Hydrogeol J (2017) 25:1165–1181
DOI 10.1007/s10040-017-1556-6

PAPER

Groundwater arsenic contamination and its health effects in India

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Received: 14 May 2016 / Accepted: 10 February 2017 / Published online: 17 March 2017

Abstract During a 28-year field survey in India (1988–2016), groundwater arsenic contamination and its health effects were registered in the states of West Bengal, Jharkhand, Bihar and Uttar Pradesh in the Ganga River flood plain, and the states of Assam and Manipur in the flood plain of Brahamaputra and Imphal rivers. Groundwater of Rajnandgaon village in Chhattisgarh state, which is not in a flood plain, is also arsenic contaminated. More than 170,000 tubewell water samples from the affected states were analyzed and half of the samples had arsenic >10 μg/L (maximum concentration 3,700 μg/L). Chronic exposure to arsenic through drinking water causes various health problems, like dermal, neurological, reproductive and pregnancy effects, cardiovascular effects, diabetes mellitus, diseases of the respiratory and gastrointestinal systems, and cancers, typically involving the skin, lungs, liver, bladder, etc. About 4.5% of the 8,000 children from arsenic-affected villages of affected states were registered with mild to moderate arsenical skin lesions. In the preliminary survey, more than 10,000 patients were registered with different types of arsenic-related signs and symptoms, out of more than 100,000 people screened from affected states. Elevated levels of arsenic were also found in biological samples (urine, hair, nails) of the people living in affected states. The study reveals that the population who had severe arsenical skin lesions may suffer from multiple Bowens/cancers in the long term. Some unusual symptoms, such as burning sensation, skin itching and watering of eyes in the presence of sun light, were also noticed in arsenicosis patients.

Published in the special issue "Hydrogeology and Human Health"

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Keywords India · Arsenic · Groundwater monitoring · Contamination · Health

Introduction

During the last 100 years, high concentrations of arsenic of natural geochemical origin have been detected in groundwater in many countries around the world. To date, arsenic in groundwater has been identified in 105 countries, with an estimate of the exposed population of >200 million worldwide at concentrations greater than the World Health Organization (WHO) guideline value for arsenic of 10 μg/L (Murcott 2012; Naujokas et al. 2013). Arsenic is a metalloid element which is generally present in natural deposits and is exposed through weathering, erosion of rocks/soils and volcanic emissions. The anthropogenic sources of arsenic contamination in groundwater may be due to mining activities, metal smelting, and electronic manufacturing processes (Naujokas et al. 2013). The major route for arsenic exposure to humans is through drinking water where it is typically present in inorganic forms of either arsenite [As (III)] or arsenate [As (V)], while another significant pathway of arsenic contamination is through the food chain (Bhattacharya et al. 2012; Halder et al. 2014). When contaminated water is used for crop irrigation, it enters the food chain (Rasheed et al. 2016); thus, when the crops are being consumed, the people are exposed to arsenic. Arsenic-related major health hazards through chronic exposure of contaminated groundwater for drinking and cooking have been registered in several countries and the Asian countries are found to be the most affected. The health effects of arsenic in Argentina were first reported in 1917 (Ayeza 1917). Skin disorders were found among residents drinking water from wells in the province of Cordoba; this incidence was well known as 'Bell Ville disease' (Goyenechea. 1917). During the 1920s, in south-western Taiwan, villagers used groundwater for drinking to avoid the microbiological contamination found in surface waters. The presence of arsenic in the groundwater eventually led to arsenic poisoning of those who ingested the water. In the affected areas, villagers also suffered from a peripheral vascular disease that can lead to gangrene, known as Blackfoot disease (Chen et al. 1985, 1986). During the 1960s, groundwater arsenic contamination and its health effects were noticed in Lagunera region of Mexico (Cebrian et al. 1983). These are the three well-known countries where large numbers of people have suffered from various diseases related to arsenic toxicity—in fact, a significant portion of the database generated on health effects of arsenic in humans is based on these studies. Currently, all these three countries have provided alternative safe sources of drinking water.

In India, the first groundwater arsenic contamination was reported in 1976 from Chandigarh, North India, when some people were identified as suffering from arsenic toxicity (Datta 1976). Eight years later, in 1984, groundwater arsenic contamination and its health effects in the lower Ganga plain of West Bengal was reported (Garai et al. 1984). Researchers from the School of Environmental Studies (SOES), Jadavpur University, joined in the efforts to assess the levels of groundwater arsenic contamination in this region at the beginning of 1988. During the SOES field survey of India, lasting 28 years, groundwater arsenic contamination and its health effects have been reported from West Bengal, Jharkhand, Bihar, and Uttar Pradesh in the flood plain of the Ganga River, and Assam and Manipur in the flood plain of Brahamaputra and Imphal rivers. Groundwater of Rajnandgaon village in Chhattisgarh state is also arsenic contaminated and some people had arsenical skin lesions including cancer but the source of arsenic in Chhattisgarh is not from the flood plains of Newer Alluvium (Holocene) as in Ganga, Brahmaputra, and Imphal rivers. Arsenic groundwater contamination in Chhattisgarh is due to natural deposition of arsenic-rich pyrite and its mobilization (Acharyya et al. 2005). The magnitude of arsenic contamination in Chhattisgarh state is much less compared to flood plain contamination. Arsenic contamination of groundwater in gold mining areas is known, but people suffering from arsenic toxicity including cancer has only been confirmed from Karnataka state (Chakraborti et al. 2013). Groundwater arsenic contamination from industrial waste release in India has also been reported (Sekhar et al. 2003); however, people suffering seriously from arsenical toxicity due to the consumption of groundwater contaminated by a pesticide 'Paris green (copper acetoarsenite)' has been recorded in Kolkata city also (Chatterjee et al. 1993; Mazumder et al. 1992).

Geology, geomorphology, and hydrogeology of the study area

The present study area consists of five states in the Ganga–Brahmaputra plain in India (Uttar Pradesh, Bihar, Jharkhand, West Bengal and Assam), and one North Eastern Hill state, Manipur. Figure 1 shows the geological map of the Ganges and Brahmaputra drainage basins. The Ganga plain is geomorphological characterized and predominated by alluvial deposit of Quaternary age. Three states (Uttar Pradesh, Bihar and West Bengal) of the current study area are located in this zone. It has been postulated that the great alluvial tract of the Brahmaputra River, which is by nature a geo-synclinal basin, formed concomitantly with the Himalayas to the north and is composed of alluvial and more recent Pleistocene sediment deposits (Goswami et al. 2014; Singh and Goswami 2011). A study based on mineralogical and stratigraphic data indicates that the two rivers (Ganges and Brahmaputra) changed their positions several times during the Holocene (Heroy et al. 2003), resulting in deposition of the sediments, which are considered very fertile (Kumar et al. 2010). The area of

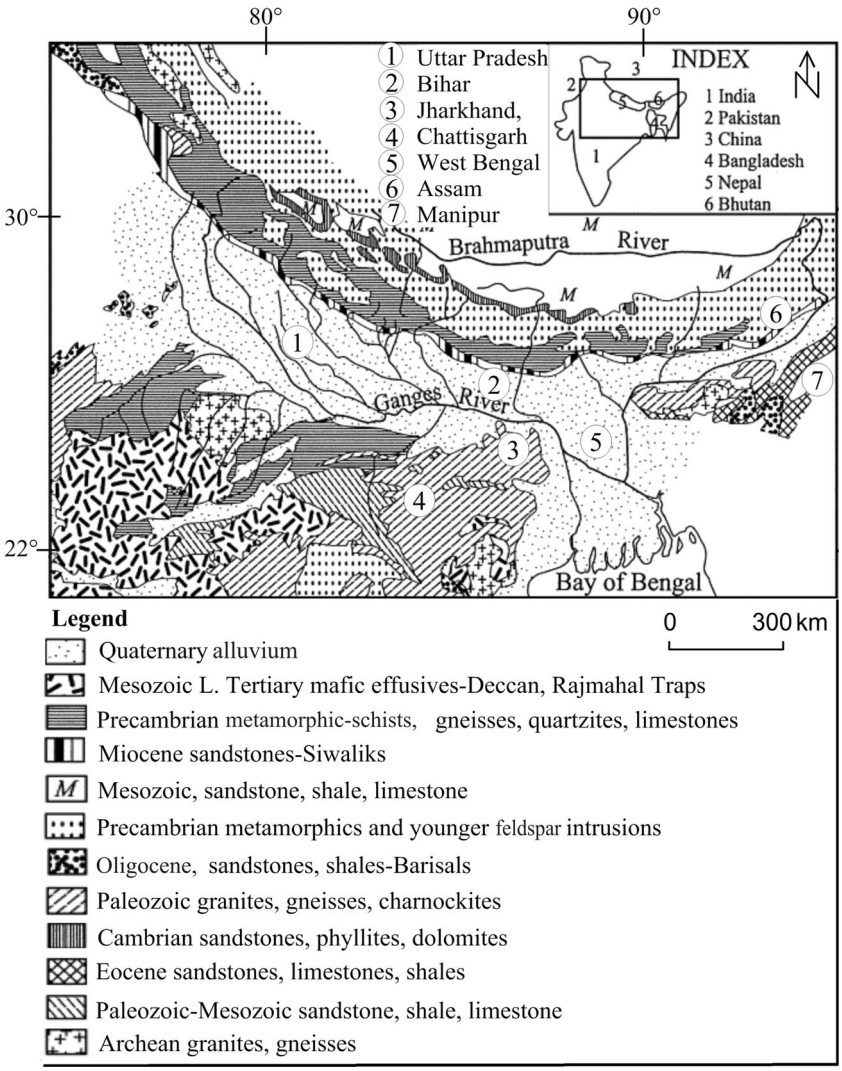

Fig. 1 Geological map of the Ganges and Brahmaputra drainage basins, focusing on states: *Uttar Pradesh*, *Bihar*, *Jharkhand*, *Chhattisgarh*, *West Bengal*, *Assam* and *Manipur* (modified from Heroy et al. 2003)

Jharkhand which was formed in the Paleozoic age consists of mainly granites, gneisses and charnockites (Heroy et al. 2003). The Manipur state comprises geologically young rock formations that were uplifted by the Tertiary orogeny of the Himalayas from the shallow bed of the Tethys Sea. The rocks are dominantly Tertiary and Cretaceous sediments with minor igneous and metamorphic rocks (Chakraborti et al. 2008).

Groundwater arsenic-contaminated states and magnitude of contamination in India

Figure 2 shows the major affected states and the magnitude of groundwater arsenic contamination across India. The chronological years of discovery and places of incidents of groundwater arsenic contamination in India is given in Table 1. Table 2 shows the groundwater arsenic concentration status in the different states considered in the present study. More than 170,000 water samples from tubewells were analysed from all surveyed states of India and half of the samples had arsenic greater than 10 μg/L. The maximum arsenic concentration was detected in a tubewell from West Bengal as 3,700 μg/L, which was 370 times higher than the WHO guideline value (10 μg/L) and 74 times higher than the Indian standard of arsenic (50 μg/L) in drinking water. Elevated concentrations of arsenic in groundwater were also detected in other states such as Bihar and Uttar Pradesh. Altogether 13.85 and 6.96 million people from all surveyed states were exposed to arsenic greater than 10 and 50 μg/L, respectively.

Sources of arsenic and mechanisms of arsenic leaching to groundwater

In nature, arsenic occurs as a component of over 245 minerals, usually ores containing sulfide, along with trace metals (Mandal and Suzuki 2002). The weathering of rocks and

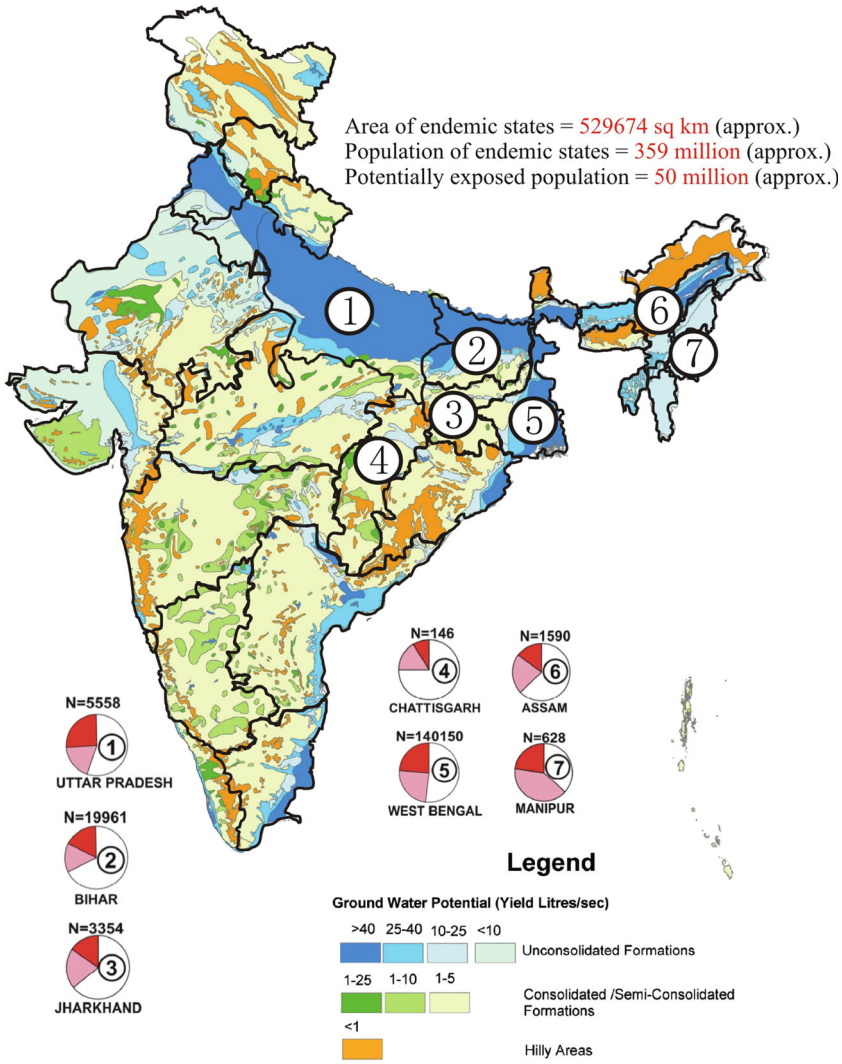

Fig. 2 Major affected states and magnitude of groundwater arsenic contamination in India along with the available groundwater potential in India

Table 1 The chronological years of discovery and places of groundwater arsenic-contamination incidents in India

Year	Place	Reference
1976	Chandigarh, North India	Datta 1976
1984	West Bengal	Garai et al. 1984
1999	Madhya Pradesh (now Chhattisgarh)	Chakraborti et al. 1999
2003	Bihar	Chakraborti et al. 2003
2004	Uttar Pradesh	Chakraborti et al. 2004
2004	Jharkhand	Nayak et al. 2008
2004	Assam	Chakraborti et al. 2004
2007	Manipur	Chakraborti et al. 2008
2009	Allahabad–Kanpur, Upper Ganga Plain	Chakraborti et al. 2009
2013	Karnataka	Chakraborti et al. 2013
2014	Majuli, Assam	Goswami et al. 2014

minerals appears to be the main source of arsenic in the soils, rocks, natural waters and organisms; mobilized is through a combination of natural processes such as weathering reactions, biological activity, transport, precipitation, volcanic emissions as well as through a number of other anthropogenic activities (Smedley and Kinniburgh 2002). It has been reported, based on the arsenic contamination scenario in Asia, that the floodplain of the rivers originating from the Himalayan Mountains and Tibetan Plateau region are also having groundwater arsenic contamination issues (Chakraborti et al. 2004). Based on this, arsenic contamination has been noticed in West Bengal, Bihar, Jharkhand, Uttar Pradesh in the Gangetic plain, Manipur in the north eastern Hill states, Brahmaputra plain in Assam, and PMB (Padma-Meghna-Brahmaputra) plain in Bangladesh. The presence of groundwater arsenic contamination found in Iran, Pakistan, China, Lao PDR, Vietnam, Myanmar, Cambodia, and Thailand also can be linked with the floodplain of the rivers originating from the same region.

Table 2 Groundwater arsenic contamination in the states of India

Parameter	West Bengal	Bihar	Uttar Pradesh	Jharkhand (Sahibganj district)	Assam	Manipur	Chattisgarh (Rajnandangaon district)
Area (km^2)	88,750	94,163	238,000	1,600	78,438	22,327	6,396
Population (in millions)	80.2	83	166	1	31.1	2.29	1.5
Arsenic-affected area (km^2)	38,861	21,271	10,375	725	8,822	2,238	6,396
Total No. of arsenic-affected districts (arsenic >50 μg/L)	12	12	3	1	3	4	1
Total No. of arsenic-affected blocks (arsenic >50 μg/L)	111	36	7	3	7	-	-
Total No. of villages where groundwater arsenic >50 μg/L	3,417	235	69	68	-	-	-
Total No. of hand tubewell water samples analyzed	140,150	19,961	4,780	3,354	1,590	628	146
% of samples having arsenic >10 μg/L	48.1	32.70	45.48	36.1	47.61	63.3	25.34
% of samples having arsenic >50 μg/L	23.8	17.75	26.51	15.4	15.47	23.2	8.22
Range of arsenic concentrations recorded (μg/L)	ND–3,700	ND–2,182	ND–3,191	ND–1,018	ND–383	ND–502	ND–880
Total No. of biological samples analyzed (hair, nails, urine)	39,624	1,833	258	367	-	57 (urine)	-
Total No. of people screened by medical group of SOES-JU	96,000	3,012	989	522	-	-	-
Total No. of people registered with arsenical skin lesions	9,356	457	154	71	-	-	80
Total No. of people drinking arsenic-contaminated water >10 μg/L (in millions)	9.5	3.1	1.1	0.15	-	-	-
Total No. of people drinking arsenic-contaminated water >50 μg/L (in millions)	4.6	1.7	0.6	0.06	-	-	-
Total population potentially at risk from arsenic contamination >10 μg/L (in millions)	26	9	3	0.4	1.2	1	-

ND not detectable

These places have been shown as red dots in Fig. 3. Numerous studies have been conducted to identify the leaching mechanism of arsenic from the sediment to aquifer and a number, such as oxidation, reduction, and recent inflow of

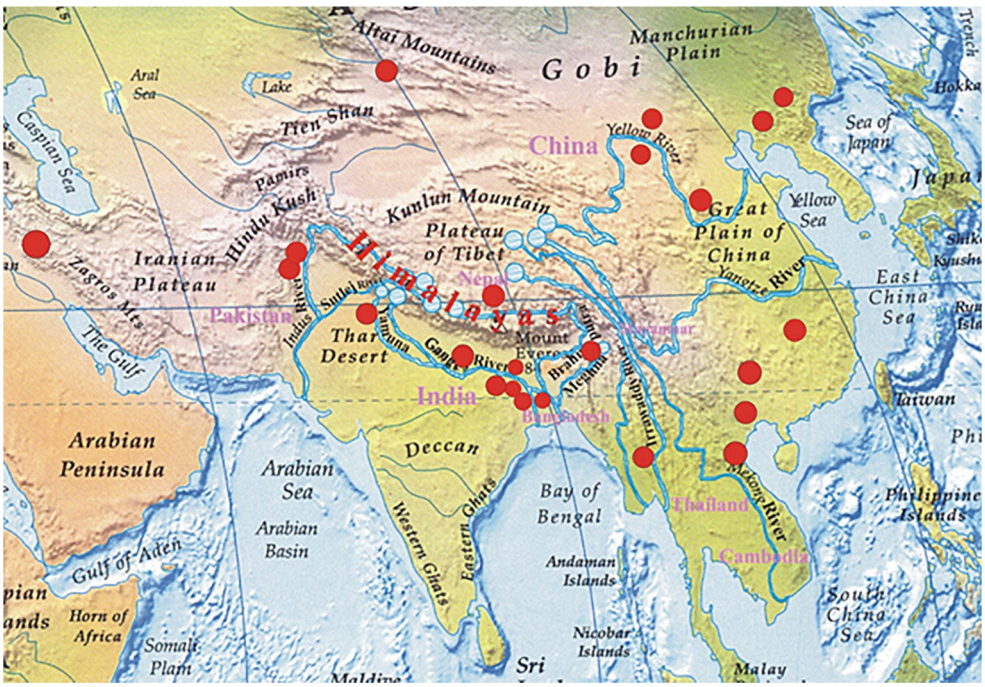

Fig. 3 The floodplains of the rivers originating from the Himalayan Mountains and the arsenic-contaminated areas in Asia. These areas are *circled in red*

carbon, have been proposed which possibly substantiate individually or collectively depending upon the geochemistry of the aquifer and organic matter present in the sediments (Ahmed et al. 2004; Anawar et al. 2003; Bhattacharya et al. 1997; Chakraborti et al. 2001; Chowdhury et al. 1999; Das et al. 1996; Harvey et al. 2002; Mukherjee and Bhattacharya 2001; Nickson et al. 1998, 2000; Ravenscroft et al. 2001). A study from West Bengal reported that metal-reducing bacteria play a crucial role in arsenic release from sediments (Islam et al. 2004). It was also reported from Bangladesh that microbially or geochemically mediated reductive dissolution of arsenic associated with Fe oxyhydroxides is the primary reason for arsenic release, and that these reducing conditions are caused by decomposition of organic matter (Anawar et al. 2011). The existing strong correlation between the arsenic level and prevalence of high concentrations of iron, phosphate, and ammonium ions, along with the weathering of carbonate and silicate minerals and surface-water/groundwater interactions, ion exchange, and anthropogenic activities (such as excessive groundwater withdrawal for agricultural irrigation) seem to be the primary processes causing arsenic contamination in groundwater (Kumar et al. 2010). From various studies of the Bengal delta, it is now well recognized that heavy withdrawal of groundwater is also responsible for arsenic contamination in groundwater (Harvey et al. 2002).

Arsenic-affected children in affected states of India

Infants and children are far more susceptible to the adverse effects of toxic substances than adults (NRC 1999). From this study on arsenic-affected states in India, it was noticed that children (usually) under 11 years of age do not show arsenical skin lesions although their biological samples contain high levels of arsenic; however, exceptions were observed when (1) arsenic content in water consumed by children is very high (≥1,000 μg/L) and (2) arsenic content in drinking water is not alarmingly high (500 μg/L) but the children's nutrition is poor (Rahman et al. 2001). High arsenic content in their biological samples prove that children in the arsenic affected areas of the Ganga-Meghna-Brahmaputra plains may not show physical manifestations but are sub-clinically affected (Chakraborti et al. 2004).

Children are at double risk as their bodies are frailer and when their system tries to expel the poison their internal organs are affected severely. This in turn retards their further growth, both physical and mental. During the last ~20 years of the preliminary survey, about 8,000 children from arsenic affected villages of affected states were screened and the survey registered about 4.5% of children with arsenical skin lesions; thus, the future generation is in significant danger. Researchers have witnessed the unimaginable sufferings of children in arsenic affected villages in many states of India, which is carrying forward generation after generation. Madanpur is a remote village in the Bhagabangola block in the Murshidabad district, West Bengal, where researchers visited two times at an interval of 10 years and witnessed apathy towards this situation. The first time, in 1992, among the 300 villagers of Madanpur, half of them had symptoms of arsenical skin lesions. Most of them were suffering from malnutrition and 40% of the children between 6 and 11 years had visible symptoms of arsenical skin lesions. The three tubewells that villagers of Madanpur used for withdrawing drinking water had an average arsenic level of 700 μg/L. During the second visit, in 2002, researchers noted that nothing had improved at all. Some of the previously identified children had died or lost their strength to work; they had become very thin young adults of very poor health. Table 3 shows the dermatological symptoms of 12 children from Murshidabad district.

Arsenic-related health effects in the chronically exposed population in the affected states of India

Mechanisms of arsenic toxicity in humans

Arsenic poisoning falls into two types, acute and chronic. Acute toxicity immediately results in nausea, vomiting, abdominal pain, severe diarrhoea and soon after peripheral neuropathy and other symptoms (NRC 1999). Chronic arsenic toxicity is usually felt after a prolonged period of ingestion of contaminated water/source, resulting in multisystem diseases. Arsenic is now a well-documented human carcinogen; however, there is no medicine to treat chronic arsenic poisoning. The focus of management is to reduce arsenic at the source.

In the gastrointestinal tract of humans, 70–90% of arsenic from the source, e.g. drinking water, is absorbed irrespective of the arsenite, As (III), or arsenate, As (V), state. Once ingested, inorganic arsenic is readily absorbed from the gastrointestinal tract and transported by blood to various organs in the human body (IPCS 2001). After entering the cell, As(V) is reduced to As(III) by glutathione (GSH). Arsenite has high affinity for the thiol (-SH) group; thus, arsenite deactivates enzymes, other functional proteins, and low molecular weight compounds such as GSH and cysteine blocking the active -SH group. Arsenic can exert its toxic effects through impairment of cellular respiration by inhibition of various mitochondrial enzymes and uncoupling of oxidative phosphorylation (IPCS 2001).

The As^{3+} species can react with the -SH group of protein and enzymes, thereby making them inactive, and increases reactive oxygen species in the cells causing cell damage. It is also reported that arsenic could inhibit 200 enzymes in the body (Chakraborti 2011). It has been regarded that multisystem noncancerous effects could be due to deactivation of

Table 3 Dermatological features of 12 children from Murshidabad district, West Bengal

Block and village	Sex/age	Melanosis						Keratosis					Non-pitting edema	No. of years with chronic bronchitis	SCC
		Palm		Trunk		Leu	WB	Palm		Sole		Dorsal			
		S	D	S	D			S	D	S	D				
Bhagowangola-2, Madanpur	M/11	–	+	++	+	+	+	+	–	+	–	–	–	3	–
Bhagowangola-2, Madanpur	M/10	+	+	++	+	–	+	+	–	+	–	–	–	1	–
Bhagowangola-2, Benipur	F/5	–	+	+	+	–	–	+	+	+	+	–	–	–	–
Bhagowangola-2, Baligram	F/11	–	+	+	–	–	–	–	–	–	–	–	–	–	–
Suti-2, Dharampur	F/10	–	+	+	+	–	–	+	+	+	+	–	–	–	–
Jalangi, Udaynagar Colony	F/10	–	++	++	++	–	++	++	++	++	++	–	–	2	–
Jalangi, Thakurngar Colony	M/10	–	++	++	++	–	++	+	+	+	+	–	–	–	–
Jalangi, Chakmathura	F/10	–	+	++	+	–	–	+	–	+	+	–	–	–	–
Domkal, Muktarpur	F/10	–	–	++	+	–	+	+	+	+	+	–	–	–	–
Domkal, Dakshinnagar	F/10	–	+	+	+	+	–	–	–	–	–	–	–	–	–
Raninagar-2, Amirabad	M/10	+	+	+	+	–	+	+	+	+	+	–	–	–	–
Raninagar-2, Hodaherampur	M/10	+	+	+++	+	–	++	++	+	+++	++	++	+	–	–

Abbreviations: + mild; ++ moderate; +++ severe; – not detected, *S* spotted, *D* diffuse, *Leu* Leuco melanosis, *WB* whole body, *SCC* squamous cell carcinoma

essential enzymatic functions by trivalent arsenic compounds and subsequent oxidative stress to cells. A few studies have detected in urine, along with all the four arsenic species [As (III), As(V), MMA(V) and DMA(V)], the presence of very unstable monomethylarsonous acid [MMA(III)] and dimethylarsinous acid [DMA(III)]. It is also considered that inorganic As^{3+} and the reduced forms—MMA(III) and DMA(III)—formed during methylation are highly reactive and contribute to the observed toxicity of inorganic arsenic. Arsenate (AsO_4^-) has similar structure as phosphate (PO_4^{3-}) and thus can substitute PO_4^{3-} in adenosine diphosphate (ADP). This substitution prevents conversion of ADP to ATP (adenosine triphosphate) that produces energy to cell. The affinity of arsenic deposition in tissues depends on the nature of the tissue and the type of arsenic species. Hair and nails are cysteine-containing proteins with an active -SH group. Trivalent arsenic has affinity for the SH group. In keratin tissues such as hair, nails, sole, and palm, cumulative deposition of arsenic occurs. During the past two decades, five monographs (IARC 2012; 2004; IPCS 2001; NRC 1999, 2001) along with a large number of reports and special issues have been published to include the research activities related to chronic arsenic exposure and various carcinogenic and non-carcinogenic health effects.

It is evident now that inorganic arsenic exposure deactivates the function of enzymes, some important anions, cations, transcriptional events in cells and causes other direct or indirect effects. Such activities of inorganic arsenic result in numerous illnesses. Repeated epidemiological investigations confirmed such illness—examples are (1) dermal effects, (2) cardiovascular effects, (3) respiratory effects, (4) gastrointestinal effects, (5) endocrinological effects (diabetes mellitus), (6) neurological effects, (7) reproductive and developmental effects, (8) cancers, and (9) other effects.

Dermal effects-A

Arsenic poisoning is mainly caused by ingestion of contaminated underground water. Signs and symptoms of arsenicosis depend on (1) arsenic concentration in drinking water; (2) the volume of water consumed; (3) duration of consumption; (4) nutritional status (immunity) of an individual. To get a clinical symptom it may take about 6 months to 10 years or even more.

The stages of arsenicosis can be divided into (1) the pre-clinical stage, (2) clinical stage, (3) stage of internal complications and (4) the stage of malignancy. Clinical manifestations can be divided into minor and major dermatological signs. The minor signs are: Mee's line on nail, pigmentation on tongue, conjunctival congestion, non-pitting oedema (Fig. 4a). The major signs are: diffuse and spotted melanosis or "raindrop pigmentation", leucomelanosis, spotted keratosis, diffuse and spotted keratosis on palm, dorsal keratosis, gangrene, multiple squamous carcinoma, Bowens. Malignancy from these diseases may occur after about 15–20 years of clinical onset of the disease as mono centric or multi centric squamous cell carcinoma. Malignancy in other organs—lungs, bladder, liver, uterus etc.—may also develop.

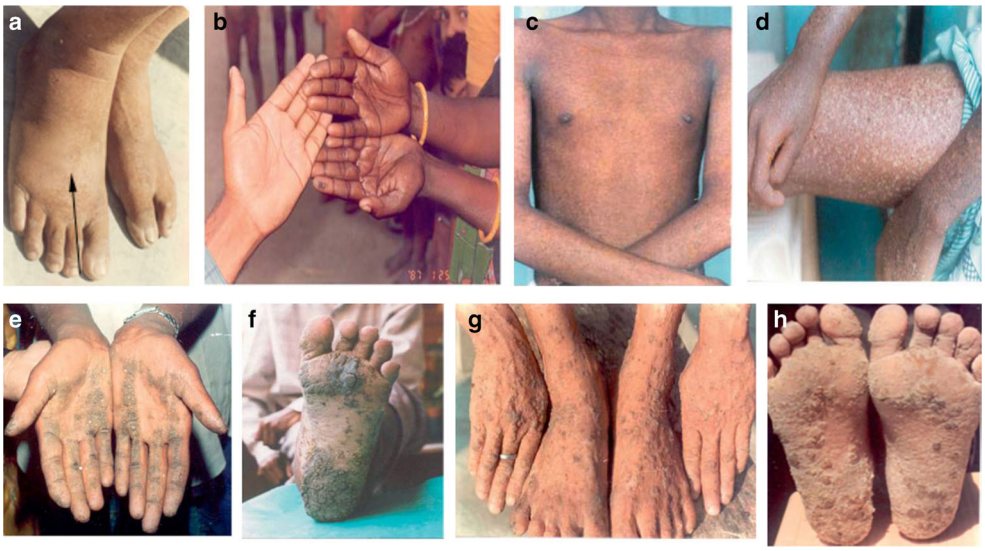

Fig. 4 Arsenical skin lesions **a** non-pitting Oedema, **b** diffuse melanosis, **c** spotted melanosis, **d** leucomelanosis, **e** diffused and nodular keratosis on palm, **f** spotted keratosis on sole, **g** dorsal keratosis, **h** an arsenicosis patient with severe keratosis who died of lung cancer

Dermal effects-B

To identify an arsenicosis patient, cutaneous manifestations are the most prominent characteristic. Normally, diffuse melanosis, that is darkening of skin in the body or palm, is the earliest symptom (Fig. 4b); however, it is not necessary that those suffering from arsenic toxicity will always have symptoms of diffuse melanosis. Usually, spotted pigmentation (Fig. 4c, spotted melanosis) is the second stage, appearing on the chest, back, or a limb, which is a very common symptom. Leucomelanosis (Fig. 4d), which is white and black spots side by side, is common in persons who have stopped drinking arsenic-contaminated water but had spotted melanosis earlier. Mucus membrane melanosis on gums, lips, and tongue may also be due to arsenic toxicity. Diffuse with nodular keratosis on the palms (Fig. 4e) and soles (Fig. 4f) is a sign of severe arsenic toxicity as is also rough dry skin often with palpable nodules (spotted keratosis) in the dorsum of hands, feet, and legs (Fig. 4g).

There are many diseases, however, that mimic arsenical dermatosis—for example, Addison's disease, which mimics diffuse melanosis; xeroderma pigmentosum is similar to spotted melanosis, and Verruca vulgaris mimics spotted keratosis. A combination of pigmentation (spotted melanosis) and nodular rough skin (spotted palmoplantar keratosis) almost points to arsenic toxicity excluding hundreds of causes of isolated pigmentation and nodular rough skin. In the case of any doubt, hair and nail analysis for arsenic will reveal the truth. There is always the question that at what concentration and drinking for how long will one show arsenical skin lesions? From the literature survey, some cases of arsenic-related symptoms have been reported at 100 or 200 μg/L concentration, but 300 μg/L exposure for a couple of years is considered adequate for the appearance of arsenical skin lesions. With high arsenic concentrations in water (say 1,000 μg/L), the skin lesions may appear within 6 months. It has also been noted that severe symptoms of arsenical skin lesions might serve as markers for other outcomes including skin cancer and internal cancer. Figure 4h shows a patient with severe diffuse and nodular keratosis who died of lung cancer. More common hyperkeratosis appears earlier than skin cancer. It is the opinion of toxicologists that the appearance of arsenical skin lesions indicates severe internal damage. As there is no medicine for arsenic toxicity, arsenic-safe water and nutritious food are considered the only remedy. It has also been reported that mild diffuse melanosis (+, mild) and mild spotted melanosis (+) may disappear with use of safe water and nutritious food. For persons affected with moderate skin lesions (spotted melanosis (++, moderate) and/or spotted keratosis (++), the skin lesions may reduce but may not vanish. For severe cases, even after several years, skin lesions especially keratosis continue even when hair, nails, and skin indicate a safe level of arsenic in the body. The spotted melanosis usually turns to leucomelanosis. Another unique feature is where the keratosis reappears even after it has been removed by ointment or surgery. It was also noticed that males had higher prevalence of skin lesions than females exposed at similar levels of arsenic through ingestion.

Further, it has been noticed that in a few cases when all members of a big family had arsenic-affected manifestations and were drinking water from hand tube-wells contaminated with a high concentration of arsenic, one or two members of the family had no skin lesions although hair, nails, and urine had high concentrations of arsenic the same as all family members.

Neurological effects

Neurological effects of acute arsenic exposure are more serious and long lasting compared to those of sub-acute or chronic

exposure. Acute arsenic toxicity affects both central and peripheral nervous systems, whereas chronic arsenic toxicity affects predominantly the peripheral nervous system. The severity usually decreases with time but may last for several years or even lifelong. Neurological effects from chronic arsenic exposure through drinking groundwater have been reported from many countries; however, more incidents of it are from Asian countries where more than 100 million people are considered to be exposed chronically to arsenic-contaminated groundwater (Chakraborti 2011).

The researchers associated with the project reported here have studied several groups of groundwater arsenic-exposed subjects from several arsenic-contaminated districts in states of India (Ahamed et al. 2006a; Chakraborti et al. 1999, 2016a, 2016b; Mukherjee et al. 2003, 2005; Nayak et al. 2008; Rahman et al. 2001) suffering from arsenic related neurological toxicity. Most of the districts investigated are highly contaminated with arsenic and people have arsenical skin lesions. The arsenic-exposed subjects were studied for their dermatological, neurological, and other non-neurological systemic involvements, including malignancies and obstetric outcomes.

Neurological observations were recorded in all subjects for items considered consistent with peripheral sensory and motor neuropathy and for other neurologic abnormalities. Items included, to characterize neuropathy, were (1) pain and paresthesia (e.g. burning) in a 'stocking and glove' distribution, (2) numbness, (3) hyperpathia/allodynia, (4) distal hypesthesia (perception of pinprick, vibration, joint-position, touch sensations), (5) calf tenderness, (6) weakness/atrophy of distal limb muscles or gait disorder and (7) reduction/absence of tendon reflexes. The features of autonomic instability and central nervous system (CNS) involvement were also investigated (Ahamed et al. 2006a; Chakraborti et al. 1999, 2016a, 2016b; Mukherjee et al. 2003, 2005; Nayak et al. 2008; Rahman et al. 2001).

Electrodiagnostic studies were performed in many subjects to evaluate peripheral nerve and central pathway functions by interpreting nerve conduction (sensory and motor nerve), electromyography (muscle function), and evoked potentials (visual, brain stem auditory and somatosensory). Quantitative sensory testing (QST) was performed in some subjects for determination of perception thresholds of different modalities of sensation, e.g. vibration, thermal stimulus and pain.

The diagnosis of arsenic neuropathy was confirmed in arsenic-exposed subjects with features of neuropathy by the presence of arsenic typical skin lesions (arsenic dermopathy) and analysis of arsenic in hair, nails, urine and the water they were drinking, and after exclusion of other possible causes and alternative explanations (Ahamed et al. 2006a; Chakraborti et al. 1999, 2016a, 2016b; Mukherjee et al. 2003, 2005; Nayak et al. 2008; Rahman et al. 2001).

The neuropathy in arsenic toxicity was further categorized into sensory or sensorimotor types and graded according to severity. The presenting neuropathic features of the study groups of chronically arsenic-exposed subjects included: sensory features of distal paresthesia ranging from a low of 18.4% to a high of 57.2%, limb pains from 9 to 18.7%, and distal hypesthesia from 22 to 46.9%, outnumbering motor features of distal limb weakness or atrophy ranging from 3 to 15.3%.

The prevalence of neuropathy ranged from a low of 37.3% to a high of 60.24%, and sensory neuropathy outnumbered motor nerve involvement (Ahamed et al. 2006a, b; Chakraborti et al. 1999, 2016a, 2016b; Mukherjee et al. 2003, 2005; Nayak et al. 2008; Rahman et al. 2001). There was a sub-acutely affected group of arsenicosis with high prevalence of neuropathy in Bardhaman district of West Bengal, India, where 33 out of 38 cases neurologically examined, i.e. 86.8%, revealed features of neuropathy, of which 76.3% had sensory and 10.5% sensorimotor neuropathy (Mukherjee et al. 2003). In the study population even the children were found to be suffering neurological complications from chronic arsenic toxicity. The prevalence of neuropathy in children up to 15 years varied from 12.5 to 33.3% (Ahamed et al. 2006a, b; Chakraborti et al. 2016a; Nayak et al. 2008), which is less compared to adults.

A cross-sectional study investigated the intellectual function among 351 children age 5–15 years from West Bengal in 2001–2003 (von Ehrenstein et al. 2007). Intellectual function was assessed with six subtests from the Wechsler Intelligence Scale for Children as well as with the Total Sentence Recall test, the Colored Progressive Matrices test, and a pegboard test. Arsenic in urine and lifetime water sources (including during the pregnancy period) were assessed using measurements of samples from 409 wells. The study found associations between arsenic and reductions in the adjusted scores of the vocabulary test, the object assembly test and the picture completion test, although they did not find associations between long-term water arsenic concentrations and intellectual function (von Ehrenstein et al. 2007).

Electrophysiological study findings revealed that arsenic toxicity damaged sensory more than motor nerves and defined clinical as well as subclinical neuropathy. Abnormal sensory nerve functions were observed in 27–60% of patients of arsenicosis with neuropathy; motor nerve dysfunctions was observed in 16.7–40% of these patients and 10% of asymptomatic subjects tested positive by the standard nerve conduction studies (Mukherjee et al. 2003, 2005; Nayak et al. 2008). Limitations of standard nerve conduction studies prompted the use of quantitative sensory testing (QST) to study not only the large myelinated but also small myelinated and unmyelinated nerve fibers. In 60–95% of clinical neuropathy cases, one or more of the sensory parameters of vibration, thermal (cold, warmth) and pain perception thresholds were increased. Subclinical sensory neuropathy due to arsenic was detected

in 30–60% of asymptomatic cases by QST compared to only 10% by standard nerve conduction studies, QST is thus proved to be a helpful screening tool.

Arsenic neuropathy is a common occurrence. The occurrence may be clinical or subclinical, predominantly sensory with small fiber involvement, sensorimotor and/or mixed fiber neuropathy, or it may be the precipitating factor for associated complications like non-healing ulcers in distal parts of extremities.

Central nervous system (CNS) and other neurological features in arsenicosis

The features of CNS involvement were important findings in the subjects involved in this study, whether neuropathic or not. Mood changes, with depression, irritability, anxiety, and/or reduced concentration, were common and affected their occupational and family activities (Mukherjee et al. 2003). Sleep abnormalities and headaches (2%) were more common than in control subjects (Mukherjee et al. 2003, 2005). Other neurological findings ranging from 9 to 13.2% comprised of tremors, proximal limb wasting, decreased vision not due to ophthalmic causes, decreased hearing (aural cause excluded), and decreased libido (Mukherjee et al. 2003, 2005).

Reproduction and developmental effects

Studies in both humans and animals have confirmed that inorganic arsenic and methylated species of arsenic can cross the placenta (ATSDR 2007). This information is an indication that arsenic may affect reproductive and developmental health effects.

Chronic human exposure to arsenic can adversely affect reproductive performance apart from other health hazards (ATSDR 2007). Several epidemiological studies have revealed the association between chronic arsenic exposure and adverse pregnancy outcome (IARC 2004; IPCS 2001). As part of this project, studies were carried out in different villages of Murshidabad district of West Bengal (Mukherjee et al. 2005), in villages in Bihar (Chakraborti et al. 2003, 2016a, 2016b), villages in Uttar Pradesh state (Ahamed et al. 2006a), and one village in the Sahibganj district of Jharkhand state (Nayak et al. 2008).

The study population was composed of married women of the reproductive age group 18–45 years who previously had at least one pregnancy. Researchers enlisted the respondents through house to house visits, and the majority were of lower socio-economic status. The respondents had been drinking arsenic-contaminated water for 5 years or more. Information was collected on each respondent's lifetime pregnancy history, which included the number of pregnancies, spontaneous miscarriage, stillbirth, preterm birth, lower birth weight and neonatal death. These women were examined clinically and their obstetric history was analyzed in detail. These studies were carried out over a 14-year period. The subjects were categorized into different groups according to the level of contamination of arsenic in drinking water; high contamination (90–800 μg/L) was associated with a high rate of stillbirths and neonatal mortality compared to the group of women supplied with arsenic-safe drinking water. From the arsenic-contaminated district of Murshidabad, West Bengal, women exposed to high arsenic-contaminated groundwater (284–1,474 μg/L) for 5–10 years had more spontaneous abortions (miscarriages), stillbirths, preterm birth, low birth weight, and neonatal deaths. Most of the women had arsenical skin lesions. During the survey in the Murhsidabad district of West Bengal, a woman was found with arsenical skin lesions whose pregnancies had been severely affected (the first pregnancy had preterm birth, the second pregnancy resulted in spontaneous abortion, and the third resulted in early neonatal death; arsenic content in the drinking water was 1,617 μg/L and arsenic content in the woman's urine was 1,474 μg/L. The WHO guideline value for arsenic in drinking water is 10 μg/L. The average concentration of arsenic in urine in the unexposed population is usually less than 100 μg/L (ASTDR U 2007).

von Ehrenstein et al. (2006) studied pregnancy outcomes and infant mortality among 202 married women in West Bengal between 2001 and 2003. Reproductive histories were ascertained using structured interviews. Arsenic exposure during each pregnancy was assessed, and involved 409 water-source wells. Odds ratios for spontaneous abortion, stillbirth, neonatal mortality, and infant mortality were estimated with logistic regression based on the method of generalized estimating equations. Exposure to high concentrations of arsenic (≥200 μg/L) during pregnancy was associated with a six-fold increased risk of stillbirth after adjustment for potential confounders. Arsenic-related skin lesions were found in 12 women who had a substantially increased risk of stillbirth. No association was found between arsenic exposure and spontaneous abortion or overall infant mortality. This study adds to the limited evidence that exposure to high concentrations of arsenic during pregnancy increases the risk of stillbirth; however, there was no indication of the increased rates of spontaneous abortion and overall infant mortality that have been reported in some studies (von Ehrenstein et al. 2006).

Cancer: the future danger

At present, the International Agency for Research on Cancer (IARC), WHO, US Environmental Protection Agency (US EPA), and other health protection authorities consider arsenic to be causing skin, lung, liver, urinary bladder, and kidney cancer. It is reported that life-time consumption of arsenic-contaminated water at 1 L/day having arsenic at 50 μg/L concentration could cause cancer in 13 people out of 1,000

population (Smith et al. 2002). Hossain et al. (2013) reported that, in a tropical country, an adult drinks about 6 L of water per day; hence, the risk of cancer is much higher. In the cohort study reported here, 1,194 registered arsenic patients with skin lesions were re-examined between January 2009 and January 2010, out of the 2,384 villagers that had been screened earlier (1995–2000) from 33 villages and 16 blocks/*thanas* from some districts of West-Bengal (India) and Bangladesh. Only these patients were re-surveyed because the study's longer-term database contained the concentrations of arsenic in the hand-driven tube-wells they had used for drinking along with arsenic data on their biological samples and details of their skin lesions. Findings of this cohort study indicate that 14% of the patients examined earlier (who had arsenical skin lesions) had died with non-healing ulcers and 48% are currently suffering from Bowens and arsenic-related cancers. On the basis of this study (SOES 2010), it was concluded "Are Millions in Ganga-Meghna-Brahmaputra Plain already exposed to arsenic-contaminated water potentially at risk from cancer"? Figure 5 shows some of the patients with Bowens and cancer from arsenic affected villages of West Bengal during this cohort study.

In the Chak-Khor Gachi village of Baduria block, North 24 Pargana district of West Bengal, over a 15-year period, the researcher's medical group identified more than 35 arsenic-affected patients suffering from multiple Bowens and 28 of them had died from liver cancer by the time of the re-survey. Figure 6a shows a cancer patient and Fig. 6b shows a patient with keratosis, from Rajnandangaon, Chattisgarh state of India.

Even during the first survey, 10–15 years earlier, the researchers were not aware of arsenic-affected villages with incidences of cancer other than suspected Bowens, squamous or Basel cell carcinoma—of course other types of cancers were present but patients were not tested for other cancers. In the course of time, it has been established that arsenic can cause other types of cancers. Now, in arsenic-affected villages of West Bengal and other arsenic-affected states of India, lung cancer and liver cancer are more common. It is noted that the appearance of severe keratosis and Bowens indicate the possibility of other forms of cancer. The local authorities and community health service providers need to address this issue to prevent future cancer 'massacres' in arsenic-affected states of India.

Cardiovascular effects from arsenic toxicity

Apart from the highly prevalent neoplastic outcomes, development of vascular disease is also common among the arsenic endemic population. Vascular disorder refers to the dysfunction of vessels—arteries, veins, capillaries, including the heart. This dysfunction may be of two types: peripheral vascular disease such as dysfunctioning of small arteries and capillaries in the peripheral areas as the skin, feet, arms, etc., and cardiovascular disease (CVD) associated directly with the heart and larger arteries. The cardiovascular diseases generally manifested are arteriosclerosis, atherosclerosis, ischemia heart disease (which results in hypertension, HTN), heart blockage, cardiac arrest, stroke, and infarction, and in the periphery, arteriosclerosis, black foot disease, gangrene, etc. Epidemiological studies have revealed high arsenic exposure (>300 μg/L) associated with the manifestation of both peripheral and CVD from different parts of the world (Srivastava et al. 2009). Arteriosclerosis is the thickening of 'intima', the interior most walls of arteries. Atherosclerosis is a variation of arteriosclerosis, where the thickening is due to the deposition of fat or high-cholesterol plaque formation, which might be manifested due to arsenic toxicity (Simeonova and Luster 2004). A unique exhibition of peripheral arteriosclerosis associated with high arsenic concentration in drinking water may be shown in 'Blackfoot disease' which was common in southwestern Taiwan. The biological mechanism behind the arsenic-induced cardiovascular deformities may be as follows. Arsenic can produce reactive oxygen like hydrogen peroxide and hydroxyl radicals which can induce alterations of nitric oxide metabolism and endothelial function (Chakraborti 2011a). Arsenic-induced cardiovascular disease in humans is now regarded as interrelated with genetic, nutritional, and environmental factors. The adverse cardiovascular manifestation in humans resulting from long-term arsenic exposure, is an independent risk factor and may be persistent and irreversible (Wang et al. 2007). Recent studies also indicate the cardiovascular implications of low to moderate arsenic exposure by ingestion, in the form of high death rates due to CVD (Medrano et al. 2010), nonlinear blood pressure (Chen et al. 2007), and high pulse pressure (Chen et al. 2007), though there is no evidence of risk of hypertension (Abdul et al. 2015). Tchounwou et al. (2003) reported that individuals exposed to elevated inorganic arsenic levels in drinking water showed a range of health effects including peripheral vascular disease, non-cirrhotic portal fibrosis, nasal septum perforation, bronchitis, and polyneuropathy.

Very limited studies have been conducted on cardiovascular effects for arsenic poisoning in India. Guha Mazumder et al. (2012) reported the association of hypertension (HTN) among an arsenic-exposed population of 280 people, compared to a control of 100 in West Bengal. The study found a dose-effect relationship between arsenic exposure and arsenic levels in hair and HTN. No further association was found for HTN among the exposed population, with or without skin lesions. A study conducted by Das et al. (2012) on 103 exposed people from Murshidabad, West Bengal, compared to the population of 107 from uncontaminated areas of Medinipur, West Bengal, found elevated levels of inflammatory cytokines (IL6, IL8, LCP1) in the exposed population, associated with the high risk of atherosclerosis.

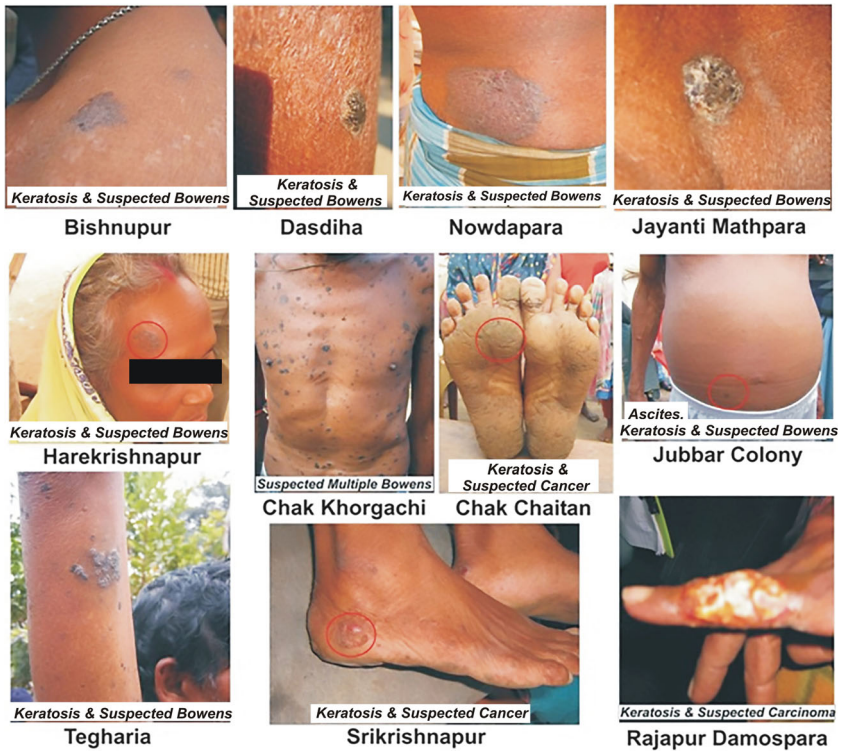

Fig. 5 Some patients with Bowens and cancer from arsenic-affected villages of West Bengal

Respiratory effects from arsenic toxicity

Inorganic arsenic shows a major adverse manifestation on the human respiratory system; the longer the exposure, the more grave the problem. One of the earliest studies on the effects of inorganic arsenic exposure toward pulmonary health was from a preliminary investigation conducted in Chile during 1970 (Borgoño et al. 1977). Several studies on arsenic-impacted areas of West Bengal have reported that ingestion of inorganic arsenic for a prolonged period causes respiratory problems, including coughs, chest sound, bronchitis, and shortness of breath.

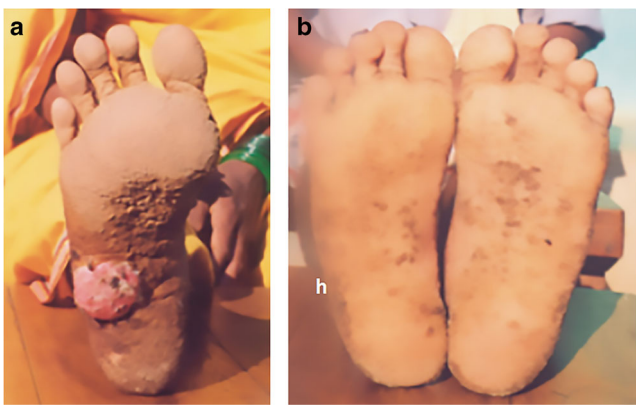

Fig. 6 **a** An arsenicosis patient with cancer and **b** keratosis on the soles, from Rajnandangaon, India

A cross-sectional survey was carried out in 1996 involving 7,683 participants in one arsenic-affected district of West Bengal, who were chronically exposed to arsenic-contaminated groundwater (Mazumder et al. 1998). One of the study objectives was to establish prevalence of pulmonary effects on the people that already have arsenic dermatological features. There was prevalence of coughs, shortness of breath, and chest sound in both males and females, and these symptoms rose with increasing arsenic content in water. The respiratory effects were more pronounced in those exposed to high concentrations of arsenic (≥300 μg/L). The study further concluded that chronic exposure to arsenic-contaminated water could cause respiratory effects (Mazumder et al. 1998).

Mazumder et al. (2005) conducted a study on the severity of chronic non-malignant bronchiectasis among the population of the arsenic-exposed region of West Bengal. Among the study's total population of 38 people (27 having arsenical skin lesions, and 11 having no skin lesions) they found a bronchiectasis severity score of 3.4 (±3.6) for the exposed population having skin lesions, compared to 0.9 (±1.6) in the population having no skin lesions. In a cross sectional study, De et al. (2004) assessed chronic arsenic poisoning on pulmonary dysfunction among the affected population in West Bengal compared with a control population. Among 107 exposed subjects, they found respiratory involvement in 33 (33.8%) of them. Most of the manifestation was obstructive lung disease, in 20 people (68.9%). Others suffered restrictive lung disease (1 person, 3.5%), mixed symptoms (8 people, 27.6%), and

malignancy (4 people, 12.2%). Ghosh (2013) showed the prevalent of nonproductive cough (49.31% subjects) and change in spirometry (23.28% subjects) as the most common symptoms in a study population of 73 from Arsenic Clinic, Institute of Postgraduate Medical Education and Research, Kolkata, West Bengal, over a period of 1 year and 4 months during 2008–2009. In a more recent study conducted in West Bengal comparing the respiratory effect of chronic low level (≤50 μg/L) arsenic exposure, Das et al. (2014) documented that the exposed population (11–50 μg/L; $n = 446$) had higher prevalence of upper and lower respiratory symptoms, dyspnea, asthma, eye irritation and headache than the control population (<50 μg/L; $n = 388$). The study further substantiated negative association between arsenic level and spirometric parameters. Bhattacharyya et al. (2014) investigated the pulmonary arterial system performance relative to the arsenic endemic population through high-resolution computerized tomography, among 194 arsenic exposed and 196 unexposed people. The exposed population showed higher prevalence of cough (odds ratio, OR, 3.23) and shortness of breath (OR 1.76), compared to the control population. Similar results were found in cases of other respiratory manifestations like bronchiectasis [mean ± SD (standard deviation): 2.41 ± 2.32 vs. 1.22 ± 1.48 (P <0.001)], pulmonary artery branch dilation—2.48 ± 2.33 vs. 0.78 ± 1.56, (P <0.001)— and pulmonary trunk dilation (0.26 ± 0.45 vs. nil) when compared to the control.

Gastrointestinal effect of arsenic toxicity

The gastrointestinal effect of arsenic toxicity may be broadly categorized by acute or subacute arsenic exposure and chronic arsenic exposure. Acute or subacute arsenic exposure, which is generally immediate but highly concentrated exposure, could result in overt gastrointestinal disturbances starting from mild to severe abdominal pain, cramping, and diarrhoea and most importantly to submucosal capillary damage from the absorbed arsenic (Tchounwou et al. 2003). The overall effects may lead to more serious conditions, like life-threatening haemorrhagic gastroenteritis and severe shock (Chakraborti 2011a). With chronic exposure when it is of low concentration, overt gastrointestinal symptoms may not be noticed or absent but some of the gastrointestinal disturbances could occur like mild esophagitis, gastritis, or colitis with respective upper and lower abdominal discomfort, anorexia, malabsorption, and weight loss (Tchounwou et al. 2003). In the West Bengal study area, 60 out of the 156 affected villagers chronically exposed to arsenic-contaminated groundwater showed dyspepsia (Mazumder et al. 1998). One of the detailed studies was conducted on 25,274 people screened from Murshidabad district of West Bengal; among the registered 4,813 patients having arsenical skin lesions, more than 50% of the patients showed gastrointestinal symptoms of anorexia, nausea, dyspepsia, altered taste, abdominal pain, enlarged liver and spleen, and ascites (collection of fluid in abdomen; Mukherjee et al. 2005). In another epidemiological study in West Bengal, nothing other than abdominal pain was observed in both the affected population drinking chronic arsenic-contaminated water (50–3,400 μg/L) and the control population (>50 μg/L; Mazumder et al. 2001). An investigation into the gastrointestinal impact of a population exposed to elevated arsenic concentrated water (>50 μg/L) but with the population not having any skin lesions (Majumdar et al. 2009) found only moderate prevalence of diarrhoea.

Endocrinological effects (diabetes mellitus) of arsenic toxicity

Ingestion of chronic levels of arsenic in drinking water is considered to be the reason for an increased risk of type 2 diabetes mellitus. To explain this, researchers are looking at the effects of arsenic on the endocrine system. It has been postulated that arsenic might act as an endocrine disruptor at doses as low as 0.4 μg/L arsenite (Kapaj et al. 2006). One study has substantiated that accumulation of arsenic in the pancreas inhibits the secretion of insulin and decreases the viability of the cells (Lu et al. 2011). There are numerous reports of the prevalence of diabetes in arsenic endemic areas of Taiwan (Tsai et al. 1999; Tseng et al. 2000), Bangladesh (Rahman et al. 1998), Millad County of Uttar Pradesh, India (Lewis et al. 1999), Cyprus (Makris et al. 2012), and Mexico (Coronado-González et al. 2007); however there is lack of prominent study to show the prevalence of diabetes mellitus among the arsenic endemic population in India overall.

Other health effects

Haematological effects are observed from both acute and chronic arsenic exposure and their results are anaemia, leukopenia, and thrombocytopenia. Reports are scanty for the population chronically exposed through drinking arsenic-contaminated groundwater and subsequent haematological effects. Most of the population suffering from arsenic skin lesions is from a poor socio-economic background. The following features were commonly observed during the 24-year field survey in the arsenic-endemic areas of India where people have been drinking arsenic-contaminated groundwater and had arsenical skin lesions:

1. Skin itching in response to sun light, burning and watering of eyes, weight loss, loss of appetite, weakness, lethargy and easily fatigued, thus limiting physical activities and work capacity.
2. Chronic respiratory complaints were also common. A chronic cough with or without expectoration was evident in more than 50% of the studied people. As reported by the villagers, the unique sound of ‘cough of arsenicosis’

from adjacent homes at night was reported to create an unusual atmosphere. The cough may be painful and sputum may contain blood to be misdiagnosed as pulmonary tuberculosis. In late stages, shortness of breath predominates.

3. Gastrointestinal symptoms of anorexia, nausea, dyspepsia, altered taste, abdominal pain, enlarged liver and spleen, and ascites were also observed in more than 50% of patients.
4. Moderate-to-severe anaemia was evident in nearly 30% of cases.
5. Conjunctival congestion and leg oedema were less common and found in 10% of the cases.

Is the WHO guideline value for arsenic in drinking water (10 μg/L) in the developing countries adequate?

The WHO guideline value for arsenic in drinking water is 10 μg/L. At present, in most of the arsenic-affected Asian countries, the permissible limit of arsenic in drinking water is 50 μg/L. Considering the large area and population potentially at risk from arsenic danger within world's arsenic scenario, India appears to be at the top of the list. It is important to note that the WHO guideline value (10 μg/L) was allocated on the basis of 2 L of drinking water per day, whereas in tropical arsenic-affected states in India, the average direct drinking water consumption is about 4 L of water per day, and total water intake (direct and indirect) is about 6 L/day (Hossain et al. 2013). In arsenic-affected areas of India, arsenic-contaminated groundwater is in use for agriculture and thus food is also arsenic contaminated, an additional source of arsenic to the consumer. It is also reported that a better nutritional status means that the population can resist arsenic toxicity (Chowdhury et al. 2000; Milton et al. 2004). By comparison, the WHO permissible limit for fluoride (F) in drinking water has long been 1.5 mg/L. For India, on the basis of the factors discussed above, the WHO has suggested reducing the permissible limit for F in drinking water in India from 1.5 to 1.0 mg/L and India has accepted this reduction; however, the permissible limit of arsenic in drinking water in India remains 50 μg/L. On the basis of the reasons discussed in the previous, the permissible limit for arsenic in drinking water in India logically should be less than 5 μg/L. Further, it is reported that the cancer risk to those drinking arsenic-contaminated water at 50 μg/L at 1.0 L/day is 1.3 people per 100 (Smith et al. 2002). A group of scientists has also opined that the WHO limit may not be safe for pregnant mothers and children (Osborn 2012). In the meantime, some parts of the world, considering the danger of the guideline value of arsenic 10 μg/L, already have reduced their permissible level of arsenic. New Jersey and South Carolina in the USA has accepted a 5-μg/L limit for their drinking and cooking, while for Australia it is 7 μg/L.

Conclusion

It appears from the overall study that the groundwater of some states in India is very contaminated with arsenic. Current statistics show that the groundwater of 7 states of India are affected with arsenic and this poses risk to 70 million people in terms of various arsenic health effects, including various forms of cancer. Children are most susceptible to arsenic poisoning. People who had been found in previous surveys to have arsenic keratosis, now suffer from multiple Bowens/ cancers in the long term. Even with higher arsenic standards (50 μg/L instead of 10 μg/L), authorities and communities are apparently not able to generally enforce the standards and some urgent intervention is needed to address the search for alternative sources of water or to implement small-scale/rural water treatment. Inhabitants of the Ganga-Meghna-Brahmaputra plains and other river flood plains have, for thousands of years, used the traditional sources of water like rivers, ponds, lakes, dug wells, and other water bodies for consumption. Even in the extreme southern part of India, rainwater harvest is a traditional procedure for drinking water. These water sources should be considered as alternative safe-water options to the affected communities, as opposed to deeper groundwater. Effective management of the vast surface water resources, and good nutrition, along with greater population awareness, could potentially minimize the impact of groundwater arsenic contamination in India.

Acknowledgements We thank all participants involved in this study for their extensive cooperation.

Compliance with ethical standards

Ethical approval This study was approved by the office of the Principal and Chairman, Institutional Ethics Committee (IEC) Medical College Kolkata, India and the Ethics committee, Jadavpur University, Kolkata, and informed consent was obtained from study participants.

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