical internal bronchial diameter more than 3 times the diameter of the accompanying artery); 3 = varicose bronchiectasis; and 4 = cystic bronchiectasis. The lungs were divided into 6 lobes (considering the lingual as a separate lobe) and a score was assigned to each lobe. A single bronchiectasis severity score was assigned to each subject by summing the bronchiectasis scores from each lobe of each lung.

Thirtythree (31%) subjects with skin lesions and 18 (12%) subjects without lesions reported chronic cough for more than 3 years (OR = 3.2; CI = 1.7–6.1). Of these 27 subjects with skin lesions and 11 of the subjects without lesions agreed to travel to Kolkata for HRCT. Overall, the participation rate was 82% in subjects with skin lesions and 61% in subjects without skin lesions. For those subjects who underwent HRCT, the average bronchiectasis severity score was 3.4 (SD ± 3.6) in subjects with skin lesions and 0.9 (SD ± 1.6) in subjects without lesions. Only 1 (9%) of the 11 subjects without skin lesions had a bronchiectasis severity score greater than 2, while 14 (52%) of the 27 skin lesion subjects had bronchiectasis severity scores greater than 2.

The unadjusted odds ratio for bronchiectasis was 10 (CI = 2.9–35) in subjects with arsenic-caused skin lesions compared with subjects having no lesions. The corresponding adjusted odds ratio was 10 (2.7–37). The adjusted odds ratio was 13 (2.6–62) in men and 6.1 (0.6–62) in women (Table 3).[37]

This study was the first investigation of HRCT findings in a population exposed to high levels of arsenic in drinking water. In this study, the authors found that persons with arsenic-caused skin lesions have a 10-fold higher rate of bronchiectasis on HRCT. Given the large magnitude of the relative risk estimate they identified, this association is not likely to be due to chance. The highly characteristic skin lesions diagnosed in this study are known to result from the consumption of arsenic-contaminated drinking water. The findings therefore provide evidence that long-term ingestion of arsenic results in increased risks of non-malignant pulmonary disease, in particular, bronchiectasis.

Table 3. Associations of arsenic-caused skin lesion with bronchiectasis found on CT.[37]

	*Bronchiectasis**		*Unadjusted OR (95% CI)*	*Adjusted OR+ (95%CI)*
	Yes	*No*		
All subjects				
No skin lesions++	3	140	1.0	1.0
Skin lesions	18	84	10.0 (2.9–35.0)	10.1 (2.7–37.1)
Men				
No skin lesions++	2	76	1.0	1.0
Skin lesions	15	52	11.0 (2.4–50.0)	12.6 (2.6–62.2)
Women				
No skin lesions++	1	64	1.0	1.0
Skin lesions	3	32	6.0 (0.6–60.0)	6.1 (0.6–61.6)

*Only those subjects with chronic cough underwent CT. Thus, bronchiectasis was defined as present when a subject had both chronic cough and bronchiectasis on CT. Bronchiectasis was defined as absent in subjects who did not have chronic cough and in subjects with chronic cough who did not have bronchiectasis on CT.

+Adjusted for age (year), gender, smoking (ever smoker versus never smoker), and self-reported history of physician-diagnosed tuberculosis (yes or no).

++Reference category

Conclusion

The results of the studies presented provide evidence that arsenic is a potent respiratory toxicant, even following ingestion. The results show that consuming water containing high levels of arsenic may lead to increased risks of pulmonary disease, characterized not only by chronic bronchitis but also alteration of pulmonary function as evident by decrease of FEV1 and FVC. The abnormalities appear to be varied and include obstructive, restrictive and combined obstructive and restrictive lung disease. Further, HRCT reports demonstrated high incidence of bronchiectasis in chronic arsenic exposed people associated with skin manifestations. The current studies included information only on adult subjects.

Information from exposed regions in Chile however, suggest that children may be particularly susceptible to the respiratory effects of drinking water arsenic. Future research is needed on the mechanisms of the pulmonary effects of arsenic, and on population susceptibility including the effects of childhood exposures. In view of the worldwide contamination of many private wells with arsenic, and the growing body of evidence linking arsenic ingestion to lung disease, patients with chronic respiratory disease, including those with bronchiectasis, should be asked if they are consuming drinking water from a private well. And if they are, this well should be tested for inorganic arsenic.

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