Arsenic Poisoning: The Existing Information

Arsenic poisoning has been detected in almost all continents. Moreover exposure to arsenic is not a new phenomenon. Arsenic has been known to be a poison since ancient times. Arsenic compounds were used as chemotherapeutic agents of different diseases in different countries. For example Fowler's solution used in psoriasis [WHO 1981]. Arsenic was the first compound used in syphilis, trypanosomiasis. Napoleon Bonaparte and Charles Darwin were known to have suffered from arsenic poisoning [Voet D 1995].

Today, most of the world's arsenic affected areas are in Asia for instance in Bangladesh, India (West Bengal), Inner Mongolia (China), Thailand and Taiwan. Besides those countries, arsenic toxicity is also found in Sweden, Germany, U.K., U.S.A., Chile and Australia. [Chakraborty D 1998 (b)].

Physical and Chemical Properties

Arsenic is a silver gray color brittle, metallic looking substance, atomic number 33, atomic weight – 74.92, specific gravity 5.73 at 25° C, melting point 81.7° C at 28 atmospheric pressure, boiling point 613°C (sublime) [Margil L Bleecker 1994].

In nature, arsenic is found as compound of other elements in both organic and inorganic form. It makes a number of compounds with different metals like iron, copper and nickel or as arsenic sulfide or oxide. In water, arsenic is found in the form of trivalent (arsenite) or pentavalent (arsenate). Arsenite is highly toxic compared to arsenate. In oxygenated water, arsenic usually found as arsenate, but under reducing condition, for instance in deep tube well water, arsenite predominates. Biological activity makes the arsenic into different organic form (like methylated etc.) [WHO, 1981].

Sources of Arsenic in the Environment

Natural Source

Inorganic

The highest concentrations generally occur as compound of copper, lead, silver or gold or as sulphide. Major arsenic containing minerals are arsenopyrite (FeAsS), realgar (As4S4) and (As2S3). Oxidised form of the arsenic are usually found in sedimentary deposits. [WHO, 1981, Hewitt DJ 1995, Susheela AK 1998]. Airborne arsenic particles are both organic and inorganic. Even in rainwater, arsenic has been detected [WHO, 1981]. Arsenic is present in water, both in the form of organic and inorganic state, but mainly in later state. There is wide geographical
variation of arsenic content in river water, other surface water, seawater and ground water. Arsenic may be released into natural water either due to oxidation of iron pyrites/arsenopyrites or due to the reduction of the iron coating on sand grains. [WHO, 1981, Susheela AK 1998].

**Biota**

Arsenic has been detected in different plant and animal products. Plants grown on soil contaminated with arsenic contain high level of arsenic particularly in roots, leaves and seeds. It was observed that the arsenic content of vegetable increased with the increase in soil arsenic content. Some studies had shown high arsenic content in the milk of cattle who were fed with plants grown in soil contaminated with arsenic. [WHO, 1981, Navarro M 1993].

**Industrial Pollution**

Copper smelter, lead ores are the chief sources of arsenic pollution. Apart from those metallurgical industries, dye and pesticide manufacturing, glassware and ceramic industries, petroleum refineries and rare earth industries pollute soil, air and water in adjacent areas. [WHO, 1981, Susheela AK 1998].

**Portal of entry into human body**

Portal of entry of arsenic into human body is through gastrointestinal tract by ingestion of water, food, medicine; respiratory tract by inhalation of arsenic polluted air and sometimes through skin by application of arsenic containing cream as drug. There are several forms of exposure through gastrointestinal tract like water, vegetables cultivated in arsenic contaminated soil, drugs (like Fowler’s solution used for the treatment of leukemia, psoriasis), marine fish, wine etc. [WHO, 1981, Susheela AK 1998]. Air borne entry of arsenic is mainly through inhalation of particles containing arsenic i.e. among smelter workers and workers engaged in the production and use of arsenic containing particle [WHO, 1981].

Normally the daily intake of inorganic arsenic should not exceed \(0.05\) mg. According to the Bureau of Indian Standards (BIS), the permissible upper limit of arsenic in drinking water should not exceed 0.05mg/lit. [WHO, 1981, Susheela AK 1998].

**Mechanism of Action of Arsenic**

Arsenic toxicity depends upon oxidation state, the chemical form and solubility of the chemical [Susheela AK 1998]. According to WHO, maximum permissible limit of arsenic is 0.05mg/liter of water [WHO, 1981]. The lethal dose of arsenic is 1-4mg/kg of body weights [Susheela AK 1998] and thus it belongs to supertoxic category. (See in Annexure II for toxicity category of chemicals).
Arsenic binds with any hydrated sulphydryl group in protein (SH group), distorting the three dimensional configuration of the protein and causing lose of its activity [Burtis CA, 1994 & 1996, Adams RD 1989]. For example, pyruvate dehydrogenase is highly sensitive to arsenic because of its interaction with two sulphydryl group (SH) present in it [Susheela AK 1998]. It has been reported that arsenic level in hair, skin and nail are relatively higher and it can be explained by high affinity of trivalent arsenic for keratin sulphhydryl group (SH) present in hair, skin and nail. [Margil L Bleeker 1994, Susheela AK 1998].

Metabolism of Arsenic

Metabolism of arsenic in the human body is complex and it depends upon the nature of compounds. Arsenic affects most of the systems of human body.

Absorption

Absorption of arsenic (inorganic) from gastrointestinal tract occurs following the ingestion of food, water beverages or drugs. Absorption depends on whether arsenic compound is in solution or undissolved particles. Among all types of arsenic solutions, trivalent is more readily absorbed. Absorption through skin is extremely limited. There is very little evidence of absorption of arsenic through skin. Some studies show the presence of arsenic in the fetus. There are some evidences where heavy ingestion of arsenic causes the death of fetus. Respiratory deposition and absorption occurs through inhalation of air polluted by arsenic. Organic arsenic can be ingested through fish or shrimps. It is readily absorbed through the gastrointestinal tract. Arsenic in animal flesh is not absorbed from the gastrointestinal tract as readily as arsenic from fish or crustacean.

Distribution

In blood, arsenic deposits in R.B.C (red cell) is 3 times more than in plasma. The elimination of arsenic from plasma is faster than R.B.C. Arsenic is widely distributed in the body, and the highest concentrations are in the liver and kidneys.

Elimination

Elimination of arsenic mainly occurs through the kidney. The elimination of arsenic maintains dose response relationship. Different experiments show that the excretory rate of arsenic reached equilibrium after 5 days; by that time 70% of the daily dose is being excreted in the urine daily. Apart from kidney, skin also contributes in elimination of arsenic through sweat and desquamation. Arsenic deposits in hair and thus hair also helps for elimination of arsenic. Arsenic is also detected in human milk and colostrum. [WHO, 1981, Dipankar Das 1998].
Biotransformation

The biotransformation of inorganic arsenic depends on the type of compounds. Pentavalent reduces to trivalent then methylated (i.e. Monomethyl Arsonic Acid (MMA) and Dimethyl Arsonic Acid (DMA)) [Margil L Bleecker 1994, WHO, 1981, Navarro M 1993]. Half-life of arsenic in body is 10 hours. 50-80% of consumed is arsenic excreted after 3 days. Methylation of arsenic in liver is the process of detoxification. The source of methyl groups for arsenic methylation is S-adenosylmethionine. Population thriving on diets low in methionine and other protein are likely to suffer more from arsenic exposure. Methionine and protein deficiencies are considered to decrease the co-factors necessary for arsenic detoxification. [Samuels MA, 1996 Abdulla M 1998]. The anti-oxidant-system in the body is also important for the detoxification of free radicals generated by certain elements. Cytotoxic action of arsenic compounds is mediated through free radicals induced by the element [Abdulla M 1998.]. Metallothionine is a metal binding sulphydral rich protein present in human body that has been suggested to have a protective role in the detoxification of many elements including arsenic [Abdulla M 1998, Gregory L Kidderis 1997]. This complex enzymatic process is also enhanced by vitamin C [Luo Zhen Dong 1998]. Hence nutrition has a very important role in detoxification of arsenic in human body. Organic arsenic is not bio-transformed.

Biological Indicators of Exposure

Arsenic levels in blood, urine and hair are the main biological indicators of exposure to arsenic. Sometimes other body tissues like liver, nail, skin, kidney, lungs are also required for measurement of arsenic [WHO 1981, Yamauchi H 1989].

Patho-physiology of Chronic Arsenic Toxicity of Different Organs and Respective Clinical Manifestations

The present study is dealing with chronic arsenic poisoning, as acute poisoning is not found in the study area. The clinical features and other complications are outcome of different type of pathophysiological effect of arsenic poisoning. The patho-physiology of arsenic toxicity of different body systems along with respective clinical features is described below.

Skin

The different histopathological changes found in skin in chronic arsenic toxicity are (i) Hyperkeratosis (ii) Parakeratosis (iii) Hypergranulosis (iv) Acanthosis (v) Papillomatosis (vi) Dysplastic changes (vii) Basal pigmentation (viii) Dermal Changes (vascular proliferation, hyalinisation, fragmentation and inflammatory cell infiltration). All changes need not necessarily
be present. Furthermore these changes may progress or regress and may be modified by therapeutic interventions.

The traditional histopathological study can reveal benign dyplastic or neoplastic nature but there is no specific histopathological picture diagnostic of arsenical skin lesions. The above microscopic changes are interpreted with the background information of arsenical exposure. [Dutta SK 1998].

Number of skin lesions has been found in chronic arsenic poisoning. Pigmentation, de-pigmentation and keratosis are the main dermatological manifestations [Chakraborty D, 1998(C), Roy S 1998]. Raindrop pigmentation (hyperpigmentation i.e. dark spot) of the skin often associated with paler spots (de-pigmentation) is commonly encountered and occurs mainly in the areas of the skin not exposed to the sun like trunk, axilla [WHO, 1981, Thomas P Habif, 1996, Fitzpatrick 1993]. The hyper-pigmented spots look like bronze colour spot [Fitzpatrick 1993]. The hypo-melanotic (de-pigmentation) areas with in hyper-pigmented skin have fancifully been likened to "rain drops on a dusty road" [Samuel L Moschella 1992].

Arsenic keratosis is discrete round, wart like or pointed keratotic lesion. It usually develops at the site of friction and trauma. It looks like corny papules usually 2 to 10 mm in diameter. A second type of arsenical keratosis appears as a slightly elevated, scaly; erythematous or pigmented patch. These appear mostly often on unexposed body areas. Arsenical keratosis tends to become painful with bleeding, fissuring and later ulceration [Thomas P Habif, 1996, Fitzpatrick 1993].

Dose-response relationship between arsenic ingested through drinking water and skin manifestation, tolerance has not been shown [Rook Wilkinson 1992]. But dose response relation between arsenic medication and hyperkeratosis was described by Fierz. Hyper keratotic lesions of the palms and soles and melanosis are uncommon among workers exposed to airborne arsenic. Only dermatosis due to local irritation has been reported [WHO, 1981, Hotta N 1998], whereas typical skin manifestations of chronic arsenic poisoning have been reported among several Japanese workers who were employed in pesticide factory where arsenic containing pesticides were manufactured [Vassileva S 1990]. In all diagnosed cases there was melanosis, either raindrop or diffuse pigmentation of skin, whereas warty lesions and hyperkeratosis in palm and sole were seen in nearly 70-80% of patients [Maidul Islam AZM 1998, Guha Mazumdar DN 1992, 1998].

In fact, according to Dr K C Saha, a dermatologist who first identified the arsenicosis patient in 1982 in Calcutta, melanosis is the first sign to appear. [Saha KG 1984, 1995] Keratosis on palm and sole is the sign of moderately severe toxicity [Chakraborty D 1998 (a)].
Photosensitivity and itching are another dermatological manifestations commonly found among arsenic patients [Rook Wilkinson 1992 Susheela AK 1998].

Chakraborty observed in Bangladesh that significant number of population exposed to high level of arsenic (evidenced by arsenic concentration of water, hair, nail) having no symptoms [Chakraborty D 1998 (a)]. It seems, body immune system along with detoxification capability against arsenic may play important role in clinical manifestation despite high level of arsenic exposure to individual.

Nervous System

Pathologically, arsenical neuropathy is of the "dying back" (axonal degeneration) type. [Adams RD 1989]. Electro-physiological studies showed progressive slowing of motor conduction velocity. [Margil L Bleecker 1994]. A very recent study shows activity of neuro transmitter producing enzyme decreases due to arsenic toxicity. [Rahman MK 1998]. Electro-diagnostic examination shows there is early neuropathy – demyelinating poly radiculopathy, later widespread axonal degeneration. [Samuels MA 1996]. In axonal degeneration, more severe involvement is found in large fibre than smaller fibre. Ultrastructural study shows an axonal degeneration (onion bulb) formation in addition to segmental demyelination, which appear earlier than axonal degeneration. [Adams RD 1989]. Recovery of sensory function may occur gradually over 2 years. [Samuels MA 1996]. Photosensitivity in arsenic poisoning is another important symptom. [Rook Wilkinson 1992].

There are number of studies showing relationship between arsenic poisoning and varying neurological disorder. Heyman reported 41 cases of suspected arsenic neuropathy in the USA long back (in 1950) [Heyman A 1950]. Cases of occupational arsenic neuropathy have also been reported from Japan [WHO 1981]. Guha Mazumdar found neurological manifestation of several cases, which consumed arsenic, contaminated ground water [Guha Mazumdar DN 1988, 1992, and 1998].

The neurological manifestation includes peripheral nervous disturbances, neuritis, retrobulbaris, chronic rhinitis, and sensory motor polyneuropathy. Numbness, tingling of distal lower extremities may be associated with hyperalgesia and spontaneous pain. Muscle tenderness, cramps and increased sweating found frequently [Margil L Bleecker 1994]. Arsenic causes sensory greater than motor neuropathy, although both large and small fibre sensory modalities get affected, proprioceptive function may be specially impaired (like cutaneous, sensation, vibration and position sensation), weakness, usually mild and confined to the foot extensors and intrinsic hand muscles, tendon reflexes diminish and gradually intensify increases. Wasting of forearms and legs especially smaller muscle of hands and feet develop. Eventually a severe stocking-glove sensory motor neuropathy develops [Samuels MA 1996, Maidul Islam AZM 1998, Lewis Rowland 1995 & Chang LW 1997].
Hearing loss due to arsenic poisoning has been reported in some cases. In Japan, hearing loss had been observed among children who consumed powdered milk containing pentavalent arsenic [WHO 1981]. Bencko of Czechoslovakia observed hearing loss among children living nearby a power plant burning local coal that had a high arsenic content [Bencko 1977]. On the other hand Milhan failed to demonstrate relationship between hearing loss and arsenic poisoning [WHO 1981].

Respiratory System

Arsenic affects the mucus membrane in the respiratory system. There is rhinopharyngitis, tracheobrochitis and sign of pulmonary insufficiency due to emphysematous lesions. [WHO, 1981].

Respiratory system is mainly affected by inhalation of arsenic. It affects the mucus membrane in the respiratory system including perforation of nasal septum. [WHO 1981] There is also reported history of broncho pulmonary disease among people who have ingested arsenic affected water. Cough, breathing difficulty respiratory sign of rales and/or rhonchi are common. Lung function test showed presence of respiratory distress among significant number of patients [Maidul Islam AZM 1998, Guha Mazumdar DN 1998, and Saha KC 1998]. There is evidence of death of patients with chronic lung disease, sometimes complicated with tuberculosis [Guha Mazumdar DN 1998].

Liver

Inorganic arsenic toxicity is associated with pathological liver changes. There are number of studies, which show changes in the liver following arsenic toxicity. A common finding is portal hypertension without sign of liver cirrhosis. Biopsy report of many studies showed enlargement and fibrosis of varying degree in the portal regions without gross damage of hepatocytes. [WHO, 1981].

Guha Mazumdar in his several studies showed enlargement of liver (hepatomegaly) (70-80%) with/without portal hypertension (30-35% of all hepatomegaly cases) [Guha Mazumdar DN 1988, 1998]. In advanced cases, portal hypertension with ascitis and hepatic failure take place [Saha KC 1998]. Nobuyuki Hotta of Asia Arsenic Network, conducted a study based on a bibliographical comparison where a lot of clinical inspections in different endemic areas of arsenicism in the world have been discussed. He noticed there is high incidence of non-cirrhotic portal fibrosis (hypertension) in West Bengal [Hotta N 1998]. In fact, several cases of death due to
arsenic poisoning were reported (in West Bengal) due to hepatic encephalopathy, portal hypertension with gastrointestinal bleeding [Guha Mazumdar DN 1998].

Cardiovascular system

Many researchers have observed pathological changes in cardiovascular system due to arsenic poisoning. Peripheral vascular disease due to arsenic poisoning is noticed in several areas. The symptoms and signs include arteritis obliterans and acrodermatitis atrophicans. In Taiwan this peripheral vascular disease is known as black foot disease. This disease leads to gangrene of the affected part and auto amputation [WHO, 1981]. Rosenbery in his study described the pathological lesions of blood vessels due to arsenic poisoning. There is initial thickening of small and medium sized arteries of most organs resulting in narrowing of the lumen. He observed vascular damage to kidney, heart, lung and intestines etc. [Rosenbery HG 1974]. Engel also reported similar findings on the effect of arsenic on blood vessels. [Robert R Engel 1994] It is noteworthy, that the pathological findings of blood vessels were found mainly among those who consumed arsenic affected water [WHO, 1981].

Non-pitting oedema with/without gangrene has been reported in several studies [Chakraborty D 1996, 1998 (b), Roy S (1998), Guha Mazumdar DN 1998, Saha KC 1998]. Gangrene occurs due to peripheral vascular disease, which is manifested as intermittent claudication [Guha Mazumdar DN 1998]. Cardiological manifestation is diagnosed in several cases. Broadening of QRS complex is the commonest Electro Cardio Graphic change (E.C.G). Among smelter workers, an increase in incidence of mortality due to cardiovascular disease was observed [WHO 1981, Luo Zhen Dong 1998].

According to Nobuyuki Hotta from Asia Arsenic Network, occurrence of peripheral vascular changes in the extremities such as black foot disease, ulcer cruris chornicus and non-pitting oedema is more common in Taiwan and also in gangetic delta [Hotta N 1998]. Chen, through his study had shown high incidence of ischaemic heart disease, hypertension and cerebral infarction among arsenic exposed patients. He also noticed abnormal peripheral microcirculation among those patients [Chien-Jen Chen 1998]. Xian Yun, in his study had shown increase incidence of peripheral vascular constriction leading to peripheral vision defect among chronic arsenic exposed patients [Ren Xian Yun 1998]. Conjunctival congestion /conjunctivitis has been found among some patients [Maidul Islam AZM, Chakraborty D 1998]. Some studies had shown incidence of post-subcapsular opacity (cataract) [Chien-Jen Chen 1998, Lai Chen See 1998]. Peripheral visual constriction is increased by arsenic poisoning [Ren Xian Yun 1998].

Haemopoetic System

In blood, arsenic combines with globin part of haemoglobin, also found in leucocyte and serum protein. [Samuels MA 1996]. Westhoff described effect of arsenic intoxication of hemopoetic system. Pancytopenia with megaloblastic erythropoisis was detected. Other features are low reticulocyte index increased marrow cellularity with erythroid hyperplasia with morphologic
evidence of megaloblastic maturation in marrow but the patients' serum folate and Vitamin B12 were normal [Westhoff DD 1975]. Feussner similarly described finding of peripheral blood and bone marrow pictures. Apart from similar findings of Westhoff, Feussner found bizarre karyorrhexis, coarse basophilic stippling and numerous Howell – Jolly bodies [Feussner JR 1979]. Some studies show that nearly half of the arsenic affected patients suffer from anaemia [Guha Mazumdar DN 1998, Luo Zhen Dong 1998].

Reproductive Organ, Foetus and New born

Lugo described the result of arsenic ingestion in pregnant women. Arsenic crossed the placental barriers and affected the foetus. Later, premature labour occurred and the newborn baby died within few hours. Autopsy revealed presence of arsenic in the foetus [Lugo G 1969]. Eastman and Susheela AK found the presence of arsenic in placenta and umbilical cord blood [Susheela AK, 1998 Eastman NJ 1931]. Nordstrom and his team conducted number of studies at a copper smelter in northern Sweden. They found the variation of birth weight according to the degree of exposure. The women who lived near copper smelter gave birth to low birth weight babies. Arsenic accumulates in breast milk of arsenic exposed women and thus new born babies can also get further exposure to arsenic [Marie Vahter 1998, Dipankar Das 1998].

Spontaneous abortion, still born children, birth defect and low birth weight are considered very serious threats from arsenic. Number of cases has been reported in different parts of the world. Nordstrom and Axelson conducted series of studies regarding occupational and environmental risk in and around smelters in northern Sweden. The fumes come out from copper smelter is full of volatile arsenic compound. They noticed, there had been spontaneous abortion among the female employees and decreased birth weight and congenital malformations among their offspring [Nordstrom 1978 (a) (b), Axelson O 1978]. Where as Nordenson I found chromosomal aberration and other defect in the same area among the same workers [Nordenson I 1978]. Several research papers found positive correlation between chromosomal defect and abortion. In fact main reason of spontaneous abortion is chromosomal defect [Szulman AE 1965, Stenchever MA 1967, Kajii T 1973, Carr DH 1967, Boue' JG 1973, 1975, James WH 1963, Kantor HI 1948 & Arakaki DT 1970]. A study conducted in a Hungarian town, found frequency of spontaneous abortion, still birth and perinatal death reduced in significant manner when the town authority started supplying arsenic free water to the inhabitants [Csanady M 1998]. As arsenic accumulates in breast milk, newborn baby starts getting exposed from early phase of life and shows early arsenic symptoms [Dipankar Das 1998].

Endocrine System

Some recent studies have shown a relationship between arsenic poisoning and diabetes. Arsenic is found damaging the cells of pancreas islets of Langerhans, which produces insulin in body [Lai MS 1994].
The prevalence of diabetes mellitus observed in a community based health survey among residents in arsenic endemic areas. Studies of Mee-shu-Lai and Chen in Taiwan showed higher prevalence of diabetes among the people who are exposed to arsenic [Lai MS 1994, Chien-Jen Chen 1998]. Rahman et al found high incidence of diabetes mellitus among the workers of copper smelters and art glass industry in Sweden and villagers drinking arsenic polluted water in Bangladesh [Rahman Maidul AZM 1998].

Chromosome & Mutation

Evans et al observed chromosomal abnormalities including chromatic breaks and gaps, chromosome breaks, translocation, rings and dicentrics were common among prolonged arsenic exposed people. They opined that arsenic in man is both mutagenic and carcinogenic [Evans S 1998]. Wu showed high incidence of cancer among arsenic exposed person in Taiwan [Wu MM 1989]. Studies by Feussner tried to establish the relationship between arsenic and bone marrow toxicity [Feussner JR 1979].

In fact in 1822, in Paris arsenic had been discovered as carcinogenic compound [Fitzpatrick 1993]. Respiratory system, skin, liver are mainly affected. Malignancy of respiratory system mainly occurs among the smelter workers engaged in the production of arsenical insecticides and also those who sprayed arsenic containing pesticides and exposed to arsenic through inhalation [WHO 1981].

Various types of neoplastic changes of the skin, including Bowen’s diseases and basal and squamous cell carcinomas have been associated with arsenic exposure. Bowen’s diseases and basal cell carcinoma of arsenical origin are usually multiple and located at the trunk [Thomas P Habif 1996, Chakraborty D 1996]. Bowen’s disease is erythematous, slightly hyper pigmented, scaling, round plaque on trunk or limbs. Some times Bowen’s disease is considered skin marker of internal malignancy [Fitzpatrick 1993, Shoko Oshikawa 1998]. Not all patients with Bowen’s disease including both sun exposed and non-sun-exposed sites have an increased risk of internal malignancy. Rather the relationship between Bowen’s disease of non-sun-exposed skin and an increased risk of internal neoplasia has been a long-standing controversy [Fitzpatrick 1993, Rook Wilkinson 1992]. Nevertheless, this kind of cancers are found having connection with the combined effect of arsenic and ultraviolet radiation [Grobe JW 1998]. Squamous cell carcinoma developed primarily from the keratosis on the extremities. The lesions are more common in the palm and sole [Thomas P Habif 1996, WHO 1981, Fitzpatrick 1993].

Haemangio endothelioma of the liver is the only form of malignancy proved to be associated with arsenic intoxication. A number of studies have also reported liver kidney, urinary bladders cancer [WHO 1981, Chien-Jen Chen 1998, Smith AH 1998, Evans S 1998, Chakraborty D 1996] due to arsenic poisoning throughout the world.
Incidence of malignancy due to arsenic poisoning in West Bengal is comparatively less but not uncommon [Chakraborty D 1996, 1998 (c), Roy S 1998]. Skin cancer is the commonest form of malignancy [Saha KC 1998]. In one study, some cases of death were reported due to arsenic related skin cancers with metastasis [Guha Mazumdar DN 1998].

Management of Arsenic Toxicity

Three methods are commonly advocated for management of arsenic patients.

Stop drinking and using arsenic contaminated water

Some studies were conducted to examine effect of safe drinking water (arsenic free) on the patients. One study was conducted in West Bengal showed that after prolonged discontinuation, the arsenic in hair, nails and skin-scale came down to normal levels but skin lesions still persist [Chakraborty D 1997]. Another study conducted in West Bengal showed a slightly different picture. Partial improvement of pigmentation keratosis and neuropathy was observed in nearly half of the cases while most of the hepatomegaly persists. Further, most distressing observation was the new appearance of lung diseases (shortness of breath and chest sign), hearing impairments and dimness of vision (Guha Mazumdar DN 1998).

Medical management

The drugs most commonly used now in arsenic poisoning are D-Penicillamin (Dimercaprol), water-soluble analogue of Dimercaprol, dimer-captopropanesulfonic acid (DMPS) and dimer-captosuccinic acid (DMSA). These drugs combine with arsenic and get excreted through urine and thus body burden of arsenic gradually reduces [Kosnett MJ 1998]. But results are not very encouraging. Improvement of keratosis and pigmentation were observed (in study of West Bengal). But there was no change in hepatomegaly rather oesophageal varices appeared in some cases which were absent earlier. Moreover, chest signs also appeared which was not found earlier. According to Dr Guha Mazumdar, neither drinking arsenic free water nor treatment with chelating agent for a short period could halt the progression of liver and lung disease in cases of chronic arsenic toxicity, though dermatological manifestation may regress is some cases. Evidence of peripheral neuropathy also regresses in some patients but new appearance of hearing loss and dimness of vision suggest progressive cranial nerve palsy in a few cases [Guha Mazumdar DN 1998]. Similar kind of opinion has been expressed by other researchers [Samuels MA 1996, Kosnett MJ 1998].

Nutritional approach

It is note worthy that, nutritional status has been found linked with clinical manifestation. These findings are based on establishing linkages between nutritional status of patients and clinical manifestations and effect of nutrition intervention and various biochemical assays. Chakraborty and his colleagues observed in different parts of West Bengal that nutritional status
of the people plays a significant role in clinical symptoms [Chakraborty D 1997, 1998 (b)]. In Bhagobangola block of Murshidabad district (nearby the present research study block of same district), Chakraborty observed malnourished children suffer more from arsenic poisoning although arsenic level in contaminated water is not much higher [Chakraborty D 1998 (b)]. Others observed similar kind of differences. Samuel and Chen found a positive relationship between nutritional status and neuro-cutaneous manifestation and cutaneous malignancy respectively [Samuel L Moschella 1992, Chien-Jen Chen 1998]. In present study also, malnourished patients had more severe manifestations. But, malnutrition is a complex issue as it is linked with socio-economic status, occupation, food habit etc.

However, researchers identified some nutrients, which have protective role against toxicity of arsenic and recommended taking those nutrients. Some biochemical assays done in different part of the world, observed inverse dose-response relationship between subsequent arsenic induced skin cancer and ischaemic heart disease and serum carotene (Vitamin A derivative) [Chien-Jen Chen 1998, Kosnett MJ 1998 & Yu-Mei Hsueh 1998]. Selenium, an antioxidant material that appears to antagonise several cellular effects of arsenic in biological system [Kosnett MJ 1998]. Study regarding effect of systemic oral use of Vitamin A, C & E along with restriction of arsenic contaminated water and topical application of 5% salicylic acid ointment to soften the keratotic patch was found to be encouraging [Maidul Islam AZM 1998]. Chakraborty suggested in order to ameliorate the symptoms, the villagers should be encouraged to eat more fresh vegetables, fruits (locally available). These vegetables are high source of vitamins and other antioxidant, which reduce the effect of arsenic toxicity [Chakraborty D 1998 (b) (c)]. Methionine present in animal protein (fish, meat etc.) can enhance detoxification of arsenic in human body [Samuels MA, 1996 Abdulla M 1998].

**Arsenic Toxicity in West Bengal**

**Extent of Problem**

Before the 1980s chronic arsenic poisoning due to contaminated ground water was unknown in West Bengal. In 1982, Prof. K C Saha, Dermatologist of School of Tropical Medicine, Calcutta, first detected arsenicosis patient from north 24 Parganas district of West Bengal and in 1985 from patients of Khulna and Raj Shahi districts of Bangladesh [Guha Mazumdar DN 1998]. Scientists of School of Environmental Studies (SOES), Jadavpur University, Calcutta started research on this issue. SOES and Department of Gastroenterology, Institute of Post Graduate Medical Education and Research (IPGMER) Calcutta conducted number of joint and individual studies [Guha Mazumdar DN 1998, Saha KC 1998, and Chakraborty 0 1996).

SOES usually conducted geological studies like measuring arsenic level in water and also nail, urine and body tissue samples. Where as IPGMER conducted medical studies (clinical,
therapeutic etc.). Apart from that All India Institute of Hygiene and Public Health (AIIPH), Calcutta and School of Tropical Medicine, Calcutta also conducted some isolated studies.

Table 2.1 shows district wise profile of arsenic contaminated blocks and villages and exposed population and manifestations in West Bengal. The report is based on studies conducted till 1994. [Chakraborty D, 1994] Arsenic cases and water contamination in Balagarh block of Hoogly district has been detected in January 1996.

Table 2.1: Arsenic Contamination and Manifestation of Toxicity in Six Districts of West Bengal (till 1994)

<table>
<thead>
<tr>
<th>Districts</th>
<th>Total</th>
</tr>
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<tbody>
<tr>
<td>Nadia</td>
<td>24 PGS(N)</td>
</tr>
<tr>
<td>Total No. of Arsenic affected blocks</td>
<td>3</td>
</tr>
<tr>
<td>No. of Arsenic affected villages</td>
<td>33</td>
</tr>
<tr>
<td>Approx. pop drinking Arsenic contaminated water</td>
<td>48,512</td>
</tr>
<tr>
<td>Approx. pop showing Arsenic related skin manifestation</td>
<td>12,128</td>
</tr>
</tbody>
</table>

Source: Chakraborty D, 1994


A study conducted in 1996 shows number of arsenic affected districts, blocks and villages have increased up to 7, 50 and 560 from 6, 37 and 312 respectively [Chakraborty D, 1996]. Studies in 1998 show number of arsenic affected districts, blocks and villages further increased up to 8, 63 and 863 respectively [Chakraborty D et al, 1998(b)]. Till May 1998, nearly 863 villages from 8 districts (of West Bengal) were affected by arsenic menace. The average concentration of arsenic in contaminated water is about 0.20 mg/lit. Approximate number of people drinking arsenic contaminated water above 0.05 mg/litre is nearly 2 million and approximately 2,00,000 people are showing arsenic related skin manifestation. The sources of arsenic contaminated water are tube well / hand pumps and also shallow and deep pump used in irrigation. Number of government supported tube well / hand pumps under Rural Water Supply Scheme were found contaminated with arsenic [Chakraborty D 1994, 1998 (b), Mallik S 1996].

Similar kind of arsenic problem has appeared in neighbouring country – Bangladesh. In fact, condition is more serious than West Bengal. In 41 out of 64 districts in Bangladesh, arsenic
in ground water has been found more than 0.05 mg/litre [Chakraborty D 1998 (a, b), Barry B 1998].

But it is worth noting that determinants and factors responsible for distribution of the disease have not been properly addressed in any study. There had been a tendency among the researchers to incorporate some social factors (like income) just to support the studies, perhaps resulted from pre-conceived idea that clinical manifestation on account of arsenic poisoning has been essentially biological. Due to piece meal nature of sociological research, there has been non-availability of systematic quantitative and qualitative data (except some brief case stories). Moreover, studies of consequences of arsenicosis on the lives of the affected have not been dealt so far. In order to tackle arsenic problem at grass root level, an all out effort is required. Hence, social epidemiological study at community level is pre-requisite to get right picture of cause and effect relationship. There is also need to review the existing control measures and intervention available like treatment facilities and seeking alternative water source.

Causes of Arsenic Contamination of Ground Water

According to the concerned scientist, entire Gangetic delta and its surrounding areas are affected by arsenic poisoning, which is due to geological reasons. Arsenic has been found in ground water in West Bengal and Bangladesh in the area of sediment of younger deltaic deposition [Chakraborty D 1997]. Arsenic has contaminated the ground water in the vast tract of alluvial aquifer in the Bengal delta plain. The arseniferous belts located in the upper delta plains are mostly characterised by complete or truncated cycles of fining upward sequences dominated by course to medium sand, fine sand, silt and clay sediments. Geochemical and hydrogeological characteristics of these alluvial sediments influence the mobility of arsenic in ground water [Bhattacharya P 1998]. Geological exploration shows that sedimentation has been going on since the Carboniferous – Permian Period (340 million years ago) to the recent time [Alam MK 1998].

Arsenic is abundantly found in the sediment as arseno pyrite (FeAsS) in iron pyrite (FeS2) rocks. Pyrite is a ubiquitous mineral (iron compound), occurring in most major rock types [Chakraborty D 1998 (b), Shahidullah M 1998, Nordstrom DK 1998]. The reason why arsenic is coming out with ground water is not completely clear. But many scientists all over the world suggested that due to heavy ground water withdrawal, the underground aquifer is aerated and the oxygen in air is responsible for degradation of arsenic rich sources. The oxygen decomposed the pyrite (FeS2) rich in arsenic and the acid released leached out the arsenic in soluble form in ground water. The decomposition of arsenic rich in pyrite (FeS2) may be through the following reactions.
2FeS₂ + 2H₂O + 7O₂ → 2Fe⁺³ + 4HSO₄⁻

4Fe⁺³ + O₂ + 4H⁺ → 4Fe⁺² + 2H₂O

FeS₂ + 14Fe⁺³ → 15Fe⁺² + 2SO₄⁻² + 16H⁺


The Fe⁺³ ions formed act as a catalyst for the further decomposition of pyrite. Kinniburgh and Welch gave the same explanation regarding ground water quality of London basin aquifers and western USA respectively. [Chakraborty D 1994, 1996, 1998 (b), Mallik S 1996, Roy, Choudhury T 1998].

However section of scientists raised question regarding this explanation. They do not agree with the previously proposed hypothesis of pyrite / arsenopyrite oxidation as the mechanism for the release of arsenic in ground water. They identified less amount arsenic in the pyrite while analysing the sediments of Bengal delta. Rather, they found a linkage between iron and arsenic. Moreover they noticed lack of arsenic in oxidised water, which is in contrary to the previous theory. The alternative mechanism proposed is that arsenic has been released upon reduction of iron oxyhydroxide. The weathering of sulphide minerals (mainly pyrite and arsenopyrite) released arsenic in water. As the arsenic rich ground water is mostly restricted to the alluvial aquifers of the Ganges delta, the source of arsenic rich iron oxyhydroxide must therefore lie in the upstream of West Bengal & Bangladesh. The weathering of sulphide minerals (mainly pyrite and arsenopyrite) released arsenic in Ganga water in upstream (like Uttar Pradesh, Bihar) and later scavenged /absorbed by iron oxyhydroxide in down stream Ganges sediments during late Pleistocene to recent times. The arsenic rich iron oxyhydroxide are now being reduced and causing the present problems. The absorbed arsenic is released to ground water in reducing conditions where microbes play a major role [Ahmed KM 1998, Nicksen R 1998]. Chakraborty also suspected role of microbes like Thiobacillus in releasing arsenic into ground water. He did not rule out the possibility of nitrate fertiliser in pyrite breakdown. Therefore, he suggested further study [Chakraborty D 1998 (b)]. Relations of iron with arsenic have been evidenced by some other researchers like presence of arsenic where iron pyrite is present (chemical reactions given earlier) [Susheela AK 1998, Bhattacharya P 1998].

Despite having controversy with regard to geo-chemical mechanism of release and mixing of arsenic in ground water, it has been well accepted that excessive use of ground water triggered the leaching out of arsenic.
Agriculture and Water Sanitation Development in West Bengal

Agriculture Development – Shifting Paradigm

After getting power, left led ruling parties decided to improve state’s agriculture sector and took it as top priority. During late 1970s – 80s, two major changes have taken place i.e. land reform and adoption of intensive agricultural area development, based on high yielding variety crops, high irrigation and chemical fertilizer inputs. [Rawal Vikal 1998, Harris John, Lieten GK 1990]. State government followed technocentric approach in order to increase food production as main objective. As a part of land reform, excess amount of land (above ceiling) was distributed among the landless labourers and financial and material support was provided to the new owners to encourage following new agricultural policy. The farmers were provided financial support in order to buy new high yielding seeds, fertiliser and pesticides and pumps for irrigation. Since beginning of land reform in West Bengal, significant growth in productivity and cropped area had been noticed. While the decade of the 1970s was marked by stagnation in agricultural production in eastern India, a noteworthy change occurred in 1980s. Between 1981 and 1991 rates of growth of agricultural production in the eastern states increased and among them, West Bengal grew fastest. Compound annual rate of food grain production in 1981-82 to 1991-92 in West Bengal and All India were 6.5 (3rd in all India level) and 2.7 respectively [Rawal Vikal 1998].

The policies adopted resulted in the following changes in agricultural practices.

Increased Frequency of Cropping and Increasing Land Area for Cultivation

Cropping frequency incremented from once or twice a year to thrice or even four times a year [Chakraborty D, 1998 (b)]. From 1977-78 to 1995-96, exponential growth rate of area under cultivation for all crops and all food grains were 0.547 and 0.387 percent per annum respectively [Lieten GK 1990, Sanyal MK 1998]. It was mostly achieved through converting forest area and wastelands into cultivable land [Niladri Saha 1997].

Changing Cropping Pattern

Changing pattern in cropping varieties was important feature observed since 80s till now. Rate of growth of rice production (land area), of two main traditional varieties i.e. “Aus” and “Aman” from the year of 1980 to 1995 were ‘−1.6’ (negative) and 0.34 respectively, where as of high yielding variety “Boro” it was 8.6 (during same period). Although “Aman” contributed nearly 64% of total output whereas “Boro” contributed 29%. But this 64% of output came from 72% of area but 29% of “Boro” rice was from 19% of lands cultivated [Rawal Vikal 1998]. And for this reason people were gradually shifting to “Boro” despite the fact it required huge amount of water for irrigation [Chakraborty D, 1998 (b)]. As far as exponential growth rate of rice (Aus, Aman and Boro) was concerned, West Bengal and Murshidabad districts were showing similar kind of trend [Sanyal MK 1998].
Rapid Increase in Irrigation

Irrigation was the most important input in agricultural development of West Bengal. From 1976-77 to 1985-86, percentage increase of net area irrigated was 74% as compared to about 20% in India.

Net Results

Increase in frequency of cultivation, increase in area of arable land and rising trend of cultivation of some particular varieties of crops (for example Boro rice) gave rise to many fold increase of water demand. This rising demand of water was met through ground water irrigation. Since then, there has been a marked increase in drawing up of ground water. Gradual shifting from traditional source to ground water has been observed in all districts of West Bengal. There has been a big expansion of tube well irrigation (area irrigated by tube well increased as much as 575%) drawing upon under ground resources. [Lieten GK 1990, Rawal Vikal 1998]. Directorate of Indian Agriculture, Ministry of Agriculture GOI (1997), showed during 1981-82 to 1992-93 in West Bengal canal irrigation increased marginally (638 to 717 thousand hectares), tank and other irrigation declined but well irrigation (ground water) increased considerably (from 444 to 712 thousand hectares).

The change in agricultural practice increased the dependence and use of ground water. This dependence led to over exploitation of ground water, which triggered its arsenic contamination. (Chakraborty D 1994, 1996) When irrigation was not required, villagers took away the pump sets. For cost cutting there was practice to leave the underground pipe open to air, which perhaps had accelerated oxidation of iron pyrite and arseno pyrite)

Water and Sanitation – New Policy

During the same period (i.e. late seventies and onwards) state health department promoted ground water for human consumption in order to avoid several water borne diseases, which were the commonest causes of morbidity and mortality in the state. With the help of public health engineering department and assistance from other agencies, state government sank several hand pumps as a part of rural water supply scheme (RWSS). Due to continuous campaign of state authority and encouraging experience (i.e. clean and easy to collect) of the users, many villagers also started spending their own money to sink hand pump in their own houses or near by. In due course of time, people got habituated and depended in using hand pumps as main source of water for drinking and domestic use. Dug wells, which were very close to surface level, could provide arsenic free water, as arsenic minerals had been located far below the water level of dug well. But due to contamination of water with microbes, often well water became unfit for consumption. Gradually the acceptance of hand pumps led to lack of use and maintenance of the dug wells, which used to be main source of water of earlier period. Later they became defunct. In many places, the dug wells were used as dumping places of garbage.
The hand pumps became a main source of arsenic contamination to human body. The arsenic at under ground level flowed from one place to surrounding areas and contaminated the hand pumps used for domestic purposes.

Other Changes in New Agricultural Policy

Food production: Nutrition Impact

In 80's and onwards, there was an increase in area sown with mustard and potato and a decline in area sown with wheat, minor cereals like barley, gram and other pulses and jute [Rawal Vikal 1998, NFI 1999, Patnaik Utsa 1996, Sadasivan Swarna 1989]. Among them, a trend in pulse production was worth to be noted. In India the traditional diet consisted of proper combination of cereal and pulses. Pulses compensate need of protein for people. In all India level there is decreasing tendency of pulse production (6.13% from 1989-90 to 94-95) but increase production of rice and wheat [NFI 1999, Patnaik Utsa 1996, and Sadasivan Swarna 1989]. Therefore per capita availability of rice and wheat was increasing with decreasing pulse availability [NFI 1999, Patnaik Utsa 1996]. Eventually there was widening of gap between nutritional requirements and their intake. Before green revolution, in 1965 per capita cereal consumption was 418.5 gm/day and in 1996 it raised to 464 gm/day. Pulse intake in those years was 65.5 gm. and 34 gm. respectively [Niladri Saha 1997]. Situation of West Bengal was worse. There was significant negative production trend of Rabi and Kharif pulse production [Sadasivan Swarna 1989]. Situation of study district (Murshidabad) was further worse, as it has been far below the average state level production [Sanyal MK 1998].

Wide spread cutting of jungles and fruit trees for agriculture, economic and housing purpose, easy availability of fruits in the villages became difficult. Conversion of waste and fallow land to arable lands also reduced the availability of normally grown green vegetables. Improved communication encouraged the villagers to sell their vegetables and fruits in nearest towns. In villages it resulted non-availability and rising price of green vegetables and traditional fruits, which were rich sources of vitamins, minerals and antioxidants and these micronutrients could have provided some protection against arsenic manifestation.

Increased crop production benefited the farmers. A favourable political environment allowed agricultural workers successfully to demand the implementation of minimum wages in agriculture. In fact, average growth rate of real daily wages of male agricultural labours between 1979-93, West Bengal showed the highest growth rate (2.8%). There was also evidence to suggest that the male-female wage differential has not been high in rural Bengal [Rawal Vikal 1998]. So, economic benefit out of increased production and wage encouraged and enabled the farmers to practice new agriculture policy. Thus despite improved economic condition, an imbalance in agricultural development and food production as well as ecological degradation led to the problem of arsenic toxicity and its manifestation.
Experience in Bangladesh

Similar kinds of features are also observed in Bangladesh. In Bangladesh green revolution started in late 70s with introduction of high yielding variety corps, particularly, ‘Boro’ rice. Irrigation (ground water) increased many folds. As a result of which rice production increased in every corner of Bangladesh with expense of declining pulse production. Government is consistently focussing on rice and wheat production with a target to make nation self sufficient on cereals. But there is significant decline of other type of food production (pulse) and availability leading to imbalance in nutritional intake [Firdousi Naher 1997].

Gaps in Existing Research

The available literature regarding chronic arsenic toxicity essentially envisaged geological and clinical perspectives. There has been no systematic collection and analysis of role of social factors with relation of chronic arsenic toxicity among the arsenic exposed population and the consequences of manifestation. The sequel of chronic arsenicosis is limited upto the manifestation with reports of death. But there are no documents so far on impact of the health problem on the lives of people and the community. The existing epidemiological research reports mostly focussed on distribution of “cases” and their manifestations and linkage with arsenic concentration of ground water.

State’s Role to Combat Arsenic Poisoning

IPGMER and some indoor wards of medical colleges in Calcutta have proper treatment facilities. IPGMER runs separate arsenic clinic (once in a week, our door) and inpatients department. But people living in remote areas cannot get access to them. State Governments, Health Departments organised some awareness camps and workshop with financial assistance from UNICEF, WHO, Rajiv Gandhi Water Mission etc. But these all are confined to city of Calcutta and district town. Special Arsenic Committee has been constituted but nothing comprehensive has come yet.

Some experts recommended promoting arsenic filter to all villages, which is not viable solution. AIHHP is trying to develop low cost filter for using in household level and community level. There are some other organisations that are also trying to develop filter. But the question still remained unanswered after filtration of water where those filtrate which has high concentration of arsenic to be kept. Improper filtrate management may lead to another kind of ecological disaster like acute poisoning (both man and live stalk), contamination of surface water (only source of arsenic free water) etc. But there is no comprehensive approach being taken at Government level. This piecemeal approach will no longer become useful, until and unless a comprehensive step and proper impact assessment are done.

The root cause of the problem is not tackled since it will adversely affect the economy of the region. Also it is still not fully ascertained. So the problem persists and is actually spreading.
geographically and increasing in severity with increasing period of manifestation. It is causing suffering to large number of people. The 'social meaning' of this physical suffering has not been studied at all, so dealing with it is not even recognised as an issue.

The methodological Issues of combining a comprehensive study of various dimensions and their linkages qualitatively with a quantitative analysis continue to confront epidemiology. Measurement categories and tool must be devised so as to incorporate the social dimensions. We discuss these issues further in the next chapter.