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Chronic Arsenic Toxicity in West Bengal — The Worst Calamity in the World

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Since 1983 large number of people are being encountered with arsenic toxicity due to drinking of arsenic contaminated water (0.05-3.2 mg/l) in 6 districts of West Bengal. Clinical and various laboratory investigations were carried out on 156 patients to ascertain the nature and degree of morbidity and mortality that occurred due to chronic arsenic toxicity. All the patients studied had typical rain drop like skin pigmentation (being inclusion criteria) while thickening of palm and sole were found in 65.5% patients. Other features included weakness (70%), gastro-intestinal symptoms (58.6%), involvement of respiratory system (57.08%) and nervous system (50.6%). Lung function tests showed restrictive lung disease in 53% (9/17) and combined obstructive and restrictive lung disease in 41% (7/17) of patients. Abnormal electromyography was found in 34.8% (10/29) and altered nerve conduction velocity in 34.8% (10/29) of cases. Enlargement of liver was found in 120 cases (76.9%) while splenomegaly in 31.4% cases. Liver function test showed elevated globulin level in 15.8% and alkaline phosphatase in 51.3%, alanine amino transferase (ALT) in 11.8% and aspartate amino transferase (AST) in 27.6% of cases. Evidence of portal hypertension was found in 33.3% patients. Liver biopsy reports of 45 patients showed non-cirrhotic portal fibrosis in 41, cirrhosis in 2 and normal histology in 2 cases. There was no correlation between the quantity of arsenic taken through water and the level of arsenic in hair, nail, liver tissues and the degree of fibrosis. There were 5 deaths of which one had skin cancer. The various non-cancer manifestations which were observed in these patients were much more severe than those reported in similar cases in other parts of the world.

ARSENIC in ground water above permissible limit (0.05 mg/l) has been detected in 6 districts of West Bengal viz, Malda, Murshidabad, Nadia, Burdwan and North and South 24-parganas[1]. First report of chronic arsenic toxicity was made by Garai *et al*[2] in 1984. At present, it is a well known environmental health hazard in the southern part of rural West Bengal[3-5]. A survey carried out in the affected districts suspected that about 8,00,000 people in an area of 3400 sq km in West Bengal are exposed to arsenic contaminated water[6]. Previously the largest number of cases of chronic arsenic toxicity have been reported from Taiwan, out of 40,421 cases surveyed 18.4% showed evidence of pigmentation while 7.1% of people had evidence of keratosis[7,8].

Patients from the affected areas have been treated at our institute since 1983. This paper presents data on the basis of studies carried on patients attending the hospital during the last 10 years.

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MATERIAL AND METHOD

A total of 156 patients, mostly coming from rural areas of the 6 districts of West Bengal were studied clinically and by relevant investigations. The arsenic contaminated water (0.05 to 3.2 mg/l) (Fig 1) which they were drinking was drawn from subsoil water by hand pump from varying depths (20-80 m). The patient population also included 20 cases from South Calcutta drinking water containing higher quantity of arsenic (5.05 to 14.2 mg/l) due to contamination of subsoil water by dumped waste of a factory manufacturing Paris green (copper acetoarsenite). Duration of intake of contaminated water usually varied from 1 to 15 years, but in some cases it was life long.

The investigations included routine blood, urine and stool examinations, liver function test (LFT), chest x-ray, ECG, blood sugar, urea, creatinine and viral markers for HBsAg by enzyme linked immunosorbent assay (ELISA). Liver biopsy of 45 hospital admitted cases was carried out for histological examination. Degree of hepatic fibrosis was graded into grade I to IV according to its severity[4]. Nerve conduction velocity (NCV) and electromyography (EMG) were carried out on 29 patients with neurological symptoms while lung function tests were done on 17 patients having feature of lung disease. Upper gastro-intestinal endoscopy was done on 60 patients sus-

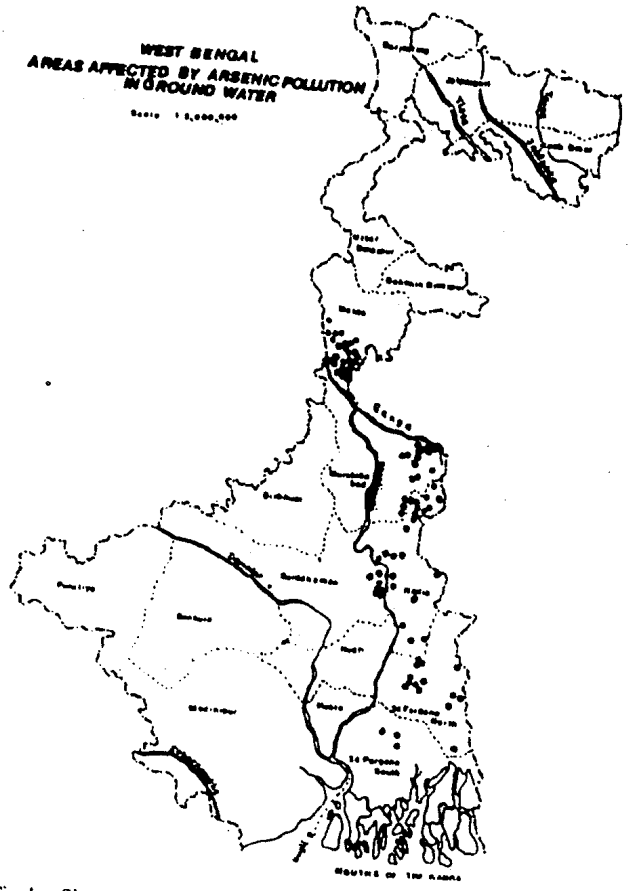

Fig 1—Showing Areas Affected by Arsenic Pollution in Ground Water

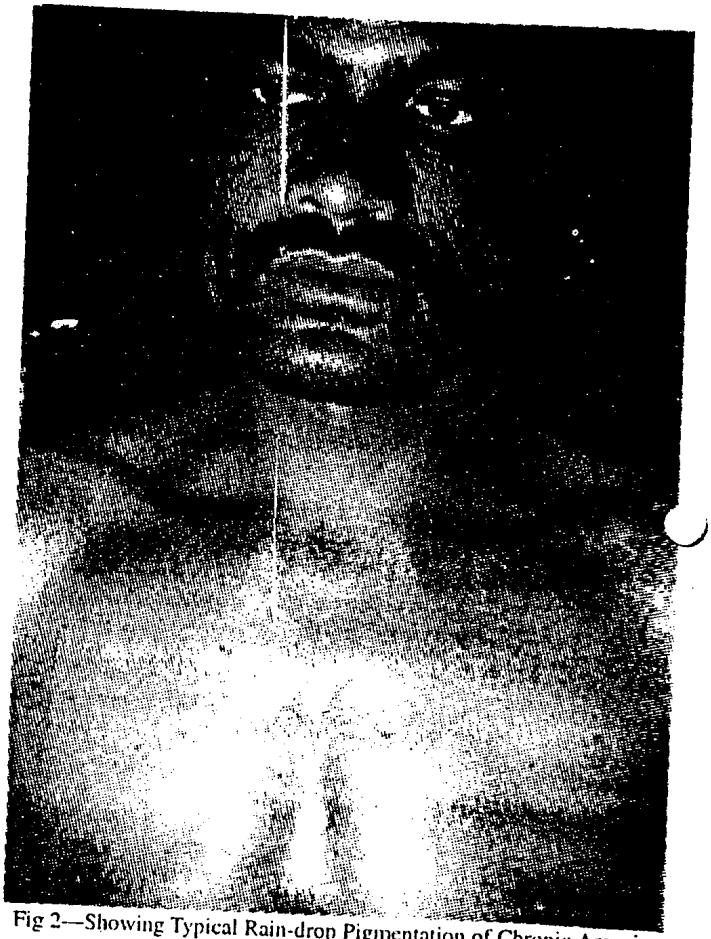

Fig 2—Showing Typical Rain-drop Pigmentation of Chronic Arsenic Toxicity

pected to have portal hypertension. Skin biopsy was done on 2 cases suspected to be suffering from skin cancer. Arsenic level in hair, nail and liver was detected by neutron activation analysis while its concentration in water consumed by the patients were estimated by atomic absorption spectrophotometry[6]. The patients were given symptomatic treatment and asked to drink arsenic free water and followed-up for variable periods.

Observations

Demographic data of the patients investigated are given in Table 1. Prevalence of various clinical features of 156 patients studied are given in Table 2. Though pigmentation (Fig 2) was seen in all (being an inclusion criteria) cases, keratosis (Figs 3, 4) was found in 96 patients (61.5%). Skin cancer was detected in 2 cases (1.3%). Weakness was a predominant symptom (70%) while anaemia was present in 47% of cases. Abdominal symptoms like pain abdomen and diarrhoea were found in 60 (38.4%) and 51 (32.6%) cases respectively. Lung function tests carried out on 7 patients showed features of restrictive lung disease in 9 (53%) and combined obstructive and restrictive lung disease in 7(41%). Evidence of polyneuropathy was found in 79 cases (50.6%).

Table 1—*Showing Age and Sex Distribution of 156 Patients with Chronic Arsenic Toxicity*

Age in years	Male	Female	Total
Below 10	2(1.6%)	0	2
10 to 20	31(24.8%)	3(9.7%)	34
21 to 40	71(56.8%)	21(67.7%)	92
41 to 60	16(12.8%)	6(19.3%)	22
Above 61	5(4.0%)	1(3.2%)	6
Total	125(100%)	31(100%)	156

Table 2—*Showing Clinical Features of 156 Cases Studied*

Symptoms	No of cases	Signs	No of cases
Weakness	110(70.5%)	Pigmentation	156(100.0%)
Paresthesis	74(47.4%)	Keratosis	96(61.5%)
Cough :	89(57.0%)	Anaemia	74(47.4%)
(i) with expectoration	53(33.9%)		
(ii) without expectoration	36(23.1%)	Hepatomegaly	120(76.9%)
Haemoptysis	8(5.1%)	Splenomegaly	49(31.4%)
Dyspnoea	37(23.7%)	Ascites	5(3.0%)
Headache	32(20.5%)	Pedal oedema	18(11.5%)
Nausea	17(10.9%)	Signs of lung disease	45(28.8%)
Pain abdomen :	60(38.4%)	Signs of polyneuropathy	21(13.4%)
(i) epigastric	39(25.0%)		
(ii) paraumbilical	21(13.4%)		
Diarrhoea	51(32.6%)		
Burning of the eyes	69(44.2%)		

tory disease as observed in patients with chronic arsenic toxicity in West Bengal have not been reported previously. Although some reports of liver damage caused by chronic arsenic toxicity were available in the literature earlier[10-13], few reports emphasised the lesion to be non-cirrhotic. Recent report of 8 cases[13] with a review of 15 cases from the literature and our previous published data of 13 cases[4] highlight that non-cirrhotic portal hypertension do occur in chronic arsenic toxicity. It was interesting to observe hepatomegaly occurring in a very high percentage (77%) of cases of chronic arsenic toxicity. Further NCPF on liver histology was found in most of the cases (92%) in whom liver biopsy could be done. However, the LFT did not show gross abnormality in most of the patients. Though incidence of NCPF was found to be high among patients having liver enlargement, portal hypertension occurred in smaller number of cases (33.3%).

The high incidence (57%) of respiratory disease among the patients with chronic arsenic toxicity caused by drinking arsenic contaminated water needs special attention. LFT carried out on a sample of 17 patients showed predominant occurrence of interstitial lung disease with or without airway obstruction. Only two reports are available in the literature of effects of the respiratory system due to exposure of arsenic *via* routes other than inhalation. A high frequency of chronic cough and bronchopulmonary disease were reported by Borgono *et al*[14] among 180 inhabitants of Antafagosta, Chile associated with drinking of arsenic contaminated water. Diffuse interstitial fibrosis was reported in post-mortem reports of 2 out of 5 children from the same area[12].

Though peripheral vascular disease has been described from Taiwan and Chile[7,8,14] none of our patients showed any manifestation. Neuropathy characterised by abnormal EMG and hearing loss as observed by us have also been reported by other workers[15] while diminished NCV was observed by Hotta[16]. However, such effects were not reported in studies from Argentina, Chile or Taiwan[17]. It appears that there is variation in clinical expression of chronic arsenic toxicity among people from different geographical regions of the world. Though nutritious diet and arsenic free water helped relief of symptoms in many cases, some still deteriorated. It is of utmost necessity to evaluate a rational treatment programme so that people showing early signs of chronic arsenic toxicity could be prevented from developing disabling complications.

Arsenic concentration is generally highest in ground water, especially where geochemical condition favours arsenic dissolution. High arsenic concentrations have been reported in water supply wells of Taiwan (up to 1.82 mg/l)[7], Hungary (exceeding 0.1 mg/l)[18], Mexico (exceeding 0.4 mg/l)[19] and United States (exceeding 0.1 mg/l)[20]. High arsenic concentration had also been reported in canals in Chile (up to 0.8 mg/l)[21] and Argentina (exceeding 0.25 mg/l)[22]. But nowhere such large number of people were found to be exposed to arsenic contaminated water, nor such severe systemic disease manifestations were expressed.

From the reports available so far it transpires that the arsenic contaminated ground water in West Bengal is generally limited in the intermediate acquifers. Water in the upper acquifers and the deeper acquifers below 100 m (except in isolated places in Murshidabad district) is free of arsenic contamination. The source of arsenic is considered to be geological[23]. However, the erratic distribution of arsenic in gorund water in the affected areas points to complex geological situation. The subsurface geology in the affected areas of West Bengal is yet to be well understood. The geochemical mechanism of "As" leaching also needs to be thoroughly investigated. Further there is a need for a good population exposure assessment to characterise the magnitude of the public health problem and to link exposure to outcome in epidemiological studies. A concerted effort is therefore needed by setting up study groups involving, environmental scientists, chemists, geologists and geohydrologists, engineers, medical and social scientists to understand the cause and effect relationship of the problem. Awareness of medical practitioners is vitally important as they are the people who need to be primarily involved in clinical identification, medical care and follow-up of patients with arsenic lesions.

Addendum :

Since submission of this paper another 64 patients from severely affected areas have been investigated. Out of these 3 patients showed evidences of peripheral vascular disease in the form of Raynaud's phenomena and gangrene of the toes.

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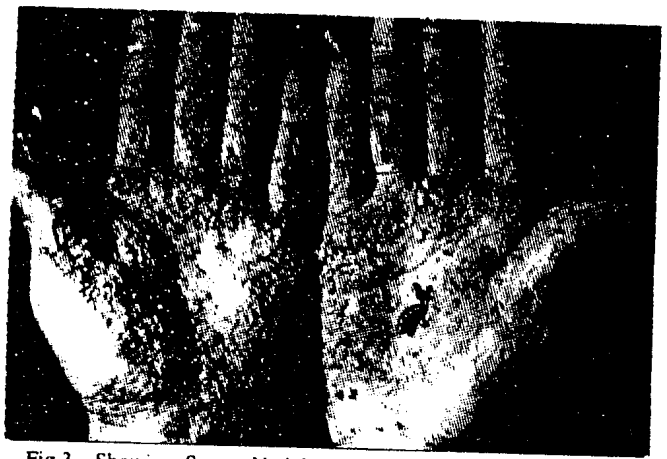

Fig 3—Showing Severe Nodular Keratosis of Hand with Keratotic Horn at the Centre in Chronic Arsenic Toxicity

Objective evaluation of neuronal involvement could be done on 29 patients. Of these, abnormal EMG was found in 10 (30.8%) and altered NCV and EMG in 11 cases (38%). Perceptive hearing loss was found in 2 cases.

Enlargement of liver was found in 120 (76.9%) cases and was palpable 2-6 cm below the costal arch. Spleen was palpable 1.5 to 8 cm below the costal arch in 41 cases (31.4%) while ascites was present in 5 cases (3%). LFT could be done in 76 patients. Abnormal globulin (range 3.5 to 4.8 g/dl) level and alkaline phosphatase (range 200 - 1206 IU/l) value were found in 12 (15.8%) and 39 (51.3%) cases respectively. Significant elevation of alanine amino transferase (ALT) (range 40-210 IU/l) and aspartate amino transferase (AST) (range 40-180 IU/l) was found in 9 (11.8%) and 21 (27.6%) cases respectively. HBsAg was present in serum in 8 cases (10.5%). Biopsy reports were available from 45 patients. Non-cirrhotic portal fibrosis (NCPF) was found in histology in 41 cases and cirrhosis in 2 cases while normal histology was observed in 2 patients. The liver histology of NCPF was characterised by expansion of portal zone of varying degrees. The fibrosis in the liver was mostly found to be mild (grade I, 53.6%, grade II, 29.6%) while moderate to severe fibrosis was found in smaller number of cases (grade III, 9.75% and grade IV, 7.31%). Portal hypertension was found in 52 cases (33.3%) as evidenced by splenomegaly and/or oesophageal varices, However, only 3 of these patients had haematemesis and melaena.

Maximum arsenic content of liver was found to be 6 mg/kg though it was undetectable in 6 out of 21 samples tested (mean 1.39±0.3 mg/kg, control value 0.16±0.04 mg/kg). Arsenic level in hair and nail of the patients (26 cases tested) was found to be 16.29±3.75 mg/kg and 42.72±5.92 mg/kg (control values 0.15±0.35 mg/kg and 0.34 ± 0.2 mg/kg) respectively. There was no correlation between quantity of arsenic taken through water and the level of arsenic in hair, nail and liver tissues.

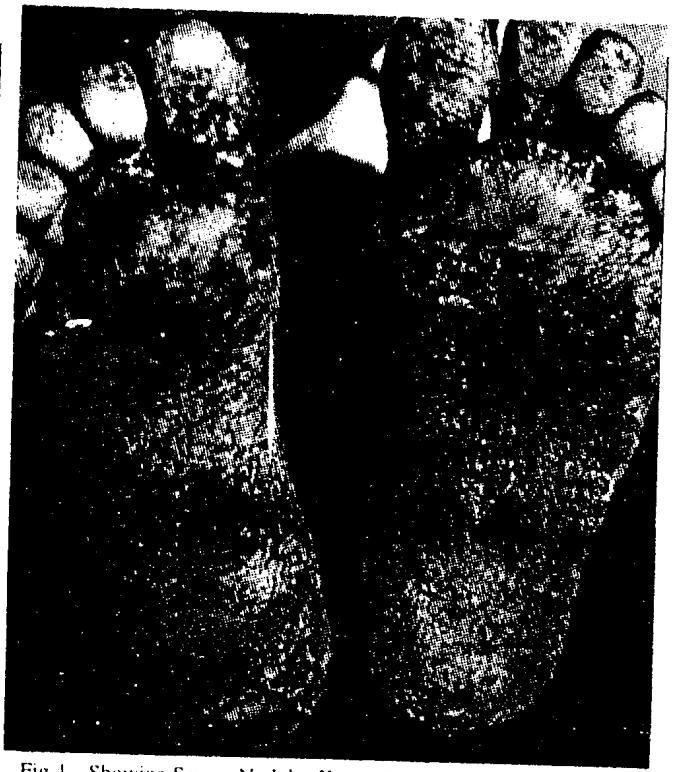

Fig 4—Showing Severe Nodular Keratosis of Sole in Chronic Arsenic Toxicity

Except slightly low haemoglobin value, no other haematological abnormality was detected in any of the cases. Urine report and levels of blood sugar, urea and creatinine values were found to be within normal limits. No abnormality in the ECG nor any evidence of renal, cardiovascular and cerebrovascular disease, Raynaud's phenomenon and gangrene of the limb could be detected in any of the cases. However, 18 cases (11.5%) showed solid oedema of the legs and/or hands.

Symptomatic treatment and drinking of arsenic free water produced some improvement in most of the patients in regard to gastro-intestinal symptoms, weakness, pigmentation and degree of keratosis. However, some still persisted to have symptoms in spite of drinking arsenic free water and there were 5 deaths, 3 of hepatic encephalopathy (2 had associated HBsAg positivity), one of variceal haemorrhage due to portal hypertension and one due to skin cancer with metastasis.

Discussion

Though the cutaneous manifestations like pigmentation and keratosis, characteristic of chronic arsenic toxicity were quite obvious in people drinking arsenic contaminated water in West Bengal, the patients showed clinical features which were often varied and severe. Few of the affected people reported to city hospitals for their treatment as most of them lived in distant rural areas and belonged to low socio-economic class.

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