2009

Consequences of Arsenic Contamination in Human Beings and Their Prevention by Applying Homoeopathic Principles

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CONSEQUENCES OF ARSENIC CONTAMINATION IN HUMAN BEINGS AND THEIR PREVENTION BY APPLYING HOMOEOPATHIC PRINCIPLES

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DEDICATED

TO

My Beloved Parents

DECLARATION

I do hereby declare that the thesis entitled '**Consequences of Arsenic Contamination in Human beings and their Prevention by Applying Homoeopathic Principles**' is prepared by me. This thesis is an outcome of one-year course work experience, field survey, microscopic tissue analysis in laboratory and application of medicines on the patients in rural area of Bangladesh, under the auspicious supervision of Dr. Md. Sarwar Jahan, Director and Professor of Institute of Environmental Science, University of Rajshahi, Bangladesh. The study was designed to innovate the preventive measures for the various consequences of arsenic through the application of Homoeopathic principles. The thesis is being humbly submitted to the Institute of Environmental Science, University of Rajshahi, Bangladesh for the degree of **Master of Philosophy** in Environmental Science. It is also declared that the contents of this thesis or any part of it were not submitted to any other institution for achieving any academic degree or diploma.

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CERTIFICATE

I have the pleasure to certify that the work presented in the dissertation entitled **'Consequences of Arsenic Contamination in Human beings and Their Prevention by Applying Homoeopathic Principles'** is an original research carried out by M Jahangir, registration no. 0022, session- 2004-05, in pertinent fulfillment of the requirements for the degree of Master of Philosophy (M. Phil.) in environmental science. To the best of my knowledge, this is the researchers own achievement and not a conjoint work. In my opinion M Jahangir has certainly made distinct contribution to the arena of environmental science through his research work. The thesis or thereof the part of it has not been submitted for any degree to any other university.

I also certify that the research work has been carried out under my direct supervision and the thesis is found satisfactory. I gladly recommend him to submit the thesis to the Institute of Environmental Science (IES), University of Rajshahi, Bangladesh for adjudication.

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ACKNOWLEDGEMENT

All praises are due to the Almighty Allah, who has given me the opportunity, courage, potency and perseverance to accomplish this job of bringing out the thesis into light.

It is a great pleasure for me to express my cordial gratitude and honor to my respected teacher and auspicious supervisor Dr. Md. Sarwar Jahan, Director and Professor of the Institute of Environmental Science, University of Rajshahi for his continuous encouragement, cordial guidance, heartiest cooperation and thoughtful suggestions during the whole period of my course and thesis works. In spite of his heavy involvement with academic and administrative works of the institute he sacrificed many many hours from his own time for the development of my thesis. Rather than I am grateful to him, not only to admit me in his institute but also for opening a door for homoeopathy in post-graduate studies under University of Rajshahi. It is unforgettable for me in my whole life.

Professor Dr. AKM Nazrul Islam, Department of Botany, University of Dhaka and Assistant Professor Dr. Md. Redwanur Rahman, were constant source of encouragement. Their constant guidance, support, painstaking reading of the drafts and numerous invaluable suggestions only made this research possible.

I am also grateful to the teachers of IES, Dr. Md. Golam Mostofa, Dr. Abul Kalam Azad, Mrs. Zakiya Yasmin and Mr. S M Shafiuzzaman and visiting teachers Dr. Md. Amjad Hossain and Dr. S Mahfuzar Rahman for their sincere cooperation during the course work.

Dr. M Shamsher Ali, Professor and vice-chancellor of South-East University, Dhaka, provided all types of inspirations to do researches and he wrote Government Universities to open a criterion of higher education for homoeopathy. Practically he

gave me a hand to do the bests in my life. I express my sincere gratitude from the core of my heart for his great contribution for stepping to the research work.

I am greatful to Chowdhury Mahmudul Hasan, Ex-Chairman, BCSIR for his direct and indirect co-operation for my research, and his contribution for Homoeopathy, as well as all indigenous medicinal system.

I am grateful to Professor (Ret.) Meghnad Saha, Carmichael Govt. College, Rangpur; Dr. Ashim Ranjan Barua, Associate Professor of Pathology, BSM Medical University; Assist. Prof. Dr. Gokul Chandra Bishwas, Sher-E-Bangla AK Fazlul Haq Govt. College, Chakhar, Barisal and Assist. Prof. M Rafiqul Islam, Brozomohan Govt. College, Barisal for their all kind of support and creative ideas during my research work.

Mr. Apurba Kumar Roy, Assist. Professor, Dept. of Genetic Engineering and Biotechnology, University of Rajshahi gratified me by his sincere cooperation during the microscopic analysis of the slides and I am also grateful to Mr. Kamrul Hassan, Prime Diagnostic Centre, Dhaka and Mr. Ruhul Amin, BIRDEM Hospital, Dhaka for helping me to prepare histological slides.

I am also obliged to Md. Aminul Islam, Md. Saiful Islam and Mahboob Shafi Shirajee, BHMS students and Mr. Gazi Khairul Alam for their cooperation and assistance in my field work for the research.

My warmest thanks to Hospital authorities, arsenic affected people and local people of Sonargaon and Sujanagar upazilas; without their cooperation this research work would not be possible.

I would like to express my heartiest thanks to all of my fellow friends specially Late Dr. Nur Islam, Dr. Md. Mustafizur Rahman Prodhan, Mr. Sudhir Chandan Biswas, Mr. Iqbal Bahar, Mr. Mohiuddin M Shahjahan Bhuian, Mr. Aminul Islam,

Mr. Nazrul Islam, Mr. Lookman Ali, Mr. Kamruzzaman and Mr. Rafiqul Islam Sheik for their unaulterated love, dua, affection, inspirations and all kinds of helps.

Last, but not least I cordially remember the inspiration and contribution of my family members specialy my beloved father and mother, without their dua the effort would not come up to the mark of satisfaction. My wife Dr. Selina Jahan, who inspired me all along with mental supports and shared my other jobs to carryout such a big work. My elder son, M. Shifat Hossain, my brother M. Alamgir Hossain and nephew Rakibul Hasan helped me to make the computer works for the research. I express my sincere thanks to them. My thanks to other two sons, M. Iffat Hossain and M. Rifat Hossain who were deprived of me in their very valuable time of education during my coursework and research period. I am also grateful to my two sisters Jebun Nessa and Ferdousi Begum for their all time support and dua for my research. I owe to my family members for their contributions.

I acknowledge the contributions of those who contributed in my research work, but I could not mention their names in this acknowledgement.

June, 2009 *M Jahangir*

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ABSTRACT

Identification of arsenicosis patient was done by the field survey of Haria and Ramgonj village of Sonargaon upazilla and Ahmadpur and Sagarkandi village of Sujanagar upazilla. Fifteen (15) from Haria, two (2) from Ramgonj, 60 from Ahmadpur and 14 patients were choosen from Sagarkandi village.

Then there had drawn a relation between age and arsenicosis and the relation between occupation of the patient and arsenicosis. The percentage of affected middle age groups (21-40) and (41-60) were respectively 76.48% and 11.76% among all affected people (15:2) and of all population only 16.04% were affected in the research area of Sonargaon upazila. In Sujanagar upazila, the percentage of affected middle age groups (21-40) and (41-60) were respectively 58.07% and 25.81% among all affected people (26:5) and of all population 62% were affected.

Occupationally all the patients were of lower middle and lower class. The main businesses of these patients were fish selling (37.5%) and service (12.5%) in the research area of Sonargaon upazila. All the women were house wife (31.25%). Maximum children were helping their parents instead of going to the school. The same picture found in Sujanagar upazila also. The main profession was business (32.3%). Maximum patients were shop keeper in little shops of Kashinathpur bazar, some were blacksmith, some were rickshaw and van puller and some were farmers; farmers and service holders were in the same rank (11.3%). Females were mainly house wife in

all villages (43.5%) and they were usually helping hands for their husbands. It was said that people of low earning were the main victim of arsenic.

Microscopic analysis was done for the keratosis of the palm of the patients. In microscopic view, there was found many germ keratoses to some giant keratosis in epidermis. Keratoses were formed in the keratin layers and they were black in colour, due to the deficiency of oxygen in cells. Other normal keratin cells of the slides were of natural skin colour. Big keratoses had an oval shaped hole in the centres. Affected keratins were flattened and rounded to the hole. It seems that the hole was belonging to a molecule of arsenic in the keratin layer. The hole can be say the eye of the keratosis.

After applying medicines to arsenicosis patients, the keratosis stopped developing instantly. And in all the cases no casualty found in these years. No evidence of concequences of arsenic found in whole research period to till now. Keratosis and ulceration cases recovered simultaneously.

Homoeopathic medicines *Arsenicum album* and *Antmonium Crudatum* scored respectively 81.25% and 55.6% recovery on arsenicosis and at the same time it prevents the concequences of arsenic contamination in human beings.

Abbreviation used

APSU	= Arsenic Policy Support Unit
BAMWSP	= Bangladesh Arsenic Mitigation Water Supply Project
BGS	= British Geological Survey
DFID	= Department of International Development
DPHE	= Department of Public Health Engineering
GOB	= Government of Bangladesh
NAMIC	= National Arsenic Mitigation Information Centre
NGO	= Non-Government Organization
NIPSOM	= National Institute of Preventive and Social Medicine
UHCH	= Upazilla Health Complex Hospital
UH&FPO	= Upazilla Health & Family Planning Officer
UNICEF	= United Nations International Children Emergency Fund
WHO	= World Health Organization

Scientific-

Å	= Angstrom ($1Å=10^{-10}$ meter)
As	= Arsenic (Atomic number 33) Diameter 0.238 nano meter ($1nm=10^{-9}m$)
As III	= Trivalent Arsenic
As V	= Pentavalent Arsenic
P^{53}	= Tumour protein 53, transcription factor in humans is encoded by the *TP53* gene.
CA	= Chromosomal Aberration
SCE	= Sister Chromatid Exchange, the exchange of genetic materials between two identical sister chromatids
AAS	= Atomic Absorption Spectrophotometer
AGT	= Average Generation Time
BMI	= Body Mass Index
BP	= Blood pressor
BFD	= Black Foot Disease
BWD	= Bell Will Disease
DWS	= Drinking Water Standard
DMA	= Dimethyl Arsenic Acid
EMG	= Electromyography
IgE	= Immunoglobulin E
OR	= Odd's Ratio

PR = Prevalence Ratio
SMR = Standardized Mortality Ratio
C Scale = Centesimal Scale
X Scale = Desimal Scale
M Scale = Millisimal Scale
vit. A = Vitamin A
vit. C = Vitamin C
vit. E = Vitamin E

Signs and Symbols-

= Number
% = Percentage
< = Less than
> = Greater than
≥ = Greater than and equal to
≤ = Less than and equal to
( ) = Parenthesis
µgm = micro-gram
nm = nano metre
km. = kilo-metre
cm. = centi-metre
ml. = milli-litre
mg = milligram
mg/l. = milligram/Litre
ft. = feet
Fig. = Figure
Ppm = Part per million
Ppb = Part per billion
et al. = *et alia* (and others)
i.e. = *id est* (that is)
e.g. = *exempli gratia* (for example)

CHAPTER -1

INTRODUCTION

1.1 General Introduction

Arsenicosis is most crippling disease in these days. Patients are suffering from many years in many countries of the world. Arsenic first found in ground water of Bangladesh in 1993 at Chapai Nawabganj in Rajshahi Division (APSU/DPHE, 2005a). But it was present in the groundwater for last 25-30 years. Now arsenic is spread out all over Bangladesh. Human gets oxidized arsenic with drinking groundwater for many years. This low dose long duration arsenic engulfing creates a slow poisoning in humans. Following long-term exposure, the first changes are usually observed in the skin, these are mainly melanosis, leucomelanosis, keratosis and then hyperkeratosis. Cancer is a late phenomenon, and usually takes more than 10 years to develop. Internally arsenic creates lungs cancer, liver scirrhosis, kidney damage, infertility in man and woman (WHO, 2001). All the symptoms are called arsenicosis. And the patients of arsenicosis have got no curable remedy for the disease and peoples got no protection from this poisonous affection till now. There are also other environmental effects on crops, vegetables, animals and fishes by arsenic poisoning. Secondarily humans are eating them and gathering more arsenic in the body. The environmental impacts of all the things are very crucial in these days. The awareness from WHO (2001) is that, arsenic in drinking water will cause 200,000-270,000 deaths from cancer in Bangladesh alone.

Natural water Sources, Types and Uses in Bangladesh

Bangladesh is the deltaic plains of the Ganges and Brahmaputra River system (Fig.-1.1). Besides these two big rivers there is a complex network of 59 international rivers (Adel, 1998) and 137 local rivers with huge fresh water. People usually drink and use in houses this profuse surface water, groundwater and often they collect rain water for drinking purpose in arsenic affected area. Surface water is easily contaminated with silts and various microbes. For this reason, groundwater is the main source of drinking water in Bangladesh because of its low cost simple technologies to setup a tubewell and relatively easy to operate it. In Bangladesh, the withdrawal of groundwater has been increasing over the last three decades. Now a days government and different NGO's offering rain water networks for drinking purpose in arsenic affected areas. The collection, preservation and use of rain water are not so easy like tubewell water. The quantity of rain water depends on geographical locations. The uses of rain water dependent on availability of manpower and cost of construction of the tank. But we know, the tubewell water is the main option for drinking purpose in Bangladesh. An analysis of data collected in the DPHE-Unicef project area in fifteen upazilas of Bangladesh reported that 88.1% tubewells are privately owned of which 84.8% are used for domestic purposes (APSU/DPHE,2005b).

The provision of arsenic free water is essential for Bangladesh to safeguard the health of the rural population. Restoration of rural health from arsenicosis and its consequences needs preventive measures.

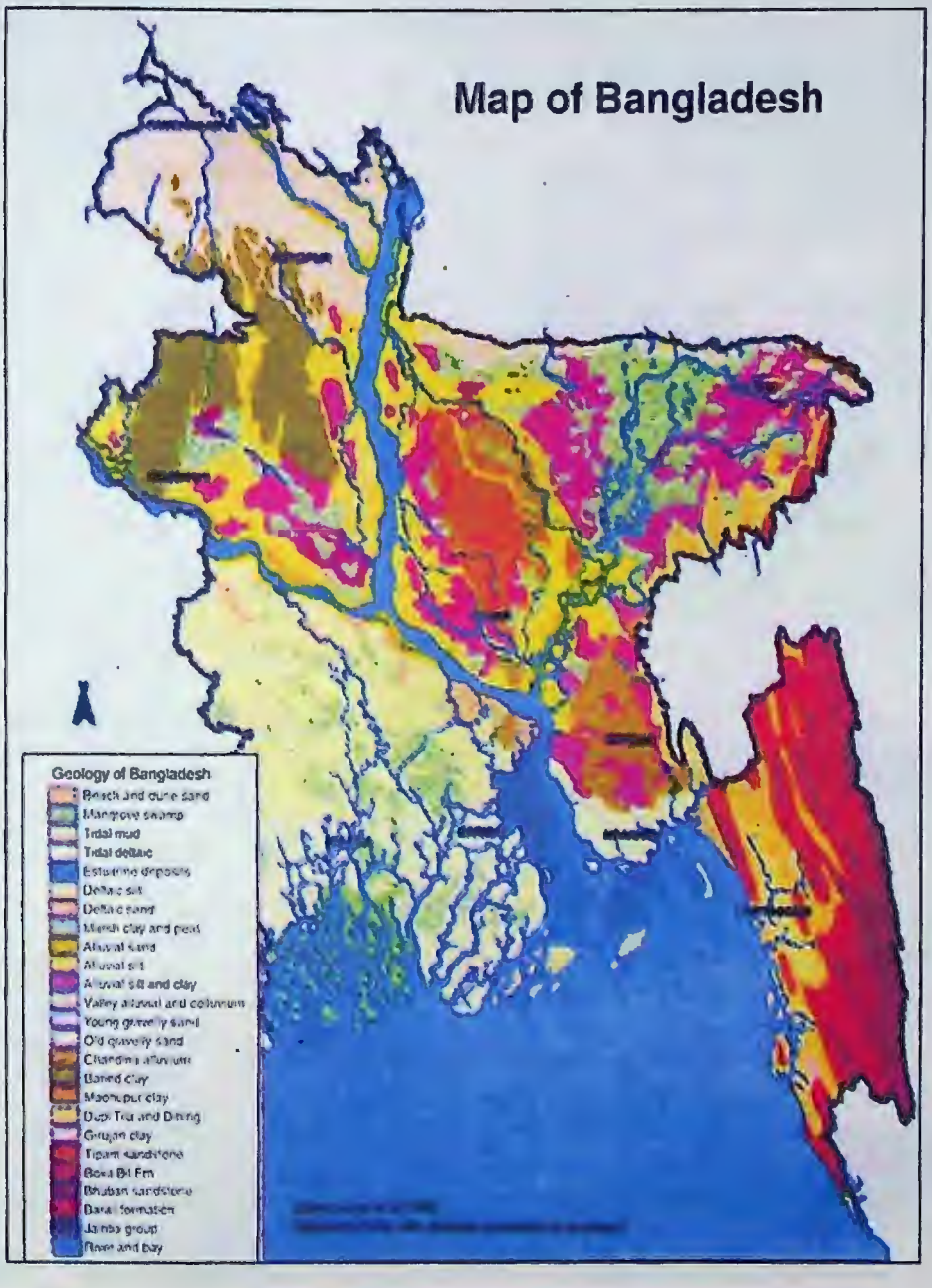


Figure- 1.1: Geomorphology and geology of Bangladesh

Source- EGIS, (1990)

1.2 Environmental problem: Global and national

Chronic arsenic poisoning is a great environmental problem in the world. Globally arsenic is the source of immense misery of human lives. It has affected millions of people globally distributed over 20 countries. In Bangladesh and its adjoining part of West Bengal (India) alone, ~100 million people are at risk (Khuda-Buksh *et al.*, 2005). Southern part of Nepal is also affected by arsenic.

1.2.1 Worldwide Arsenic Problem

All the continents of the world are affected by chronic arsenic contamination. Hundreds of millions of people across the world are suffering from this poisoning for consumption of arsenic contaminated water. Arsenic in groundwater has been detected in many countries of the world. These are mainly USA, UK, Canada, Latin America, Parts of Europe, Vietnam, Thailand, Cambodia, China, Taiwan, Japan, Australia, Argentina, India, Pakistan, Nepal and some other countries of Africa (Rahman, 2003). A research of Zaldivar (1974) described about the arsenic exposure via drinking water in Chile, Mexico and Taiwan in the early 1900s. Such as:

Argentina: Most probably Argentina is the country where arsenic pollution found at first in the world. Researches on arsenic began in Argentina in 1938. The disease caused by arsenic poisoning is known as 'Bell Will Disease' in Argentina. Mostly 20,000 people were affected by chronic arsenic poisoning.

Mexico: Arsenic first detected in the country in 1963. Mostly 127,000 people were suffering by chronic arsenic poisoning. Here many people are suffering from different consequent problems of arsenic poisoning, including cancer.

Chile: Arsenic contamination in water was first detected in 1957 in a province of Chile. Many people died from the consequences of arsenic. Seven percent of total death from 1989 to 1993 was caused by arsenic poisoning.

USA: Arsenic concentration in ground water of USA is low. According to a statistical report 0.045 mg/litre arsenic was found in California's groundwater. About 3 million people of the country are drinking arsenic contaminated water.

China: Arsenic affected patient first found in China in 1953. First medical survey was done in 1964. Second survey was done in 1991-93. According to the survey reports, 1,545 arsenicosis patients were found among 9202 people. Of them, 88% received arsenic from food, 7% from water and 5% from air due to coal burn.

Japan: Arsenic first found in Japan before 50 years. There were 217 arsenicosis patients found in two villages- Turoko and Matsu in 1995. Coal mines were the causes of arsenic pollution.

Taiwan: First water treatment plant was established in 1956. Before that they were confused the disease as 'black foot disease'. But the black foot disease (BFD) caused by chronic arsenic poisoning first found in 1920s,

locally known as Wu Chiao Ping. This peripheral vascular disease leads to progressive gangrenes of the legs (Abernathy, 2001). At present, almost 100,000 people are suffering from arsenic consequent diseases.

Mongolia: First arsenic detected with fluoride in groundwater in 1962. But first arsenicosis patient were detected in 1988. After that, 1,774 arsenicosis patient were identified in 1989. It is thought that the problems arose due to continued irrigation for agriculture by groundwater in arid region.

Thailand: First arsenicosis patient was found in 1987. The situation is worst in Ronpibul district of Srithammarat province. A survey found more than 1,500 patients with proven skin cancer.

The Philippines: Arsenic first detected in human bodies, which were living on both sides of the rivers Matingao and Marbol after a geothermal power plant was set up on Mount Apo in 1992. The geothermal plant is suspected to be the cause of arsenic contamination in water. A total of 39 arsenic affected patients were detected in 1995.

New Zealand: Recently arsenic was detected in the country.

India: West Bengal is the worst affected state in the country. Arsenic first found in the state in 1983. Then 63 patients were identified in two districts. Later on, these two districts rouse to 9 districts in 2001 when 2,700 peoples were found arsenic affected.

A study was conducted to investigate the presence of arsenic in 246 ground water samples from Brahmaputra and Barak valley in Assam. Atomic absorption spectrophotometer analysis revealed the presence of

arsenic ranging from 0.01-0.445 mg/l, which was regarded as toxic to human health (Paul and Kar, 2004).

Nepal: Unicef estimated that arsenic contamination could affect more than 1.4 million people across 20 districts in Terai, that was about 47 percent of Nepal's total population, nearly 90 percent of whom depend on ground water for their daily needs (Lawti, 2006).

1.2.2 Arsenicosis Problem in Bangladesh-

According to a British Geological Survey study in 1998 on shallow tube-wells in 61 of the 64 districts in Bangladesh, 46% of the samples were above 0.01 mg/L and 27% were above 0.05 mg/L. When combined with the estimated 1999 population, it was estimated that the number of people exposed to arsenic concentrations above 0.05 mg/l is 28-35 million and the number of those exposed to more than 0.01 mg/l is 46-57 million (BGS, 2000 and WHO, 2001), (Fig.-1.2); the southern, south-western and north-eastern regions of the country are mostly affected (Ali *et al.*, 2004). Shallow ground water aquifer in Bangladesh is contaminated with arsenic and the source of contamination is geogenic. The arsenic affected ground water of Bangladesh contaminates surface soils and plants thereby arsenic enter the food chain. Contaminated tubewell water and food creates serious health problems to human beings. Almost 38,000 arsenicosis cases were identified in different hospitals (Kabir, 2005). According to a research, 85 million out of 130 million country people live in arsenic risk zone (Rahman, 2003).

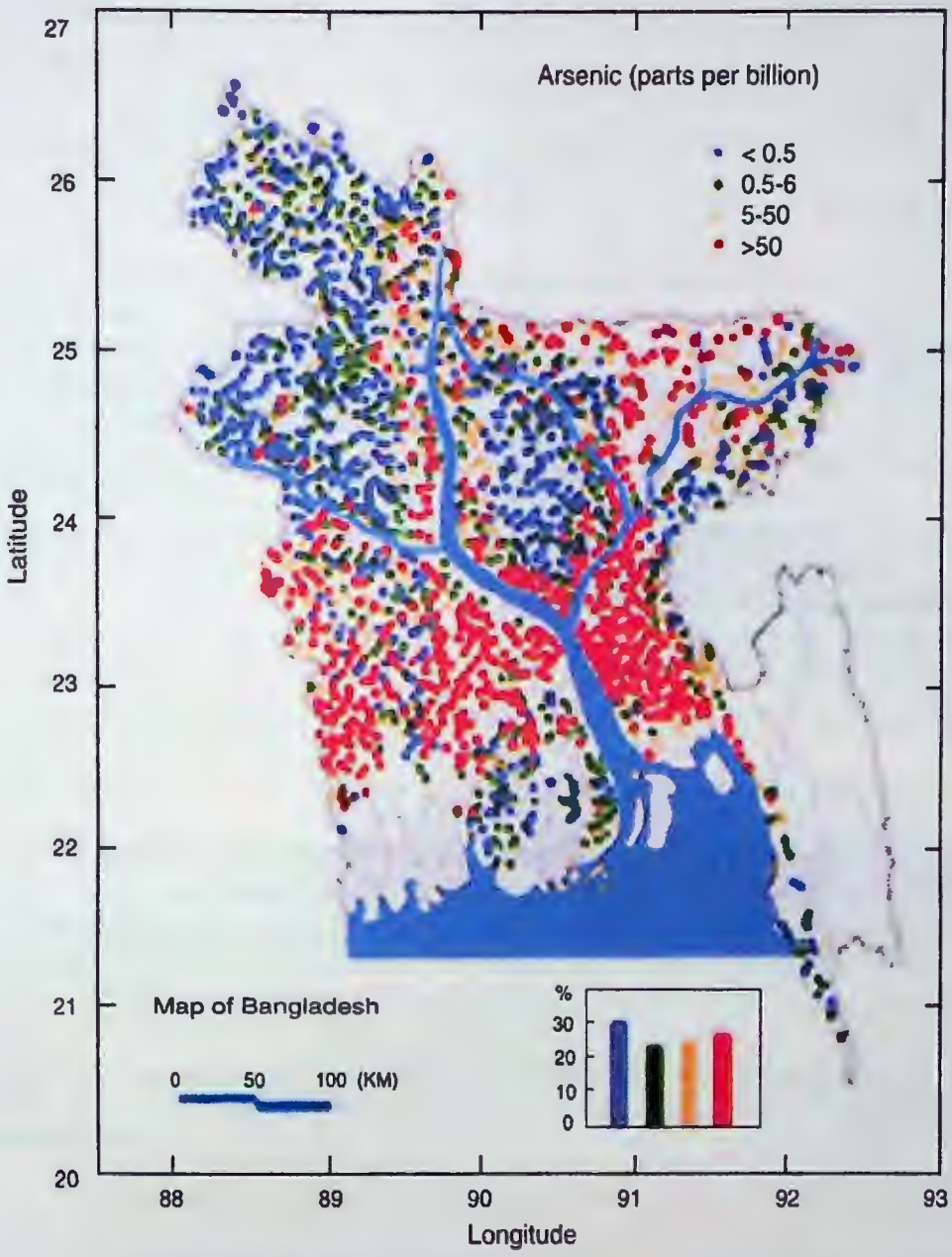
People are gathering arsenic in their body directly by drinking shallow tubewell water, and indirectly by foods, which are cultivated by arsenic containing irrigation water (ground water).

Huge numbers of people are arsenicosis affected and day by day the numbers are increasing. According to the government estimation (BAMWSP, 2006) the current figure of arsenicosis affected people are 38118 out of which 46% is male and 54% is female. Union wise number of patients exceeds 100 per 10,000 population are presented in table- 1.1.

Table-1.1 : Data on affected areas and population by Division (BAMWSP, 2006)

Bagerhat	Kachua, Sarankhola, Rampal, Morelgonj, Mollarhat, Bagerhat, Fakirhat, Chitalmari.	589	Barisal	Gournadi, Agoiljhara, Banaripara, Bakergonj, Mehendiganj, Ujirpur, Muladi, Babugonj, Barisal Sadar.	558
Bhola	Lalmohon, Bhola.	12	Bogra	Dhunat, Gabtoli, Shibgonj.	338
Brahmanbaria	Nasimagar, Kasba, Brahmanbaria, Akhaura, Sarail, Banchharampur, Nabinagar.	1056	Chandpur	Hajigonj, Matlab, Faridgonj, Shahrasti, Kachua, Haim char, Chandpur Sadar.	2603
Chitagong	Sitakunda, Mirsharai.	134	Chuadanga	Jibonnagar, Alamdanga, Chuadanga, Damurhuda.	804
Comilla	Meghna, Brahmanpara, Nangolkot, Burichang, Laksham, Debiddar, Daudkandi, Chandina, Chouddogram, Barura, Homna, Muradnagar.	4036	Cox's Bazar	Ukhia	15
Dhaka	Dohar, Keranigonj, Dhamrai.	136	Dinajpur	Birgonj	6
Faridpur	Boalmari, Modhukhali, Sadarpur, Nagarkanda, Alphadanga, Bhanga, Char Bhadrasan.	1452	Feni	Feni sadar, Sonagazi, Dagan Bhuiyan	451
Gaibandha	Polashbari, Sadullapur, Sundargonj, Gobindogonj	238	Gazipur	Kaligonj	6
Gopalgonj	Kashiani, Kotwalipara, Gopalgonj Sadar, Tungipara.	571	Hobigonj	Ajmirigonj, Baniachang,	26
Jhalokathi	Jhalokathi Sadar, Nalchity.	123	Jamalpur	Sharishabari, Bakshigonj, Jamalpur Sadar.	232
Jessore	Abhoynagar, Keshabpur, Bagherpara, Chougacha, Manirampur, Jessore Sadar, Jhikorgacha.	970	Jhenaidah	Harinakunda, Shailkupa, Moheshpur, Kotchandpur, Jhenaidah Sadar, Kaligonj	343
Khulna	Paikgachha, Phultola, Terokhada,	463	Kishoregonj	Karimgonj, Hossainpur, Pakundia,	543

	Batiaghata, Dumuria, Dhigalia, Rupsha, Dacope.			Kuliarchar, Bajitpur, Kishoregonj, Bhairub.	
Kurigram	Raumari, Nageswari, Ulipur, Rajarhat, Kustia, Daulatpur, Bheramara, Mirpur, Kumarkhali.	1494	Lakhmipur	Ramgonj, Raipur, Ramgati, Lakhmipur Sadar.	1631
Madaripur	Madaripur, Kalkini, Shibchar, Rajoir.	743	Magura	Mohammadpur, Salikha, Magura.	565
Manikgonj	Shibaloy, Harirampur, Daulatpur, Singair, Ghior, Saturia.	339	Moulovibazar	Kamalgonj, Rajnagar, Kulaura, Moulovibazar.	78
Meherpur	Gangni, Meherpur, Mojibnagar.	849	Munshigonj	Lohajong, Tongibari, Sreenagar, Gazaria, Munshigonj Sadar, Serajdikhan.	974
Mymensingh	Gauripur, Nandail, Mymensingh Sadar, Haluaghat, Phulpur, Dhobaura.	269	Netrokona	Durgapur, Kalmakanda.	612
Naogaon	Porsha, Manda.	40	Norail	Lohagora, Narail, Kalia.	249
Narayangonj	Araihazar, Bandar, Sonargaon.	636	Norshingdhi	Raipur, Norshigdhi, Belabo, Monohardi, Shibpur, Palash.	1210
Natore	Lalpur, Bagalipara.	378	Nawabgonj	Nawabgonj Sadar	499
Netrokona	Madan, Alpara, Kendua, Mohongonj, Khaliajuri.	419	Noakhali	Chatkhil, Companigonj, Shenbagh, Noakhali Sadar Begumgonj.	2743
Pabna	Sujanagar, Sathia, Ishordi, Bera.	577	Pirojpur	Motbaria, Nazirpur, Nesarabad, Bhandaria, Pirojpur Sadar	62
Rajbari	Pangsha, Rajbari, Goalandaghat.	826	Rajshahi	Charghat, Puthia, Bagha, Paba, Mohanpur, Godagari, Tanore, Baghmara, Boalia, Durgapur.	640
Rongpur	Pirghachha	296	Satkhira	Shyamnagar, Kaligonj, Kolaroa, Debhata, Ashasuni, Tala.	1252
Shariatpur	Zanjira, Bhedargonj, Dhamuidya, Goshairhat, Naria, Shariatpur Sadar.	804	Sherpur	Nalitabari, Sreebardi, Nakla, Sherpur Sadar.	585
Sirajgonj	Belkuchi, Kamarkhanda, Kazipur, Sirajgonj, Raigonj, Shahjadpur.	538	Sunamgonj	Jamalgonj, Sunamgonj, Sulla, Dowarabazar, Dharmapasha, Derai, Chhatak, Bishambarpur.	627
Sylhet	Balamgonj, Golapgonj, Gowinghat, Zakigonj, Bishonath, Kanaighat, Companigonj,Beanibazar.	368	Tangail	Nagarpur, Delduar, Tangail Sadar, Mirzapur, Basail.	94
Chapai Nawabganj	Chapai Nawabganj, Bholahat, Nachole, Shibganj, Gomastapur.	1822		**TOTAL**	**38120**



Source : Groundwater Studies of Arsenic Contamination in Bangladesh DPHE/BGS/DFID (2000)

Figure-1.2 : Arsenic contaminated area in Bangladesh

1.3 Pertinent Issues and Rationale for the Research

Alternate Safe Water Initiatives and Water Crisis

Screening has had continuing in allover the country gradually by the government and different NGO's after identifying arsenic in ground water of Chapai Nawabganj in 1993. In the mean time British Geological Survey came at Bangladesh to perform a primary survey on arsenic contaminated ground water (BGS, 2000). Deep tubewells installation is the principal water supply in the arsenic affected areas. Rain water harvesting unit have been the next most commonly installed units. The arsenic mitigation programs have installed over 21,000 alternative water supplies, with DPHE-UNICEF being the largest program. In arsenic mitigation programs, rain water harvesting has been the most commonly installed options, with dug wells the second most common. The use of deep tubewells has been more limited in these programs largely because of restrictions enforced on the use of deep tubewells. Appendix figure-1.1 shows the breakdown of mitigation options by technology type (APSU/DPHE, 2005c).

The government of Bangladesh (GOB) has initiated and implemented a number of major programs, with support from development partners and a range of national and international NGO's. The programs (APSU/DPHE, 2005d) are—

i) The implementation of a national tubewell screening by Bangladesh Arsenic Mitigation Water Supply Project (BAMWSP) and other stakeholders.

ii) Awareness raising programs in the affected upazilas.

iii) Implementation of pilot level mitigation programs.

iv) Finalization of a protocol for patient identification and management,

v) A number of research projects.

1.4 Health Effects of Arsenic Poisoning

Toxicity depends on various options of arsenic itself and different bodily functions and nature. Chemical and physical factor of the compound is one of the factors. And the quantity of arsenic poison, duration of taking it, immunity of the person and health are also the other factors of the intensity of toxicity. All the compounds of arsenic are toxic for the human bodily mechanisms and functions. It is generally recognized that the soluble inorganic arsenicals are more toxic than the organic ones, and the trivalent forms (As III) are more toxic than the pentavalent ones (As V). The toxic effects may be of **two types.** These are –

a) Acute effects (short-term exposures),

b) Chronic effects (long-term exposures).

1.4.1 Acute Effects (short-term Exposures)

Ingestion of large doses of arsenic usually results in symptoms within 30 to 60 minutes, but may be delayed when taken with foods. Acute arsenic poisoning usually starts with a metallic or garlic like taste, burning lips and dysphagia. In a research, Brayer *et al.,* (1997) reported that violent vomiting may ensure and may eventually lead to hematemesis. These gastrointestinal symptoms are the result of intestinal injury caused by dilatation of splanchnic vessels leading to mucosal vesiculation. These vesicles rupture causing bleeding, diarrhoea and protein wasting. Gastrointestinal symptoms often result in dehydration and electrolyte imbalance and may lead to the

development of hypotention and hypoxia. After the initial gastrointestinal problems, multiorgan failures may occur, followed by death. Survivors of acute arsenic poisoning have been shown to develop hepatomegaly, melanosis, bone marrow suppression, hemolysis and polyneuropathy resulting from damage to the peripheral nervous system (Abernathy, 2001). It is found that, if anybody eats 70 –180 mg of trivalent arsenic oxide, then he will die within few minute to few hours (Haq, 2002).

Study reports of acute effects

Cullen, *et al.,* (1995) studied on children, non fatal but nevertheless serious acute effect had been observed after exposure to as little as 0.7 mg of As_2O_3. But significant change found in repeated exposure of high levels of arsenic in a short time. Armstrong *et al.* (1984) reported two of nine died, four developed encephalopathy and eight showed gastrointestinal symptoms after oral exposure of 108 mg/l of water for one week (Abernathy, 2001 and Kusiak *et al.,* 1991).

1.4.2 Chronic Effects (Long-term Exposures)

If anybody gets arsenic in small quantity for a long time, then he suffers with the long-term exposures of arsenic poisoning, which is named as Arsenicosis. Here the symptoms are *melanosis* and *lucomelanosis* of the skin, *keratosis* of the palm and feet, *cracks* of the foot, *ulceration* and *necrosis* on the palm and foot, and sometimes *cancers* on the said parts. Then death is the final destination.

There are multiple end-points, with several different organ systems being affected. These are mainly the skin, respiratory, cardiovascular, immune, uro-genital, reproductive, gastro-intestine and nervous systems. The diseases of these systems are described below-

1.4.2.1 Vascular Diseases

Both the large and small blood vessels are affected by long-term arsenic exposure and produce various vascular diseases. Black foot disease (BFD) and other peripheral vascular diseases are noticed after small vessels affection. But the large vessels affect focused in cardio-vascular and cerebro-vascular diseases. Some work has been done on the possible link between arsenic exposure and hypertension (a known vascular disease risk factor). The findings of key studies in this area of research are summarized by WHO (2001) as follows in table- 1.2.

Table 1.2 Effects of arsenic on vascular system (source: WHO, 2001)

Authors, Study design	Study Population	Source and level of arsenic exposure	Health effects, metric of exposure and measure of association				Comments
Chen et al. (1988) case-referent	241 BFD patients and 759 age-sex-residence matched controls	Well water arsenic concentration = 1140 μ g/L, with progressive decrease since 1956	Exp. Time yrs <1 1-29 >30		Peripheral vascular disease OR 1.0 3.0 3.4 p<0.001 for trend		OR adjusted for nutritional factors, history of BFD, education and evidence of skin lesions
Chen et al. (1988b) cohort	789 BFD patients	Well water arsenic concentration = 1140 μ g/L, with progressive decrease since 1956	End point periph. vasc. Disease Cardio-vasc. Disease Cerebrovasc. Accid. ***p<0.001;**p<0.01		$SMR_{national}$ 1243*** 209*** 118 NS	SMR_{local} 351*** 160** 107 NS	No adjustment for potential confounders
Wu et al. (1989) ecological	Mortality and population daya for 1973-1986 in 42 villages in Taiwan	Well water arsenic concentration = 1140 μ g/L, with progressive decrease since 1956	Age adjusted mortality rates per 1000,000 As exposure All vascular diseases Males Females Peripheral vascular diseases Males Females Cardiovascular diseases Males Females	<0.30 364 278 23 18 126 1	0.30-0.59 421 371 58 48 154 153	≥0.60ppm 573 386 60 35 260 145	No increase in cerebrovascular accidents in either males or females at any exposure dose. Used published Taiwan data from 1964 to 1966; The Natelson method was used (Tseng et al, 1968;Kuo, 1964)
Engel & Smith (1994) Ecological	Mortality study from 30 US countries 1968-84	Arsenic in drinking water	Diseases of arteries, arterioles and capillaries Expos. category μ g/L		SMRs (CI) Males	Females	No effects were observed for all circulary diseases,

			5-10 10-20 >20	110(110-120) 110(100-110) 160(150-180)	110(110-120) ischaemci heart 110(100-120) disease or cerebral 190(170-210) vascular disease. Expected numbers of deaths generated using US mortality rates. Arsenic Concentrations were From public water Supply records.
Rahman et al. (1999a) cross-sectional	1595 people from 4 villages in Bangladesh. 1481 exposed to arsenic and 114 Non exposed Controls.	Arsenic in drinking water. For 39,36,18 And 7%, the exposure was <0.5, 0.5-1, and >1 mg/L, and unknown, resp.	Expo category mg/L yrs 0 <5 5-10 >10	PR*for hypertension (CI) 0.8(0.3-1.7) 1.5(0.7-2.9) 2.2(1.1-4.4) 3.0(1.5-5.8)	Used existing arsenic water measurements (measured by flow-injection hydride generation AAS). Hypertension defined as>140 mmHg systolic BP together with > 90 mmHg diastolic BP. Study limited to the 1595 individuals out of 1794 eligible, who were at home at the time of the interview. 114 persons were considered unexposed and were used as the reference group. *PR = Mantel-Haenszel prevalence ratio adjust for age, sex and BMI.
Jensen & Hansen (1998) cross-sectional	32 arsenic exposed workers and 26 non-exposed referents	Avarage urinary As 35.9 for the exposed and 14.5 μ mol/mol creatinine for the referents	Avarage systolic blood pressure 128 among the exposed, and 120 among the referents, p = 0.023		The exposed group included taxidermists, garden fence makers, week-end cottage constructors, wood impregnators, electric pole impregnators, new house constructors.

Note- OR- Odd's Ratio; PR- Prevalence Ratio; SMR- Standard Mortality Ratio.

Cardio- and cerebrovascular diseases

Studies linking exposure to arsenic and mortality from cardio-vascular diseases are also in table -1.2. The standard mortality ratios (SMR's) for these diseases were elevated for areas with an arsenic concentration of greater than 20μg/l, but were close to 1.0 in the two lower concentration categories (5-10μg/l and 10-20μg/l) for both sexes. A study of Chen CJ, *et al.* (1988) says, multiple logistic regression analysis showed that artesian

well water consumption, arsenic poisoning, familial history of BFD and undernourishment were significantly associated with the development of BFD. The life-table method used to analyze cancer mortality of 789 BFD patients followed for 15 years showed a significantly higher mortality from cardiovascular diseases, peripheral vascular diseases and carcinoma of bladder, skin, lung and liver among BFD patients as compared with general population in Taiwan. A study of Engel and Smith (1994) says, SMR's for aneurisms and arteriosclerosis were also elevated for arsenic concentrations greater than 20μg/l. The SMR's for congenital anomalies of the heart and for congenital anomalies of the circulatory system were elevated in females at arsenic concentrations of greater than 20μg/l only. According to a study of Lewis and Calderon (1998) we found, the results indicate a significant excess of deaths for cardiovascular diseases (including hypertensive heart disease) among males (SMR-2.20) and among females (SMR-1.73) and all other heart diseases among females (SMR-1.43). When SMR's were analyzed according to low (<1000 ppb or μg/l), medium (1000-4999 ppb or μg/l) and high arsenic exposure values (5000 and >5000 ppb or μg/l), the increases of hypertensive heart diseases were not sequential (i.e.- there was no dose-response relationship) from low to high exposed groups (SMRs-2.37, 1.91, 2.29 respectively for low, medium and high exposure groups).

Hypertension

Hypertension with/without heart disease found in some arsenicosis patients. The increase of arsenic in the body oxygen deficiency occur, this can creates the stenosis of the arteries, which can produce hypertension of the patients.

In a research in Bangladesh, Rahman, *et al.* (1998) studied on a total of 1,481 subjects exposed to arsenic contaminated drinking water and 114 unexposed subjects were analyzed for their time-weighted mean arsenic level. There was a significant dose-response relationship between arsenic exposure and increase of blood pressure ($P<0.01$). The study of Jensen and Hansen (1998) analyzed in a group of 40 Danish workers exposed to arsenic in different trades (average urinary arsenic level 22.3 μmol/l creatinine; twice that of the referent), blood pressure was slightly elevated among the exposed group, reaching statistical significance for the systolic but not the diastolic value (Abernathy, 2001).

1.4.2.1 Cancer

Cancer can be developed after a long-term exposure of arsenic in the human body. Cancer is the end-effect of any arsenic related diseases; such as- vascular, respiratory, uro-genital, liver and skin diseases. WHO (2001), recommended cancer causes, due to arsenic are as follows:

- The symptoms and signs that arsenic causes, appear to differ between individuals, population groups and geographic areas. Thus, there is no universal definition of the disease caused by arsenic. This complicates the assessment of the burden on health of arsenic. Similarly, there is no method to identify those cases of internal cancer that were caused by arsenic from cancers induced by other factors.

- Long-term exposure to arsenic via drinking-water causes cancer of the skin, lungs, urinary bladder, and kidney, as well as other skin changes such as pigmentation changes and thickening (hyperkeratosis).

- Increased risks of lung and bladder cancer and of arsenic-associated skin lesions have been observed at drinking-water arsenic concentrations of less than 0.05 mg/L.

- Absorption of arsenic through the skin is minimal and thus hand-washing, bathing, laundry, etc. with water containing arsenic do not pose human health risk.

- Following long-term exposure, the first changes are usually observed in the skin: pigmentation changes, and then hyperkeratosis. Cancer is a late phenomenon, and usually takes more than 10 years to develop.

- According to some estimates, arsenic in drinking-water will cause 200,000 – 270,000 deaths from cancer in Bangladesh alone.

The findings of selected epidemiological studies based on the Taiwanese populations are briefly outlined below; additional details are provided in table-1.3.

Table 1.3 : Studies of cancer following exposure to arsenic (Source: WHO, 2001)

Authors, Study design	Study Population	Source and level of arsenic exposure	Health effects, metric of exposure and measure of association					Comments
Chen et al (1985) Ecological	BFD- endemic area of Taiwan; mortality 1968-1982	Drinking water up to 1.14 mg/L, Decreasing with take into use of reservoir water starting in 1956		SMR (CI), Males		Females		Small intestine, oesophagus, rectum, somach, naso-pharynx, leukaemia, thyroid were not significantly elevated in males or females. Population of Taiwan as the reference
			Bladder:	1100 (933-1267)		2009 (1702-2316)		
			Kidney:	772 (537-1007)		1119 (938-1400)		
			Liver:	170 (151-189)		229 (192-266)		
			Colon:	160 (117-203)		168 (126-210)		
			Lung:	320 (286-354)		413 (360-466)		
Chen et al (1986) Case-referent	69 bladder, 76 lung, 65 liver cancer decedents in Taiwan in 1980-1982. 65 live controls matched by age and sex.	<40 years of use of Artesian water in black foot disease endemic area up to 1.14 mg/L	OR for years of use of arsenic contaminated water:					Deceased Cancer cases. OR's adjusted for age, sex, cigarette smoking, tea drinking, vegetarian habit, vegetable consumption frequency and fermented bean consumption frequency, when the factor was significant at p<0.1. Referents from the same area
			Site	None	1-20	21-40	>40	
			Bladder	1.0	1.3	1.7	4.1 ($p<0.01$)	
			Lung	1.0	1.1	1.5	3.0 ($p<0.01$)	
			Liver	1.0	0.9	1.1	2.0 ($p<0.1$)	
Chen et al (1988b) Cohort	Cohort of 789 Blackfoot disease patients (15 years and 7278 person years of follow-up)	Drinking water concentrations 350-1140 µg/L	SMR	National ref rate		Local ref rate		10.6% lost to follow-up
			Bladder	3880	(p<0.001)	255	(p<0.01)	
			Kidney	1953	(NS)	160	(NS)	
			Prostate	1729	(NS)	268	(NS)	
			Lung	1049	(p<0.001)	284	(p<0.01)	
			Liver	466	(p<0.001)	248	(p<0.01)	
			Colon	381	(p<0.05)	230	(NS)	
			Esophagus	305	(NS)	222	(NS)	
			Stomach	194	(NS)	202	(NS)	
Chen & Wang (1990) Ecological	Mortality from malignant neoplasms in 1972-83 in 314 percincts and townships in Taiwan	74% or precincts had <5% wells with ≥50µg/L As, 15% has 5-14% and 12% had ≥15% such wells. Village mean used in analysis.	Statistically significant association between arsenic level in well water and mortality from the cancer of the lung, liver, kidney, bladder, skin, prostate and nasopharynx after adjustment for indices of urbanization and industrialization					Nearly all cancer deaths among the arsenic exposed included in the Chen et al (1985) study No numerical risk estimates given

According to Cuzick *et al.*, (1992), a significant excess of bladder cancer mortality occurred (observed/expected ratio =5/1.6; p=0.05) among 478 patients treated with Fowler's solution (1% potassium arsenite) in Lancashire, England, during the period of 1945-1969. No excess was found for other causes of death. In a sub-cohort of 142 patients examined for signs of arsenicism around 1970, all 11 subsequent cancer deaths occurred in those with signs of arsenicism (p = 0.0009).

Smith *et al.*, (1998) investigated cancer mortality in a population of around 400,000 people in a region of Northern Chile exposed to high arsenic levels in drinking water in past years. Arsenic concentrations from 1950 to the present were obtained 570 µg/litre by 1979 and 100 µg/litre by 1980. The findings provide additional evidence that ingestion of inorganic arsenic in drinking water is indeed a cause of bladder and lung cancer. It was estimated that arsenic might account for 7% of all deaths among those aged 30 years and over.

As compared with the general population in Taiwan, both the standardized mortality ratio (SMR) and cumulative mortality rate were significantly high in black foot disease endemic area for cancers of bladder, kidney, skin, lung, liver and colon were 1100, 772, 534, 320, 170 and 160 respectively for males, and 2009, 1119, 652, 413, 229 and 168 respectively for females. SMRs of cancers were greater in villages where only artesian wells were used as the drinking water source than in villages using both artesian and shallow wells, and even greater than in villages using shallow wells only (Chen *et al.*, 1985, 1986).

Roth (1958) reported that autopsies of 49 winegrowers, who had showed signs of arsenic intoxication, among them 40 found skin cancer, 19 lung cancer and 13 both of them. Cancer risks amongst copper smelter workers exposed to arsenic in the air were initiated in the 1960's; these studies were primarily concerned with the development of respiratory cancers, in particular lung cancers. Over the past 20-30 years, research effort has also focused on the likely relationship between various types of cancers and exposure to arsenic through the consumption of drinking water. Much of this type of work has centered on populations in the BFD-endemic parts of Taiwan, but there are reports of elevated cancer risks at multiple sites (notably lung, skin, bladder, kidney and liver) from other parts of the world including Japan, Chile and Argentina where subjects of the population are exposed to arsenic contaminated drinking water (Abernathy, 2001).

1.4.2.2 Genotoxicity and Related End-points

Arsenic toxicity can create hereditary changes in cells. Some analyses showed the evidence of arsenic or arsenic effects on various tissues, including blood, buccal and bladder cells as well as sections from tumour or Bowen's disease. Studies of this type usually take the form of three distinct groupings according to the focus of investigation:

a) p^{53} (protein 53 or tumour protein 53) mutations in tumour samples,

b) Sister Chromatid Exchange (SCE), chromosome aberration (CA), and RI in cultured lymphocytes, and

c) MN in exfoliated bladder and buccal cells (possible target tissues from the direct exposure to arsenic from drinking water).

A brief description and a summary of the key findings of all three types of studies are as follows (table-1.4).

Table – 1.4: Genotoxicity of arsenic in exposed humans (Source: WHO, 2001)

Author, study design	Study Population, end-points measured	Source and level of arsenic exposure	Health effects, metric of exposure andmeasure of association				Comments
Warner et al (1994) cross sectional	18 exposed and 18 referents in Nevada MN in bladder and buccal cells	High exposure: well water As concentration>500 (average 1312)µg/L. Referents, average 16µg/L	Bladder cells MN/100 cells (SE) Exposed 5.00 (1.50) 1.82(0.53) Both acentric and whole chromosomes increased. No effect on MN in buccal cells.		Males Referents 2.14 (0.46) Females 1.28 (0.31)	Freq. ratio (CI) 2.34 (1.27, 4.29) 1.43 (0.76, 2.65)	Referents age- and smoking matched
Ostrosky-Wegman et al (1991) cross-sectional	13 exposed and 15 less exposed habitants in North Mexico; CA, SCE, HPRT mutations in lymphocytes	Average drinking water As concentration for The exposed 390µg/L, 19-60µg/L for the referents		CA% (SD) 2.55 (1.73) 3.00 (2.82)	SCE (SD) 9.10 (2.7) 8.80 (1.6)	Vf (SD)* 2.42 (2.26) 5.03 (2.99)	Complex chromosomal aberrations (dicentrics, rings, translocations) increased among the more heavily exposed (0.73% vs. 0.16%)
			High esp. Low esp. All nonsignificant;*: HPRT mutations				
Hsu et al (1997) cross-sectional	15 cases of Bowen disease and 34 referents from the BFD endemic area	Well water arsenic concentration = 1140 µg/L, with progressive decrease since 1956	Patients Referents	SCE/cell (SE); p 8.42 (51) 6.94 (0.37); <0.05		HFC% (SE); p 17.89 (2.83) 8.59 (1.66) <0.05	Referents matched for age, sex and residence

A pilot study on a hydroarsenicism-exposed group was designed by Ostrosky-Wegman, *et al.* (1991). Blood and urine samples were taken from 11 individuals chronically exposed and from 13 individuals with lower exposure to the metal. The high exposed group excreted greater amount of As, nevertheless the *rec* assay (*rec* assay is not a mutation assay, it is very useful, in addition to mutagenic assays, for preliminary screening programs) showed negative results. An interesting finding is that the cell-cycle kinetics exhibited a significant difference between the groups were studyed, the average generation time (AGT) was longer in the highly exposed group. The percentages of chromosomal aberrations and the frequencies of sister chromatid exchanges (SCE) were similar in both populations, although complex aberrations were more frequent in the highly exposed group, which

also showed a higher average variation frequency in the HGPRT exposed assay.

Another research was done by Cherry, (2008), on 600 villages on arsenic contaminated area of Bangladesh. They found an increased risk of stillbirths were associated with arsenic contamination. They were studied on pregnancies and outcomes (n = 30,984) in Gonoshasthaya Kendra for two calendar years, together with existing data on 26 socioeconomic and health factors. In the result, the overall stillbirth rate was 3.4% (1056/30,984) and increased with estimated arsenic concentration (2.96% at <10 μg/l, 3.79% at 10 μg/l to 50 μg/l, 4.43% at ≥ 50 μg/l).

1.4.2.3 Diabetes Mellitus

Abernathy (2001) says, diabetes mellitus has been linked with drinking water arsenic exposure. Lai *et al.* (1994) assessed the relationship between ingested inorganic arsenic and prevalence of diabetes mellitus in 891 adults residing in southern Taiwan. Their study found that residents in the BFD-endemic areas had a two-fold increase in the prevalence of diabetes mellitus when compared to residents in Taipei and the entire Taiwan population. An excess mortality from diabetes among the arsenic exposed population in four townships, relative to local and national rates has been collected by Abernathy (2001).

Diabetes mellitus is also influenced by arsenic exposure as found in a study of Bangladesh. Rahman *et al.,* (1998) used the presence of Keratosis as an indicator of arsenic exposure and showed elevated risks for diabetes in those exposed to arsenic in their drinking water (prevalence ratio = 5.9).

1.4.2.4 Neurological Effects

It is well known that acute arsenic poisoning can cause neurological effects, but there are very few researches on long term arsenic exposure. Hindnarsh *et al.,* (1977), for example, reported a positive association between electromyography (EMG) abnormalities and arsenic levels in drinking water and hair samples in residents of Waverley, Nova Scotia, Canada. Among those using water with more than 1 mg/l arsenic, the frequency of EMG abnormalities was 50%.

Workers at a copper smelting plant exposed to arsenic trioxide were examined for peripheral neuropathy (Feldman *et al.,* 1979). A total of 70 factory workers and 41 non-workers were evaluated. The data suggested an association between arsenic exposure and a higher number of peripheral neuropathological disorders in both sensory and motor nerves and electropathological abnormalities (reduced nerve conduction velocity and amplitude measurements) among the exposed workers.

1.4.2.5 Reproductive Effects

Along all other arsenic effects on different human organs, arsenic has also been linked to adverse effect on reproductive system. A WHO (Cherry *et al.,* 2008) report said that the pregnancy could be hampered with drinking arsenic water. A recent study in Bangladesh said still births were happening by drinking arsenic water. Abernathy (2001) mentioned a few studies, which suggested increased foetal, neonatal and postnatal mortalities and elevations in low birth weights, spontaneous abortions, still births, pre-eclampsia and congenital malformations. Another study on workers and their families

living in the vicinity of the Ronskar copper smelter in Sweden, they have reported an increase in the prevalence of low birth-weight infants, higher rates of spontaneous abortions and elevations in congenital malformations among female employees and in women living close to the smelter relative to women living further away a field. According to another research, the frequency of pregnancy complications, mortality rates at birth and low birth weights were significantly higher in 49 maternal-infant pairs living near a Bulgarian copper smelter, relative to country-wide rates. Placental arsenic levels were also found to be higher for the women in the smelter area than they were living in the non-smelter area.

1.5 Tolerable rate of Arsenic in human body

Arsenic accumulates very slowly in long duration arsenic poisoning. But there is a tolerable range in human body. The World Health Organization (WHO) recommended value for arsenic in drinking water is 0.01 mg/l (WHO, 1993, 2001) and Bangladesh government recognized level is 0.05 mg/l (WHO, 1996). In fact, maximum in taking arsenic is expelled out with urine. Arsenic found in urine within 2-8 hours of in taking. After in taking, 75% arsenic released with urine in a week. Some of them are accumulating in skin, hair and nail, and coming out of the body by perspiration and cutting of nail and hair. More over, some of them are remaining in skin, hair, nail and interstitial spaces of cell membrane (of palm and feet). Normal values of arsenic in different parts of the body are as follows- in blood < 2.5 microgram/litre, in urine < 50 microgram/litre, in hair 0.08-0.25 microgram/gram and in nail 0.43-1.08 microgram/gram (Haq, 2002).

1.6 Manifestations of Arsenicosis

The toxicity of arsenic depends on the chemical and physical forms of the compound, the quantity of it, the duration of in taking and the age and sex of the exposed individuals. Arsenic toxicity may take several years to expose it's manifestation in human beings. The disease is called 'arsenicosis', due to the toxicity of arsenic in human body. The manifestations of arsenic are as follows (Reza, 2006):

1. Skin disorders: hyperpigmentation, hyperkeratosis, and gangrene of the fingers and toes.
2. Lung cancer in gold miners and others after chronic exposure.
3. Nasal septal ulceration and perforation.
4. Peripheral neuropathy (particularly after repeated high dose exposure).

DPHE (Department of Public Health Engineering) says in its article 'Impact on Human Health', the first symptom of arsenicosis is melanosis, where the limbs of the body have brackish/dusky appearance and then rest of the body is affected. Gradually black and white spots appear on the body, a stage known as spotted melanosis. The spots may then become hardened and resulted in keratosis. This is not painful or itchy in the beginning but in the later stage may start rotting and develop into gangrenous ulcers, the pre-cancerous stage. The hardening of palms and soles of the feet is called diffuse keratosis. Wart-like seeds can grow on the keratosis of palms and soles. Tumours may also occur, which is known as spotted keratosis. Due to arsenic toxicity limbs may be affected by gangrenous ulcers, which in some cases result in amputation of the affected limb. General weakness, burning sensation, hot flush and chronic coughs may also affect patients (APSU/DPHE, 2005e).

WHO (2001) say, long-term exposure to arsenic via drinking water causes cancer of the skin, lungs, urinary bladder, and kidney, as well as other skin changes such as pigmentation changes and thickening (hyperkeratosis). Following long-term exposure, the first changes are usually observed in the skin: pigmentation changes, and then hyperkeratosis. Cancer is the late phenomenon, and usually takes more than 10 years to develop. The symptoms and signs that arsenic causes, appears to differ between individuals, population groups and geographical areas.

1.7 Prevalence of Arsenicosis

Globally the interest in the health risks of exposure to inorganic arsenic has been primarily focused on carcinogenic effects and in particular the development of skin and lung cancer, although there is increasing interest in other adverse health effects and a number of epidemiological studies are underway to investigate these (APSU/DPHE, 2005f). Other studies have suggested that there are links between arsenic exposure and cancer of the liver, kidney and prostate (Abernathy, 2001). Peripheral vascular disease and cardiovascular disease have been associated with the arsenic in drinking water. There also appears to be a link between arsenic in drinking water and an increased risk of diabetes mellitus and hypertension (Rahman, 1999).

The prevalence of cough, shortness of breath and chest sounds (crepitations and/or rhonchi) in the lung rose with increasing arsenic concentrations in drinking water. These respiratory effects were most pronounced in individuals with high arsenic water concentrations who also had skin lesion. Prevalence odds ratio (POR) estimates were markedly

increased for participants with arsenic induced skin lesions who also had high levels of arsenic in their current drinking water source (500 µg/l) compaired with individuals who had normal skin and were exposed to low levels of arsenic (< 50 µg/l) (Rahman, 2003).

1.8 Homoeopathic Therapeutics

1.8.1 Homoeopathic Cure

Homoeopathic therapeutic has a great role to cure a disease and to antidote (prevent) a micro-organism and to expel out a foreign particle (e.g.- thorn, glasses, piece of iron etc.) from the body. The rule of cure and the process are as follows-

The general rule is **'Similia Similibus Curanter'** (Hahnemann, 1833), it means ***similar repeals similar.*** There are many scientific evidences or examples of this similarity. Antigen-antibody reaction in the body is the best example of this.

The process is – 'when a causative organism (bacteria/ virus/ fungus/ others) enter the human body, it multiply its number. They secrete toxins, which create diseases in the body. The immaterial form (energy level) of the medicine has the same constitutional nature to the toxin (energy level) of the organism. And according to the Homoeopathic rule both should create the similar disease picture in the body. After exceeding the quantity of immaterial medicine more than the toxin's quantity, then the toxin repeals from the body or on the other hand, the medicinal energy neutralizes the toxin's energy. This is the general phenomena of the Homoeopathic cure.

It is important that, manufacturing of Homoeopathic medicines are quite different from the others. All the medicinal systems are applying molecular form of any crude substance/s or chemical/s as a medicine. But Homoeopathy alone is applying non-molecular form of any crude substance to the patients. Some researchers suspect that 'arsenic is still using in homeopathy' in molecular form, according to the guide line of Thomas Flower's treatment. He introduced "Liquor Arsenicalis"- a 1% solution of potassium arsenic (Rahman, 2006). The 1% solution is 1:100, respectively 1gm medicinal substance and 100gm vehicle. According to Avogadro number this solution containing 6.023×10^{23} numbers of molecules. But in homoeopathic preparation of medicine, clinically used potencies are 30c = 30x100 = 3000; 200c = 200 x 100 = 20,000; M(1000)c = 100,000; $\overline{X}$ = 10M(10,000)c = 1,000,000; $\overline{L}$ = 50M(50,000)c = 5,000,000; $\overline{C}$ = CM(100,000)c = 10,000,000; $\overline{D}$ = DM(500,000)c = 50,000,000 and $\overline{M}$ = MM(1000,000)c = 100,000,000. So the ratio of drug substance and vehicles are respectively- (1:3000); (1:20,000); (1:100,000); (1:1,000,000); (1:5,000,000); (1:10,000,000); (1:50,000,000); (1:100,000,000). (c) is centesimal, means 100 times dilutions in each time.

In a research of Jahangir (1998), the molecular weight of UNO_3 (uranium Nitrate) is-

$UNO_3 = 238 + 14 + (16 \times 3)$

$= 300$

$\therefore$ 300 gm UNO_3 contains 6.023×10^{23} molecules.

Then 1 gm UNO_3 contain 20.08×10^{20} molecules.

The molecules belonging to 20 in number in 11c potency, and in 12c potency the molecules in – 2000 in number (Appendix Table – 1.1). In the circumstances, molecules are completely absent in 12c (12 centesimal scale) potency. But homoeopathic doctor's lowest prescribing potency is 30c. All the increasing potencies from 12c are beyond Avogadro number and they are not containing molecules. Like Uranium Nitrate, Arsenic and other chemicals create no molecular poisoning by Homoeopathic medicines in prescribing dose.

An article in encyclopedia (wikipedia, 2009) says, "In homeopathy, a solution that is more dilute is described as having a higher potency, and more dilute substances are considered by homeopaths to be stronger and deeper acting remedies. The end product is often so diluted that it is indistinguishable from the dilutant (pure water, sugar or alcohol).

X Scale	**C Scale**	**Ratio**	**Note**
1x	-	1:10	described as low potency
2x	1c	1:100	called higher than 1x potency
6x	3c	10^{-6}	
8x	4c	10^{-8}	allowable **concentration of arsenic** in U.S. drinking water
12x	6c	10^{-12}	
24x	12c	10^{-24}	has a 60% probability of containing one molecule of original material if one mole of the original substance was used."

By this part of the article it is easily understood that the prescribing potencies (30, 200 and higher) are not containing any molecule of the original substance, if the original substance is used arsenic itself. So there is no chance of arsenic contamination in human body with *arsenicum album*$_{30}$ or higher potencies.

In another research Benveniste (1988) found the evidence of molecules in 1 x 10^{14} with serial tenfold dilution of goat anti-human IgE. Beyond this number they found no molecules.

So the specialty in Homoeopathy is that, there is not using any harmful molecules, except the potential energy.

1.8.2 Prevention of disease

Medical science says, the disease process in man or community can be intercepted at various levels in the course of its natural history. In this respect, there are 5 levels of prevention or intervention (Reza, 2006), which are as follows-

a) Health promotion	1. Primary prevention
b) Specific protection	
c) Early diagnosis and treatment	2. Secondary prevention
d) Disability limitation	
e) Rehabilitation	3. Tertiary prevention

In this classification, immunization is taken under specific protection in primary prevention and our program, prevention for vigorous effect of arsenic (ulceration, gangrene, cancer *etc.*) by treatment of the arsenicosis

patients are under secondary prevention. Here are some important points for treatment or curative measure. These are –

1. Treatment of the case is prevention for susceptible hosts.
2. In some diseases early treatment is essential to prevent secondary complications.
3. Early treatment is an important part of prevention in respect of certain diseases and conditions, *e.g.*- carcinoma, cardiovascular diseases, wounds, liver disease, lung diseases, kidney derangements *etc.*
4. Treatment relieves pain, arrest or cure disease and this prevent disability and death.

By all of these conditions treatment is also a part of prevention (Reza, 2006). As arsenic is a metal, immunization is not possible in this case, but preventive measure and control of hazards in general environment is best for it.

1.8.3 Homoeopathy in arsenic prevention

As all homoeopathic medicines are tested on healthy human beings and the technique is as follows-

First the medicinal substance (in mater and immaterial forms) in gross quantity has produced in human being. After a certain time, it produces some artificial symptoms. These are *medicinal symptoms*. When these medicinal symptoms are alike (similar symptoms) to the *disease symptoms*, then it is called that the disease is susceptible to the medicine. And practically the particular medicine cures the disease. This is called the Homoeopathic cure. Practically medicines cure the disease, and at the same time, the medicines immunize the body against the disease, due to the law of similarity. This

immunization lasts for several times (days, months, years or whole life). This mechanism depends upon the immunization power (memory T-cells) of the body. This is the prevention against the disease force.

Basically homoeopathy gives a good result in hyperpigmentation, hyperkeratosis and gangrene cases. And arsenic produces a same scenario in arsenicosis disease. So the research proceeds to find a nice remedy in homoeopathy which can cure the arsenicosis manifestations and can prevent the consequences of arsenicosis. On the other hand homoeopathic medicines are not expensive, as well as there is no toxic effects or side effects, due to its immaterial dose administration.

1.8.4 Homoeopathic Researches on Arsenicosis

Researches on arsenicosis are very few. Recently, Department of Zoology, University of Kalyani and Directorate of Research, Bidhan Chandra Krishi Viswavidyalaya, West Bengal jointly has done a research on human blood and urine by producing homoeopathic medicine ***arsenicum album*-30** (Khuda-Bukhsh, *et al.*, 2005). They have trialed on few arsenic affected people and found reduce number of arsenic in their blood and urine.

1.9 Aim and Objectives of the Study -

Arsenicosis disease is a crucial disease in the country now. Peoples of the *most of the districts are arsenic affected.* It is now utmost important to do researches on arsenic poisoning. Arsenic of the ground water *affecting* the *soils, crops, vegetables, fishes, cattle* and *secondarily affecting* the human with them. As ground water is the main source of drinking water for

human beings, so peoples are being contaminated easily with arsenic containing ground water.

So it is the need of the day to save the innocent peoples of the country from this destructive disease, arsenicosis through extensive and intrinsic researches. And the findings shall add to the human knowledge which will help in reviewing the monitoring process and environmental hazard management for future sustainable development and resources protection in the country.

Accordingly the present study was undertaken to fulfill the following objectives:

1. To assess the present status of arsenicosis and its treatment.
2. To determine the clinical sign and symptoms of arsenicosis.
3. To reveal microscopic structure of keratosis.
4. To find out the preventive homoeopathic medicine.

CHAPTER – 2

MATERIALS AND METHODS

2.1 Description of Research Spot:

Site Selection

Two villages, named Haria (Boidyo para) and Ramgonj (a part) of Sonargaon upazilla under Narayangonj district and two other villages, Ahmadpur and Sagarkandi of Sujanagar upazila of Pabna district were selected purposely for the research activities.

Sonargaon upazila is situated in the eastern part of Narayangonj district. It is 24km from the Dhaka city and around 20km from Narayangonj. Sonargaon upazila is bounded by Araihazar and Rupgonj upazila to the north, to the east Meghna upazila, to the south Munshigonj sadar and Gajaria upazila and to the west Bandar and Narayangonj (Fig.-2.1).

About 117.66 Sq.km. area of Sonargaon Upazila consists of 10 (ten) unions and 1 (one) pouroshava sustaining a total population of 305640, (157660 men and 147980 women), (Appendix table-2.1). On an average there are 5.02 numbers per family and population density per sq. km. is 2597 (SUDP, 2004).

Sujanagar upazilla is situated in the south-east corner of Pabna district. It is 120km from Dhaka city by Aricha-nagarbari way and 180km by jamuna bridge way (Fig.-2.2).

About 334.40 sq.km area of Sujanagar upazila consist of 10 unions, 191 mausas and 180 villages. Total population of the upazila is 2,51,192 (130,757 male and 120,435 female). The population density and literacy rates are 751/sq.km and 37.7% (SYB, 2001).

The peoples of this flood plain use both surface water and ground water for their household works, but only ground water as the potable water. According to SUDP (2004) average depth of ground water level is 17.51 ft and there are 798 arsenic free and 1598 arsenic contaminated tubewells.

Figure – 2.1: Map of Narayangonj District indicating Study village (■) in Sonargaon upazila

Figure-2.2: Map of Pabna district indicating Study village in Sujanagar upazila

Though the prevalence of the disease arsenicosis had been reported from last 15 (fifteen) years, but the peoples become conscious about it only for the last 5(five) years. There were 252 arsenicosis patients in Sonargaon upazila and 258 in Sujanagar upazila according to Bangladesh arsenic mitigation water supply project (Table-2.1).

Table- 2.1: Survey of tube wells and houses of the arsenic patients (BAMWSP,2006)

Stakeholder: Unicef

				Tube well Survey				Household Survey				Patients
Sl	District	Upazilla	# of union /# of vill	Total TW	# of Safe TW	# of conta. TW	% of Conta.	# of House hold	Popula-tion (Male)	Popula-tion (Female)	Total	Total
33	Narayangonj	Sonargaon	11/365	24539	9128	15411	62.80	25233			64492	252
Stakeholder: BAMWSP												
147	Pabna	Sujanagar	10/172	19872	16107	3765	18.95	40374	154005	127304	281309	271

Source: National Arsenic Mitigation Information Centre, Email: namic@bdcom.com

2.2 Methods and Analysis

Here the materials are the arsenic affected peoples, who are the arsenic contaminated water users. First of all, there were located the arsenic contaminated tube-wells and the people used that water for drinking and household activities. The arsenic affected and non-affected ones surrounding the tube-wells in the selected areas were identified.

2.2.1 Population Counting

People, who were drinking tube-well water, were recorded as per tube-well, by different age group and occupation wise. Arsenic affected and non-affected people were counted under each tube-well. Age and occupation

are closely related with arsenic poisoning. So these two heads were analyzed carefully and separately. The arsenic affected peoples were designated with their identity numbers for easy maintenance of records.

2.2.2 Observation and Interview Methods

This study was based on the people surrounding the arsenic affected tube-wells. The data were collected on the basis of following two methods:

1. **Observation Method,** and
2. **Direct Personal Interview Method.**

2.2.2.1 Observation Method

The observation had done systematically in a planed way and then checked for its validity and reliability. In course of observation the following aspects relating to the study always had kept in mind:

- Each and every person were observed carefully,
- Recording of data from the observation was performed, and
- Accuracy of the observation and recording were ensured through repetition.

2.2.2.2 Direct Personal Interview Method

Investigating questions had been asked in a face-to-face contact to the persons. Interview had been conducted on the basis of disease picture and the personal data and disease data were recorded in a pre-prepared data sheet. Following things were carefully observed in course of taking an interview:

1. Information had obtained in greater depth and details to make that more accurate.

2. There had a greater flexibility to restructure the question depending on the reaction of the patients.

2.2.3 Sample Collection and Preservation and determination of arsenic load

About fifty liters of ground water was pushed out of the tube well to remove all the settled water in the pipe and to get the ground water in natural condition. Water was collected in unused plastic bottles from five different tube wells (T-1, T-2, T-3, T-4 and T-5) of Haria and Ramgonj village of Sonargaon upazila and one tubewell (T-6) of Ahmadpur village of Sujanagar upazila, which were used as the source of drinking water of arsenicosis patients. The arsenic load (concentration) was determined instantly by using Merck arsenic kit (analytical test strips).

2.2.4 Microscopic Analysis of Keratosis

Two types of analysis were done on human. These are -

A – **Cross-section analysis of Keratosis** (laboratory method) under compound and high resultant computerized electric microscope in the Genetic Engineering and Biotechnology Department of University of Rajshahi.

B – Arsenicosis **patient's symptoms analysis** (clinical method) to find out the sensitivity to the medicine.

2.2.4.1 Collection and preservation of samples

Here samples were the keratosis of the palm and feet skin of arsenic affected persons who were selected for this study (Appendix table-3.3).

Collection and preservation of samples were done according to the medical analysis of pathology (Khaleque, 1982).

1. **Skin samples collection**

The samples were the formation of the keratosis in palm. Skin samples with nodules were collected after washing and moisturizing the surface of the palm and feet. Full matured, pin headed nodules and simple skins were collected for cross-match each other. Before collecting the samples the hands were washed with light hot water and then cut with a sharp blade. Whole skin layer was not cut, but only superficial layer was taken for the examination.

2. **Skin samples preservation**

Freshly collected samples were put into a bottle of **'buens fluid'** for 18 hours instantly after collection. After that these were washed with 30%, 50% and 70% alcohol successively. And these were replaced in 70% alcohol for final preservation.

Skin samples were cross-sectioned and set on glass slide. The slides were preserved after finding good ones under compound microscope. Very good samples were preserved aside after marking on slides.

2.2.4.2 Preparing Permanent Histological Slides:

Block Making and Sectioning

One single and small piece of sample was taken and cut into small pieces for permanent slides. First of all dehydration had done of the samples.

Then embedded in paraffin and made blocks (3x7 cm.) of them and sectioned with microtome machine.

Staining and Mounting

After sectioning the micro-samples were double stained. Eosin staining had done for cell cytoplasm and haematoxylin for nucleus. Gurr (1969)'s staining solution had chosen for the work. The method has mentioned below-

Solutions required

A. Ehrich acid haematoxylin.

B. Van Gieson stain, picro ponceau S, biebrich scarlet or eosin.

Method

1. Sections were taken down to water.
2. Stained for 5(five)-10(ten) minutes in the concentrated haematoxylin solution.
3. Rinsed with water.
4. Transferred to tap water and allowed to remain until sections were blue, about 3(three)-5(five) minutes.
5. Counterstained with eosin for 1(one) minute, Van Gieson for 3-5 minutes; picro ponceau S for 5(five) minutes; biebrich scarlet acetic for 2(two) minutes or congo red 0.05 % for 1(one) minute.
6. Rinsed with tap water.
7. Passed through alcohols to dehydrate.
8. Maintained with Xylene then balsam or Xam.

A micro-sample then placed on a clear slide and dropped D.P.X on it. The name of the tissue was labeled and stored it. Several slides were made for specific patients.

2.2.4.3 Microscopic Findings

After staining and mounting of the samples, microscopic photographs were performed. High resultant computerized electric microscopic had been chosen for best photographs of the sections of keratosis with high resulation, *e.g.*- 100, 400 and 1000 times magnification of the sections.

2.2.5 Clinical Analysis of Arsenicosis Patients

2.2.5.1 Separation of arsenicosis patients

From all population of the research area, arsenic affected peoples were identified, according to the clinical features through examining the patients physically and asking relevant questions.

2.2.5.2 Cancer Search

Cancer is in the main consequences and the third stage of Arsenicosis disease. Searches for cancerous symptoms in the patients were noted.

2.2.6 Medicine Selection Methods

Symptomatically relevant medicines were selected, according to the Homoeopathic law of 'Similarity'.

There are hundreds of skin affectionate medicines in the treatment system of Homoeopathy. Out of which seven (7) selective medicines were chosen for our research, considering symptometologically very closer to arsenicosis.

The particulars of selective medicines are mentioned below (table-2.2):

Table- 2.2: Selected Homoeopathic Medicines for arsenicosis patients.

Name of Medicine	Source	Formula	Medicine code
Arsenicum album	White oxide of Arsenic	As_2O_3	M-1
Antimonium crudatum	Sulphate of antimony	Sb_2S_3	M-2
Alumen	Common potash alum	$K_2SO_4Al_2(SO_4)_3 24H_2O$	M-3
Silicea	Silicon di-oxide	SiO_2	M-4
Calcaria fluorica	Fluoride of lime	CaF	M-5
Arssnicum Sul.Flavum	Arsenic trisulphate	AsS_3	M-6
Cuprum metallicum	Metallic copper	Cu	M-7

2.2.7 Application criterion of the medicines

For finding the preventive medicine, first of all there had a need to find out the sensitivity of medicine in arsenic affected persons. And to make the treatment more perfect, the medicines were applied on the patients group-wise.

Patients were divided into four (4) groups of Sonargaon upazila. Single medicine was applied upon each group of four (4) patients. Four medicines were applied on those four (4) groups. This was the searching for sensitive medicines, and its duration was one month.

This criterion was continued until one or more activating medicines could find out. Then the patients were divided into two groups: keratosis group and melanosis group. Activating medicines were applied on those two groups of patients to find the top most priority of the medicine(s).

Two top priority medicines on the basis of the result of Sonargaon upazila and one new medicine were applied on arsenic affected persons of Ahmadpur village of Sujanagar upazila. Three selective medicines were administered in three selective groups. According to the symptoms priority basis, patients were divided into-

a. Melanosis and leucomelanosis,
b. Hardness of the skin of palm and feet,
c. Keratosis, diffuse Keratosis and spotted Keratosis.

After 6.5 months duration of treatment, there was found super curative medicine. The medicine was highly effective to destroy the affinity of arsenic poisoning, as well as to expel out the arsenic itself from the human body.

Applications of the medicines were started from 21st February 2006 to 5th September 2006 for the patients of Sonargaon upazila and 10th July 2007 to 07th March 2008 for the patients of Sujanagar upazila.

In the beginning, medicines were applied on (**21st February 2006**) on 16 patients of two villages (Haria and Ramgonj) of Sonargaon upazila.

Same medicines were applied on the same patients for the **2nd term.** Only high potencies were chosen for the medicines on **1st April 2006.**

After obtaining the result, the medicines were applied **(1st.term)** on the appreciating patients on **8th June 2006.** But few changes were made in the medicinal group depending on the result obtained.

Result varies in most cases. So a change had done in the application of medicine in 2nd phase of treatment. There were mainly two groups of patients: **1**- Patients who had Keratosis and **2**- Patients who had pigments (Melanosis and Lucomelanosis).

High potency medicines were administered on the patients on **2nd August 2006** on the basis of previous result (2nd term) and these medicines were continued for 2(two) successive months for the better perspection.

In Ahmadpur village, there had been selected 62 patients of arsenicosis disease in the treatment criteria. The medicines were started from 10th July 2007 to 07th March 2008 to obtain preventive medicine.

CHAPTER-3

RESULTS AND OBSERVATIONS

The study was carried out by four continuous processes.

One- identifying arsenicosis patients by clinical (symptomatic) analysis and questionnaire to the patients.

Two- field analysis of tube well water in arsenic contaminated area.

Three- microscopic analysis of keratosis of the palm of arsenicosis patient.

Four- finding preventive medicine for consequences of arsenicosis by applying homoeopathic medicines on the patients after confirming curable nature of the disease, arsenicosis.

The findings of the study are presented below:

3.1 Population Counting:

3.1.1 Population Counting – Family wise

Family wise affected and non-affected people were counted and recorded in both Sonargaon (Appendix Table-3.1) and Sujanagar (Appendix Table-3.2) upazila.

In Sonargaon, all people were drinking tubewell water. All arsenic contaminated shallow tube-wells users were not arsenic affected.

In Sujanagar, all people were drinking filter tank water. One shallow tubewell found in the area and it was below danger level (0.05 mg/l).

3.1.2 Population counting – by different age group

All the People were divided into four parts. Children were counted in one group (growing age- gaining height and weight). Adults (fixed height

and weight gaining age) were divided into two groups for their long duration and all olds were counted in one group.

Arsenic digestion is very much depending on nutrition of the body, so height and weight of different ages were essential for affected and non-affected people.

In Sonargaon, middle age adult persons were mainly arsenic affected, but not all adults were affected. The affected persons percentage of middle age (21-60) were 88.24% (Table-3.1b) (Fig.-3.3). And of all population only 16.04% were affected (Table- 3.1a).

106 peoples of 22 families were living in a para (community). Before 6(six) years they have had 4(four) shallow tube-wells and now they have 5(five) semi-deep tube-wells (350 ft), which were more or less arsenic contaminated.

In Sujanagar, maximum adult people were arsenic affected. The percentage was 83.88% of all adult groups (21-60). Few non-affected adults were in the arsenic affected area (17.24%). In the other area of the village, negligible was affected.

Table 3.1a and 3.1b present the numbers and percentages of the arsenic affected and non-affected people of different age groups in Sonargaon and Sujanagar upazila on the basis of Appendix Table- 3.1 and 3.2.

Table-3.1a: Arsenic affection by age groups of Sonargaon and Sujanagar upazila

Parameters	Upazilas	Age groups				Total
		0-20	21-40	41-60	61+	
Non-affected	Sonargaon	45 (97.83)	29 (69.05)	11 (84.62)	4(80.00)	89 (83.96)
	Sujanagar	48 (90.6)	6 (14.29)	4 (20.00)	0(0)	58 (48.33)
Affected	Sonargaon	1 (2.17)	13 (30.95)	2 (15.38)	1(20.00)	17 (16.04)
	Sujanagar	5 (9.4)	36 (85.71)	16 (80.00)	5(100)	62 (51.67)
Total	Sonargaon	**46 (100)**	**42 (100)**	**13 (100)**	**5 (100)**	**106 (100)**
	Sujanagar	**53 (100)**	**42 (100)**	**20 (100)**	**5 (100)**	**120 (100)**

The figures within parenthesis indicate percentage (%)

Table-3.1b: Arsenic affected peoples and their percentage

Upazilas	Age groups				Total
	0-20	21-40	41-60	61+	
Sonargaon	1 (5.88)	13 (76.48)	2 (11.76)	1 (5.88)	17 (100)
Sujanagar	5 (8.06)	36 (58.07)	16 (25.81)	5 (8.06)	62 (100)

The figures within parenthesis indicate percentage (%)

In Sonargaon, it was found that 5.88%, 76.47%, 11.76% and 0% of people were affected by arsenic among the people of age groups 0-20, 21-40, 41-60 and 61+ respectively (Table-3.1a) and of the total affected persons 5.88%, 76.48%, 11.76% and 5.88% belongs to the age groups 0-20, 21-40, 41-60 and 61+ respectively (Table-3.1b).

In Sujanagar, the result was 8.06%, 58.07%, 25.81% and 8.06% of people were affected by arsenic among the people of age groups 0-20, 21-40, 41-60 and 60+ respectively (Table-3.1a) and 8.06%, 58.07%, 25.81% and 8.06% of the total affected persons belongs to the age groups 0-20, 21-40, 41-60 and 61+ respectively (Table-3.1b).

Figures 3.1a and 3.2 represent the Total Population, Arsenic Affected and Non-affected in Sonargaon and Sujanagar Upazilas respectively.

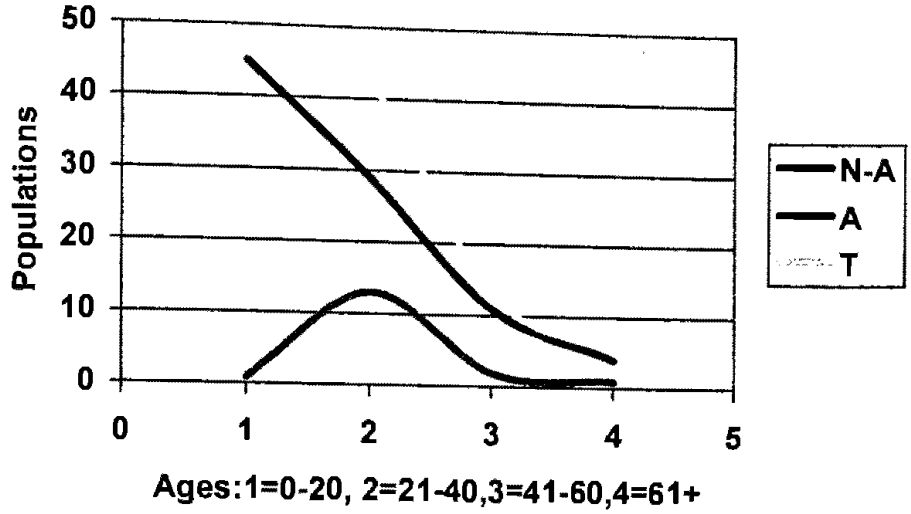


Figure-3.1: Relation between arsenic affected and non-affected people with their different age groups of Sonargaon Upazila
[Age wise Arsenic Affected (A), Non-affected (N-A) and Total Population (T)
(Age: 1= 0-20 years, 2 = 21-40 years, 3 = 41-60 years and 4 = 61+ years)]

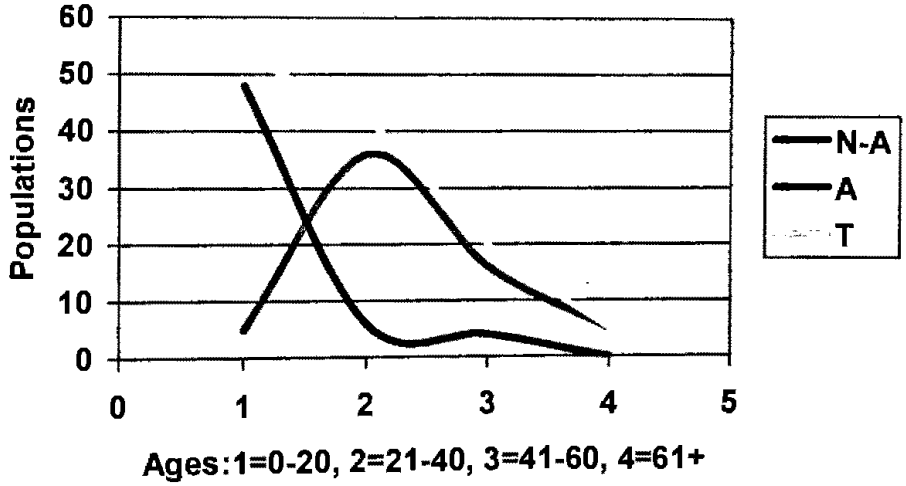


Figure-3.2: Relation between arsenic affected and non-affected people with their different age groups of Sujanagar Upazila
[Age wise Arsenic Affected (A), Non-affected (N-A) and Total Population (T),
(Age: 1= 0-20 years, 2 = 21-40 years, 3 = 41-60 years and 4 = 61+ years)]

Fig.-3.3: Arsenic affected people of middle age groups (Ages 21 to 60 years) of Sonargaon

3.1.3 Occupation Relation with Arsenicosis

Occupation is important for arsenicosis patients. Here mentioned every one's daily works, which he or she was doing regularly.

In Sonargaon, the people of Haria and Ramgonj village were very poor. Their main occupation was fish selling. Besides of this they were use to grow different seasonal crops and fruits in the field. Some were shopkeepers. There were negligible service holders, but few drivers.

Actually they were low earning people. Occupationally peoples of this area are poor. They were living with fish selling, shop keeping and farming (Appendix Table-3.3). Maximum children were not going to the school due to their poverty. So they always remained in tense.

We found that the man or woman of middle age, who is getting the responsibility of the family and who has a tension for his earning, he is suffering in arsenicosis. There were found 37.5% people who were doing business (fish selling) were affected of all the patients (Table-3.2). The housewives (31.25%) were the second highest among all arsenic affected people.

Table-3.2: Occupation of arsenicosis patients of Sonargaon and Sujanagar upazila

Main occupation	No. of patients		%	
	Sonargaon	Sujanagar	Sonargaon	Sujanagar
Business (Fish selling)	6	20	37.5	32.3
Agriculture	1	7	6.25	11.3
Service	2	7	12.5	11.3
House wife	5	27	31.25	43.5
Students	2	1	12.5	1.6
Total	16	62	100	100

In Sujanagar, Ahmadpur village is a very big village. In a certain area of the village people were found arsenic affected. The people of the area were poor and low earning. This area is nearer to the Kashinathpur Bazar. Occupationally maximum people were doing business in the Bazar. So the businesses were maximum (32.3%) among all affected people. They were better in socio-economical position. In between business, shop keepers were more in number (22.6%). Other businesses were little business (6.5%), rickshow puller (1.6%) and fish selling (1.6%). Farmers were the second highest rank (11.3%) in occupation, then workshop worker (6.5%). The black smith, teacher and student were negligible in percentage. All the affected women were housewife (43.5%), except one. She was teacher (1.6%). Only one affected child was found student (1.6%). Others were non-affected and not going to the school (Appendix Table-3.4 and 3.5).

3.2.1 Observation and Interview Results on Arsenic Affected Patients in Sonargaon upazila of Narayangonj District

Through intensive investigations it was revealed that the awareness about the arsenicosis patients was developed in this area at the very begining of the twenty first century. General peoples had no idea before that. Once, an NGO worker came to see his relative there. Suddenly, he found a boy playing on the road. The boy had no shirt on his body. He found the boy had some black and white spots on his chest. He suspected that as **arsenicosis**. He asked the boy and his father to avail the opportunity for laboratory diagnosis free of cost from his office. Father agreed to do that. Within a few days the boy was recognized as an **arsenicosis** patient.

But in fact, peoples had been suffering with this crippling disease for last 15 years. Then they thought it as a skin disease. By the alerting news in television before 10(ten) years, they assumed it might be arsenicosis disease. Once an arsenicosis affected patient went to NIPSOM at Mohakhali, Dhaka to be sure about it. He got the brief idea about the disease and medicines from NIPSOM. He was also adviced to continue the medicines and to send such other patients to visit NIPSOM.

After that NGO's and government's hospital authority became alert about it and all of them set up arsenic program in this area. DPHE and different NGOs tested the tube wells of this upazila. They found, 6 (six) unions out of 10 (ten) unions and 1(one) pouroshava were arsenic affected. After that government hospital, DPHE and NGOs took different measures against arsenic poisoning in the area.

Previous Treatments of Arsenicosis Affected Patients

First of all NGO's extended their helping hands for treating these arsenicosis patients, BRAC was the pioneer of them. Upazila health complex hospital (UHCH) of Sonargaon also made a separate cell for them, under upazila health & family planning officer. They were giving Evagren tablets (containing anti-oxidant, vitamin C and minerals as Zinc, Selenium, Copper and Manganese) and Salicylic acid Ointment for arsenicosis treatment. According to hospital report, the total population of the upazila was 363608, the children population (9 month-10 years) was 92551 and recorded arsenicosis patients were 38 in number. Different campaigns were arranged by the hospital in different time of the year.

According to patients observation Carocet tablets (contains Beta carotine, Vitamin C and di-α-Tocophenol Acetate) was better for arsenicosis patients. It was made by Baximco Pharmaceuticals ltd. This tablet was to same extent effective to reduce the disease but could not make it cure. These medicines were available in the open market. But the patients did not continue the therapy consistently. The medicine carocet had the same nature like Evagren. But it was not much better like the first one.

Very few patients got NIPSOM's treatment. Its medicines were E.vit, Ratinol Forte, and C-vit. They got more or less better result with it, but with the cessation of treatment, the arsenicosis symptoms reappeared and the patients did not continue the treatment offered from NIPSOM.

3.2.2 Observation and Interview Results on Arsenic Affected Patients in Suja Nagar Upazilla of Pabna District

The south-east part of Pabna district was highly affected by arsenic in ground water, mainly Sujanagar, Sathia and Bera upazilas. Ahmadpur village of Sujanagar was widely affected and many peoples were suffering with arsenicosis. Two men and one woman had died with arsenicosis in Ahmadpur. So peoples of the village were very much worried about the arsenicosis disease. Now one patient was severely affected by arsenicosis. He had melanosis on chest, keratosis on palm and feet, and oedema of the leg. His brothers and sister were died by oedema of the leg.

But a family of Sagarkandi village was dangerously affected with ulceration on chest, arms and legs. Two patients were died with arsenicosis. Now one patient was severely affected. But they were not interested to take

medicines except from upazila health complex (UHC) hospital. UHC authority urged them to take only their prescribed medicines.

3.3 Arsenic Concentration Result of Tube-well water

The results of analysis of tubewell water in Sonargaon and Sujanagar upazila are presented in the table 3.3a and 3.3b-

Table-3.3a: Analysis of tube well water of the research area of Haria and Ramgonj

Tube wells	Samples Results (mg/l)	Bangladesh DWS	WHO DWS
T-1	0.30	0.05	0.01
T-2	0.10	0.05	0.01
T-3	0.01	0.05	0.01
T-4	0.05	0.05	0.01
T-5	0.01	0.05	0.01

Note: DWS - Drinking Water Standard

Table-3.3b: Analysis of tube well water of the research area of Ahmadpur

Tube wells	Samples Results (mg/l)	Bangladesh DWS	WHO DWS
T-6	0.05	0.05	0.01

In Sonargaon upazila, two tube-wells were exceeded the drinking water standard (DWS) value (T-1, T-2) and one was alarming (T-1). Three tube wells were within Bangladesh drinking water standard (T-3, T-4, T-5) and two tube wells were within WHO standard (T-3, T-5). (T-1) tubewell was in Ramgonj village, rests were in Haria village (Table-3.3a).

In Sujanagar upazila, maximum people were using well tank filter water (Fig.- 3.4 and 3.5) and rain water. One tubewell (T-6) found in use in a family of Ahmadpur village. The (T-6) tubewell was within the range of Bangladesh standard (Table-3.3b). Local heath complex allowed them to drink the water.

Fig.- 3.4 : A typical dug well with an attached tube well.

Fig.- 3.5 : Water tank from which gravity feeds the houses.

3.4 Microscopic Analysis Results of keratosis

The present study is on the keratosis of the palm of the patients (Fig.-3.2). The keratosises were present on the epidermis of the palm skin. The cut part of the epidermis with keratosises were preserved on the spot and made thin layers of epidermis with keratosis for analysis under microscope.

In microscopic view, there found many germ kratosis in epidermis. Few very little to many giant germ keratosises are found. Germ keratosises are as little as a dot, but their colors are black (Fig.-3.3-3.8). Black color cells are due to oxygen deficiency in cells. It means there are functional changes in keratin layers. As they grows

bigger, there found morphological changes in them. There is a big hole in the centre of keratosis and black keratins are rounded to the hole. Shapes of the black keratin cells are changed. Somewhere there are diffused cells. But all of these made circles like galaxy around the holes (Fig.-3.8-3.11). The shape of the hole is like a ball, somewhere oval, somewhere large oval like candle light. The borderline of the hole is smooth. The surrounding cells (keratosis) are dense and harder than other normal stratified keratin cells. One keratosis found unbroken centre, there is no hole, but there is an egg shell like covering on the hole (Fig.-3.12). This figure is seems to be a galaxy. It means, the centre is controlling the surrounding cells, or/and the surrounding cells are misarranged by the centre of a keratosis.

In very bigger and matured keratosis, it breaks from the outer cells of the arrangement and creates cracks. As keratins are waterproof substance, it has no tonicity. For atonic character once cracks starts it runs to the inner side of the epidermis. Corneus layers breaks easily due to its nature. When it lasts for many days cracks happens in granular and mucous layer also. After mucous layers it runs to dermis part. Then bleeds by breaking capillaries and easily infection take place. Finally gangrene and cancer have formed. According to WHO (2001) report, 'following long-term exposure, the first changes are usually observed in the skin: pigmentation changes, and then hyperkeratosis. Cancer is a late phenomenon, and usually takes more than 10 (ten) years to develop'. In our examined keratosis figures many stages were found towards cancer.

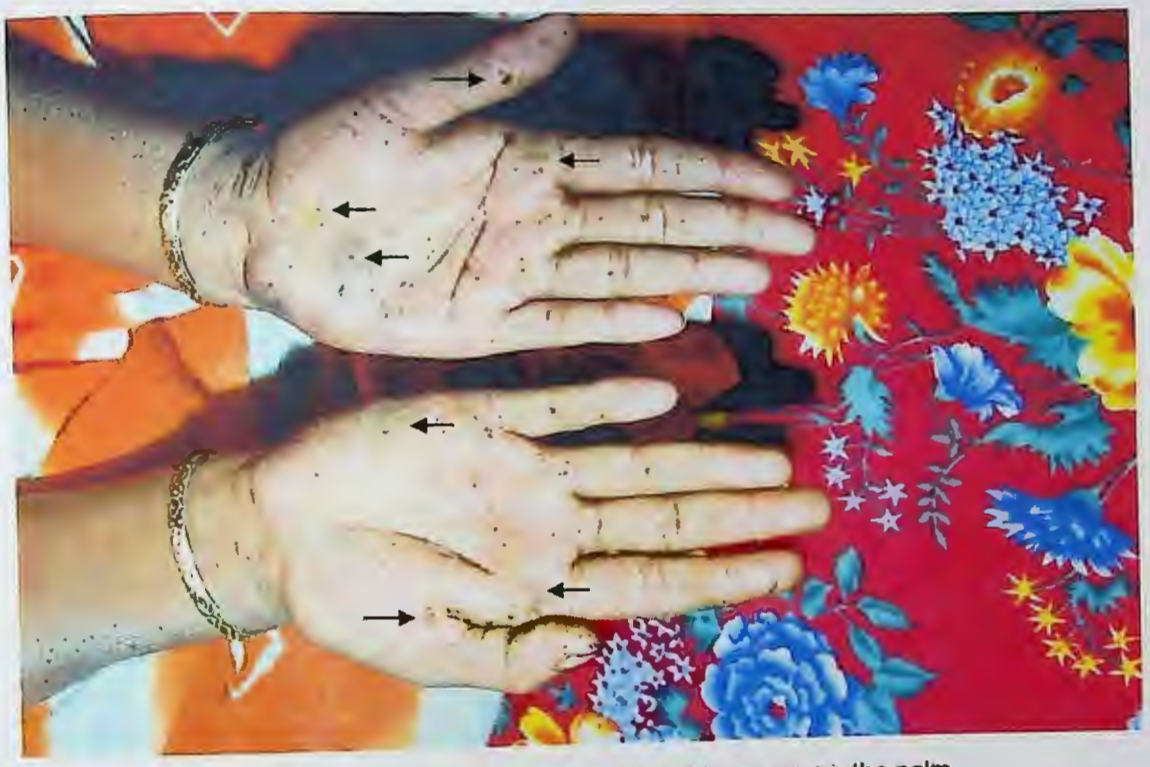
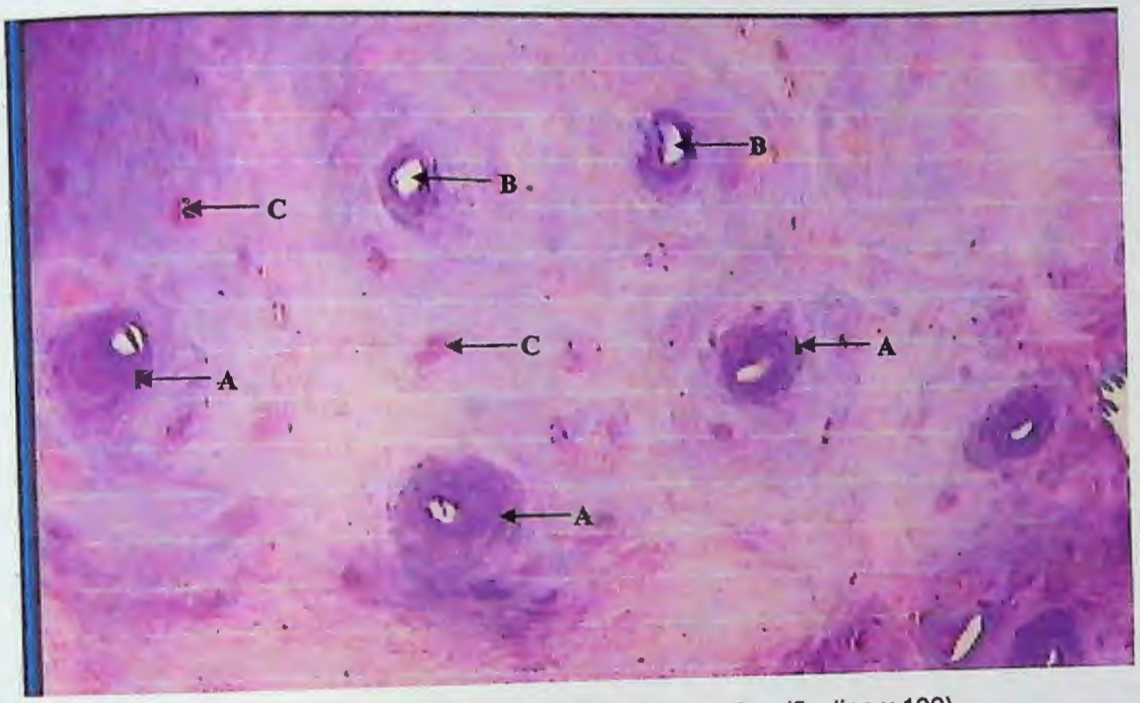


Fig.-3.6 : Raindrop pigmentation of keratoses are on the palm (⟶) indicating the keratoses



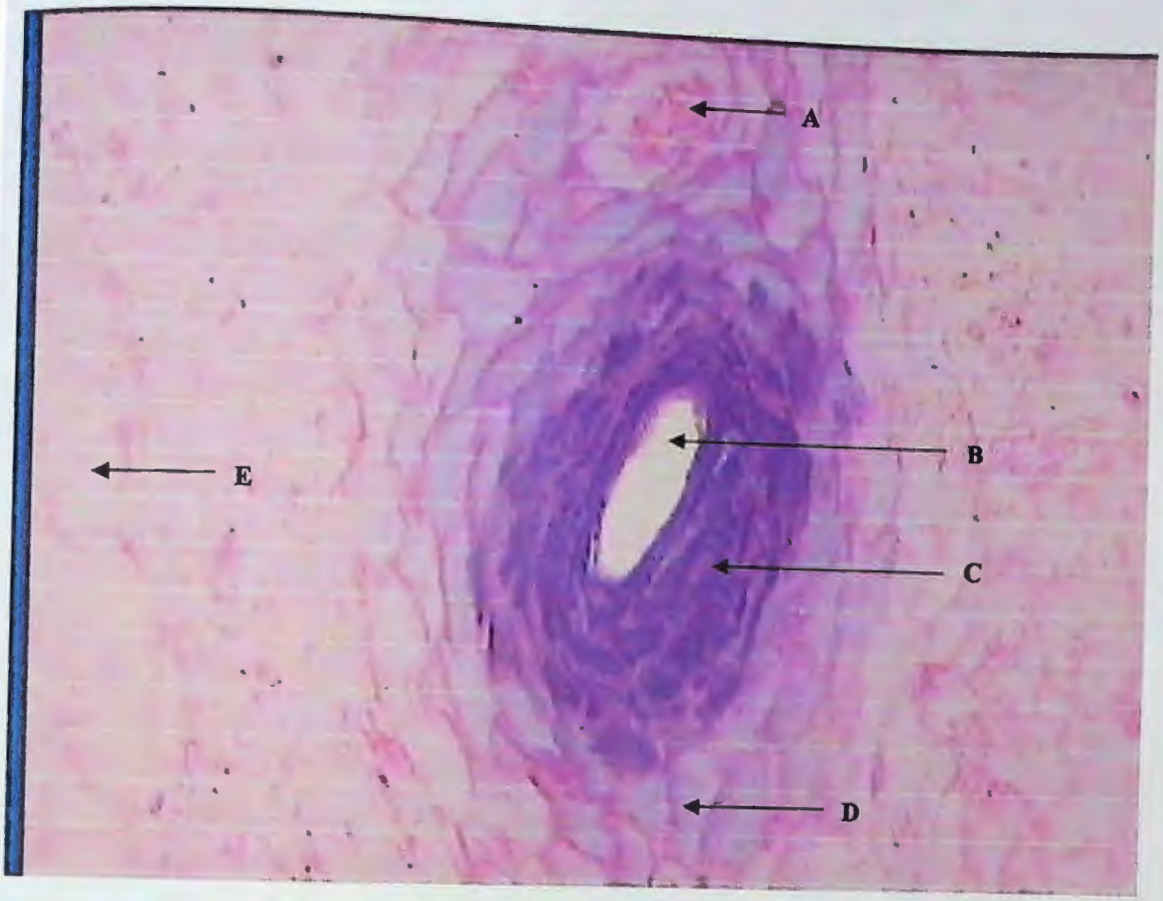
(Few keratoses are found in the epidermis. Magnification x 100)

Fig.-3.7: Microscopic picture of keratosis

A – Giant germ keratosis

B – Hole

C – Germ keratosis



(Keratosis in the epidermis. Magnification x 400)

Fig.-3.8: Microscopic picture of keratosis

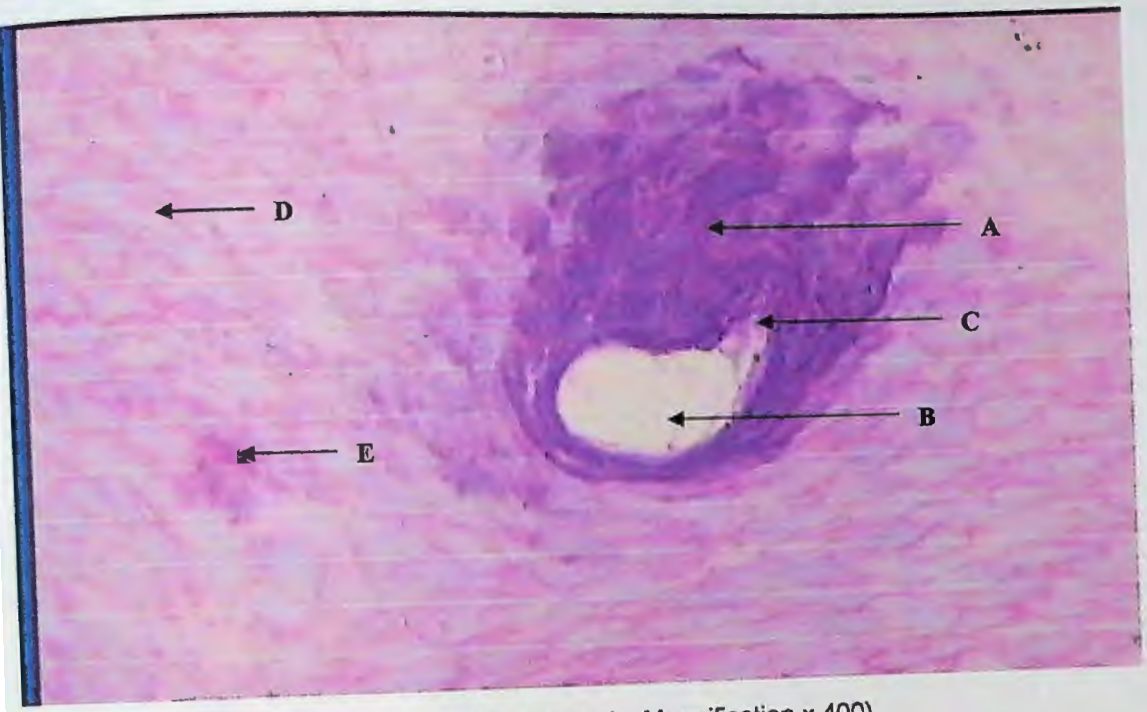
A – Shape changed in keratin cell

B – Large oval hole

C – De-oxygenated dis-shaped keratin cell surrounding the hole

D – Shape changing of keratin cell

E – Normal keratin cells



(Keratosis in the epidermis. Magnification x 400)

Fig.-3.9: Microscopic picture of keratosis

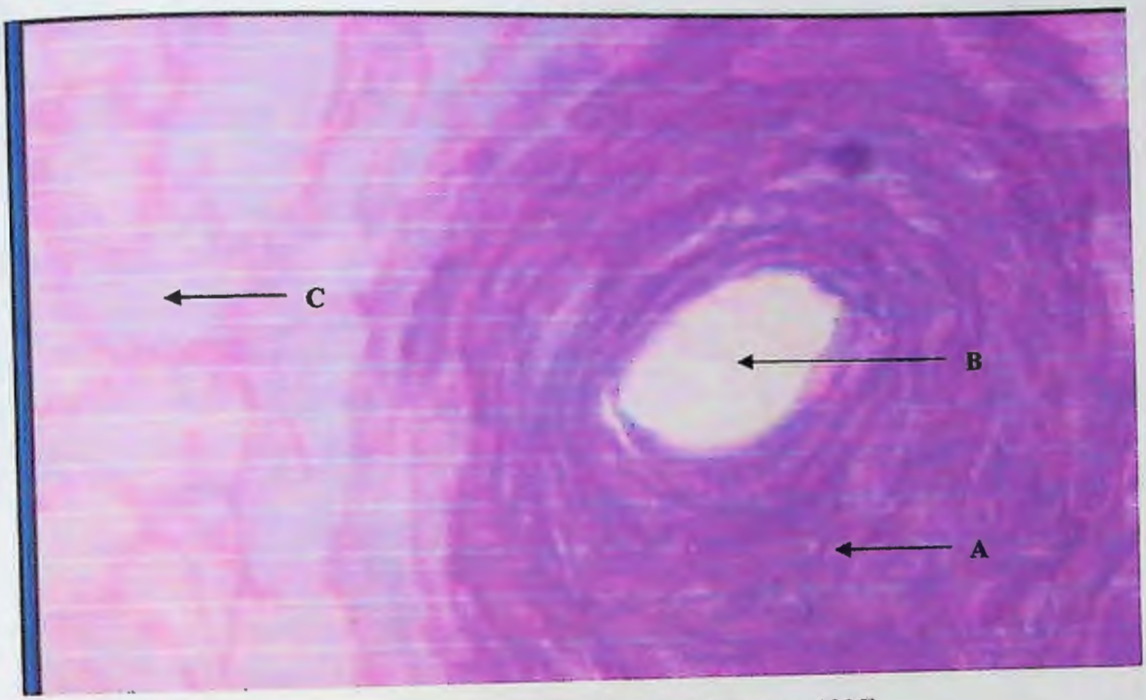
A – De-oxygenated dis-shaped keratin cells

B – Oval hole

C – Hole is spreading by breaking de-oxygenated cells

D – Normal keratin cells

E – Destroying the nature of keratin cells



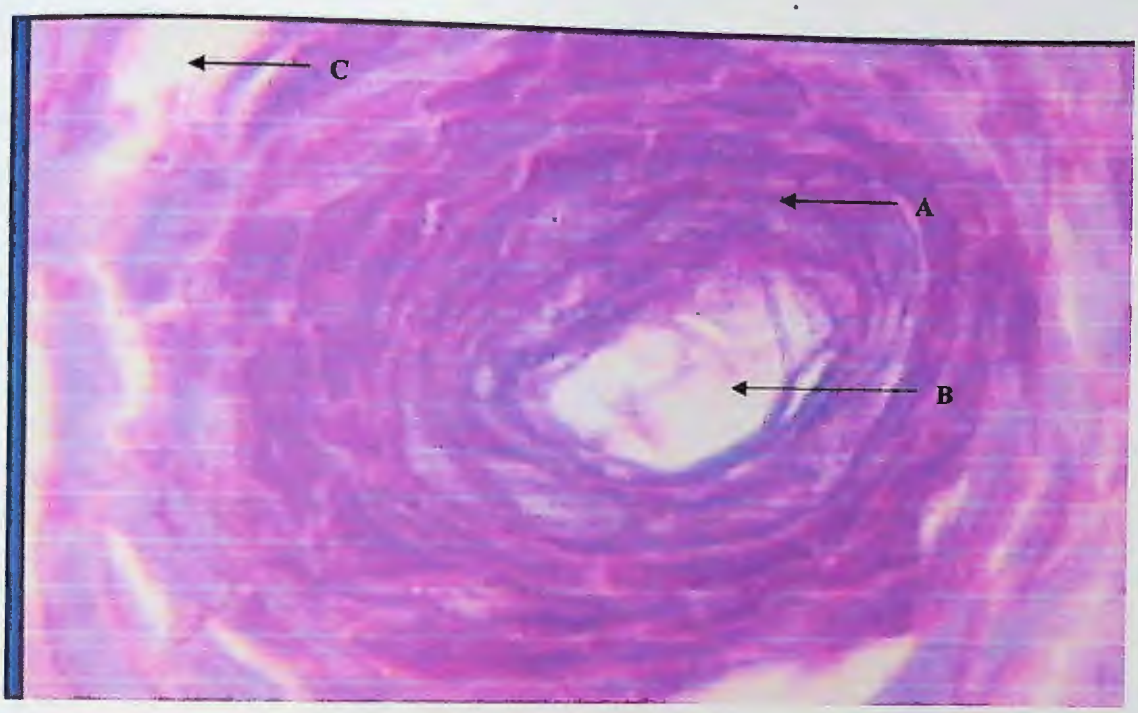
(Keratosis in the epidermis. Magnification x 1000)

Fig.-3.10: Microscopic picture of keratosis

A – De-oxygenated dis-shaped keratin calls

B – Oval hole in the centre

C – Normal keratin cells



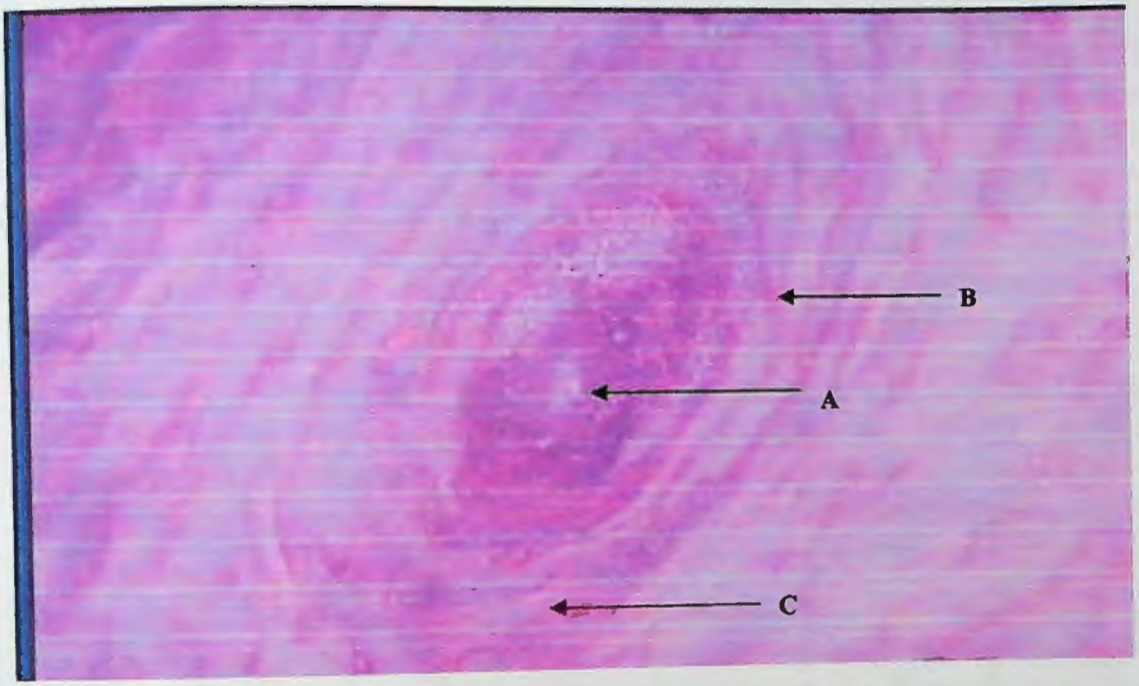
(Keratosis in the epidermis. Magnification x 1000)

Fig.-3.11 : Microscopic picture of keratosis, centre ruptured

A – De-oxygenated dis-shaped keratin calls

B – Oval hole in the centre

C – Breaking of normal keratin cells outside the de-oxygenated cells



(Keratosis in the epidermis. Magnification x 1000)

Fig.-3.12 : Microscopic picture of keratosis, centre is intact.

A – Intact de-oxygenated keratin cells in the centre (no evidence of hole)

B – De oxygenated dis-shaped keratin cells

C – Normal cells are destroying its nature

3.5 Clinical Features and Complications

All patients presented with raindrop skin pigmentation, that is melanosis and leucomelanosis are common in patients. Among them 5(31.25%) have keratosis of 16 patients. 7(43.75%) with white hard little nodules of 16, 3(18.75%) has cracks, 9(56.25%) have melanosis and 16(100%) have leucomelanosis in Sonargaon (Table-3.4).

Table- 3.4: Different Complications of arsenic affected people of Sonargaon Upazila

ID NO	Symptoms (Physical)												
Code	Extremities				Chest			Back			Face		
0512	AHFLT	Spots	Pimple	Crack	Spot	Pimple	Crack	Spot	Pimple	Crack	Spot	Pimple	Crack
001	AHF	BW	B		B			B			B		
002	HF		B								W		
003	F	B			W								
004	H F		B	C	W			W					
005	L	W			W						W		
006	HL		W		A-BW			BW					
007	HF		BW	C	BW	BW		BW					
008	HF		W		BW								
009	HT	BW	W		BW			B					
010	LHF	L-W	W		W								
011	T	W			W								
012	HF		W		W								
013	HF	AL-W	W		W			W					
014					BW			BW					
015	AF	BW	B		BW								
016	HF	BW		C	W			W					

Notes: A- Arm, H- Hand, F- Feet, L- Leg, T- Thigh; B- Black, W- White, C- Crack.

Black pimples-Keratosis, Black spots-Melanosis, White spots-Leucomelanosis.

In Sujanagar, Ahmadpur village was another picture. In male, there were 4(11.8%) melanosis, 26(76.4%) were melanosis with keratosis, and 4(11.8%) for absolute keratosis. In female, 5(17.9%) had melanosis, 19(67.9%) for melanosis with keratosis, and 4(14.2%) for absolute keratosis.

All together in male and female, 9(14.52%) have melanosis, 45(72.58%) have melanosis with kerarosis, and 8(12.9%) have keratosis only (Table-3.5a and 3.5b).

Except these arsenicosis manifestation mostly all patients noticed about physical weakness, burning sensation in chest and body, dryness and anxiety. Few patients complaints of nausea, vomiting, headache, cough. Some had abdominal colic, aversion to food. Some had chest pain, dyspnoea and asthmatic problem. But one was found with kidney problem and oedema of legs (P.no.- 1001) in Ahmadpur. It is mentionable that his one brother and one sister died of oedema of legs. Another casualty found in Sagarkandi, that is, one patient found with skin ulceration on chest, back and extremities (P.no.-3002). His two brothers died of the ulceration (Fig.- 3.13, 3.14 3.14).

Important is that, oedema of leg and skin ulceration were rare cases. But these complications turn a patient to his death.

Table- 3.5a: Complications of arsenic affected people (male) of Ahmadpur village

ID NO.	Symptoms (Physical)												
	Extremities				Chest			Back			Face		
	AHFLT	Spots	Pimple	Crack	Spot	Pimple	Crack	Spot	Pimple	Crack	Spot	Pimple	Crack
1001	HF		B										
1002	HF	B	B										
1003	H		B										
1004	HF	B		C				BW					
1005	HF	B	B										
1006	HF	B	B										
1007	AHL	BW	B		BW								
1008	AL	B											
1009	HF	B	B										
1010	H	B	B										
1011	LF	B	B										
1012	LF	B	B										
1013	HF	B	B		B			B					
1014	HL	B	B										
1015	AHF	B			B			B					
1016	LF	B	B										
1017	ALF	B	B										
1018	HF	B	B										
1019	H	B	B										
1020	H		B										
1021	HF		B										
1022	HF	B	B										
1023	AL	BW			W								
1024	LF	B	B										
1025	F	B	B		B								
1026	H	B	BW										
1027	F		B										
1028	AHLF	B	B										
1029	HF	B	B										
1030	LF	BW			B								
1031	HF	B	B										
1032	HL	B	B										
1033	HF	B	B										
1034	H	B	B										

Notes: A- Arm, **H**- Hand, **F**- Feet, **L**- Leg, **T**- Thigh; **B**- Black, **W**- White, **C**- Crack.

Black pimples-Keratosis, **Black spots**-Melanosis, **White spots**-Leucomelanosis.

Table- 3.5b: Complications of arsenic affected people (female) of Ahmadpur village

ID NO.	Symptoms (Physical)												
	Extremities				Chest			Back			Face		
	AHFLT	Spots	Pimple	Crack	Spot	Pimple	Crack	Spot	Pimple	Crack	Spot	Pimple	Crack
2001	AFL	BW											
2002	HF	B	B										
2003	HF	B	B										
2004	LF	B	B										
2005	HL	B	B										
2006	HL	B	B										
2007	HF	B	B										
2008	HF	B	B										
2009	H		B										
2010	AL	BW			B								
2011	AL	BW			BW								
2012	H		B										
2013	HF	B	B										
2014	HF	B	B										
2015	AL	BW			B								
2016	AL	BW			B								
2017	HF	B	B										
2018	HF	B	B										
2019	HF	B	B										
2020	H		B										
2021	HF	B	B										
2022	HF	B	B										
2023	H		B										
2024	HFL	B	B										
2025	AHL	B	B		BW								
2026	AHL	B	B		B								
2027	HFL	B	B										
2028	HF	B	B										

Notes: **A**- Arm, **H**- Hand, **F**- Feet, **L**- Leg, **T**- Thigh; **B**- Black, **W**- White, **C**- Crack.

Black pimples-Keratosis, **Black spots**-Melanosis, **White spots**-Leucomelanosis.

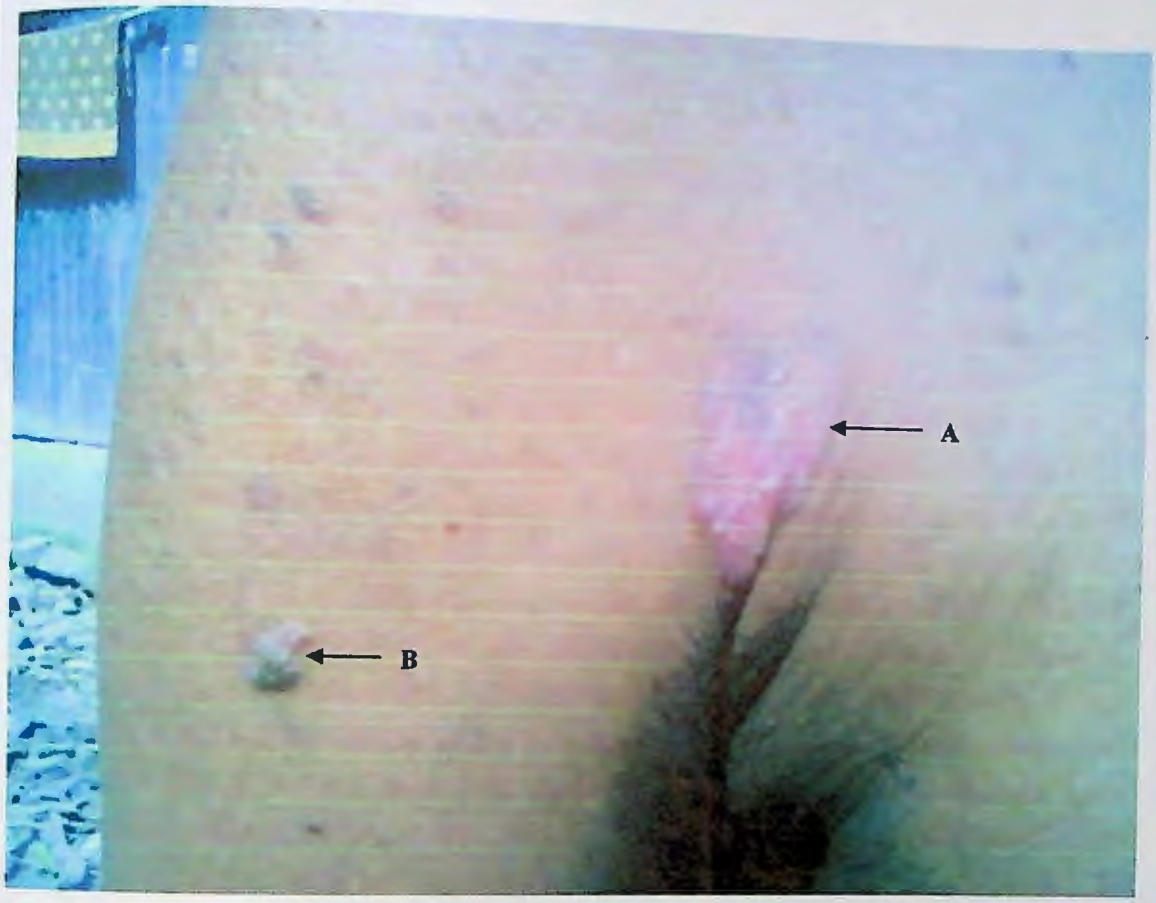


Figure-3.13: Melanosis, Leucomelanosis and rotten ulcer of arsenicosis patient.

A – Rotten ulcer

B – Hard eruption on melanosis

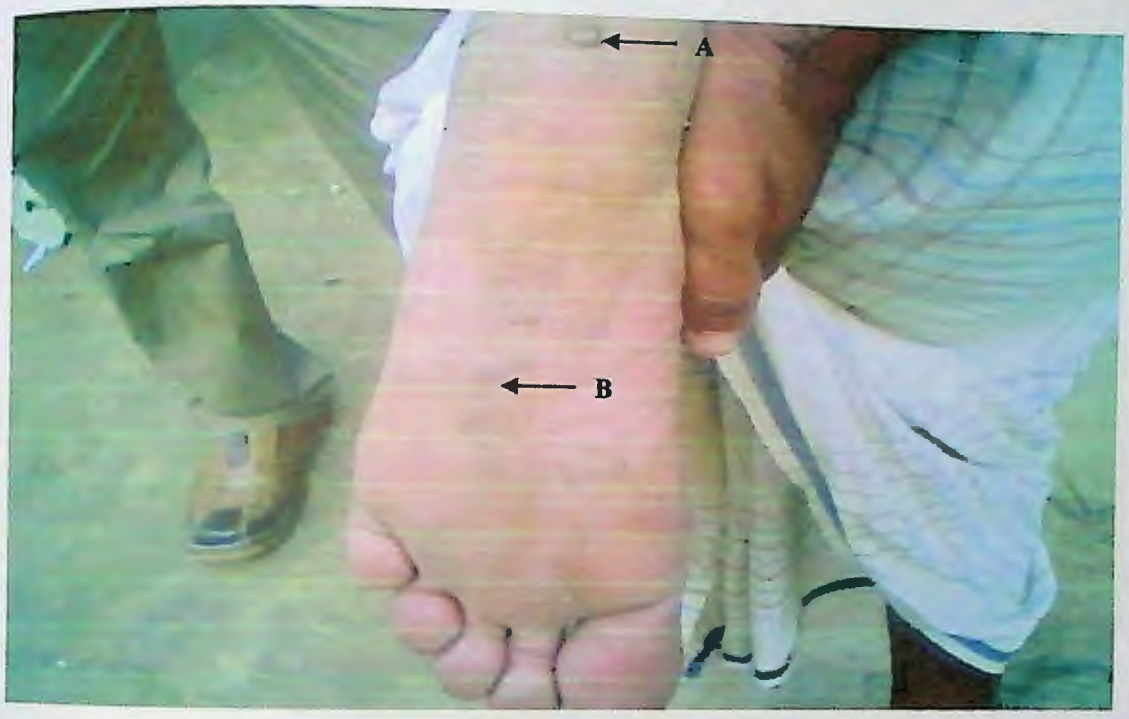


Figure-3.14: Spoted Keratosis and Keratosis on the foot of arsenicosis patient in Sujanagar

A – Spoted Keratosis

B – Newly growing Keratosis

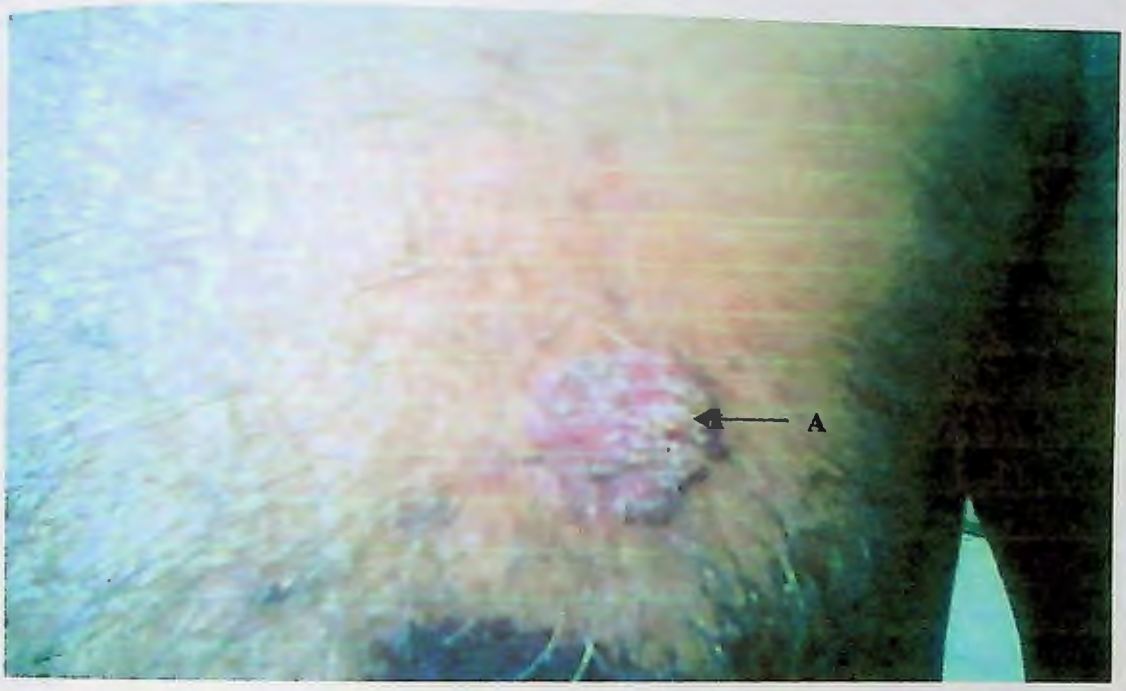


Figure-3.15: Ulceration on arsenicosis patient in Sujanagar, Pabna

A – Ulceration turning to necrosis

3.6 Results after Applying Medicines in Sonargaon

Four (4) selected medicines were applied on 16 patients of 4 (four) groups of arsenic effected patients. There were more severe and less severe patients in between them. There were melanosis, luco-melanosis, keratosis, hyperkeratosis, cracks symptoms in the patients.

There had found most satisfactory results by the research. The results were as follows –

A. Results After 2nd Month

In **1st phase of treatment,** the patients were divided into 4 (four) groups. Each group had 4 (four) patients. Separately indicated medicines were chosen for each group. The date of application of the medicines was **21st February 2006.** The results (**on 1st April 2006**) of these groups were as follows–

G-1: Black keratoses were returned to white color. Little transparent nodules felt on touch beneath the skin of the palm.
No changes of melanosis and lucomelanosis.

G-2 : Not any particular changes were found.

G-3 : White nodules were found only at the border of the palm.
No other changes were found.

G-4 : Black keratosis were returned to white color. Patient claims keratosis were reducing in size.
Melanosis and lucomelanosis were reducing.

B. Results After 4th Month

Same medicines were applied on the patients **at 1st April 2006** for the next 2 (two) months. In maximum cases little improvement found in these days. But few had miraculous result.

* **The group wise results (on 8th June 2006)** were as follows-

G-1: Normally the **white nodules** (before 1st. treatment it was **black keratosis**) were not found. The evidences of the nodules were felt after pressing on the palm (in male).

In female, hard white nodules were found as usual. Her full palm skin was hard from previously (before treatment).

G-2: White spots (pigments) were as usual.

White and black nodules were as usual.

G-3: No trace of white nodules on the sides of the palm (in female).

Other parts of the palm skin were soft (in female).

White and black pigments were as usual (in both cases).

G-4: The areas of white nodules (previously black keratosis) were reducing (in female).

White and black pigments were reducing from chest (in male).

No changes of white pigments of the leg (in female).

* **Person wise results on 8th June 2006:**

1. *Patients of G-2 had no result. So G-1 group medicine (M-5) was selected on behalf of G-2.*
2. *Patient number 003,004, 011, 014, 016 had no results. So they had taken in a separate group (G-5), because of their symptoms were similar and given them another medicine (M-6).*
3. *And two patients (005,015) were discarded from the treatment, because they were not interested to take medicines.*
4. *Patient number 010 had tremendous result.* **** She has no keratosis on her palm.*** *But there were not other persons of the group in the field.*

C. Results After 5th Month (Intermediate result)

Some changes came on the application of medicines (**on 8th June, 2006**). Patients group G-1 and G-2 combined together. And patient number 003, 011, 016 were separated for G-5. One new medicine (M-6) was applied on few unchanged (G-5) patients (on **6th July 2006**). After one month of the treatment, there were found a great change in the patients. All the patients were divided into two groups: (1) Patients with keratosis and (2) patients with pigments (Melanosis and Lucomelanosis). There were found one medicine sensitive on keratosis. But no results were found for the pigments. The differential diagnosis was as follows **(group wise)**-

G-1: Found medicinal aggravation for producing long term same medicine.

G-2: Found no changes.

G-3: No evidence of keratosis on the palm.

G-4: Found medicinal aggravation.

*** Person wise results found on 6th July 2006:**

1. *Patient no.002, 009, 010 had **better results** of keratosis than previous trial.*
2. *P.no.012 had an excellent result in this term. **He had no keratosis on his palm and feet.** Previously he was absent in the field.*
3. *P.no.004 had no results.*
4. *P.no.005 and 015were cancelled previously.*
5. *P. no.003, 004, 011, 014, 016 had no remarkable results.*

D. Result after 6th Month (full term result)

Full term treatment was divided into two parts. These were-

i) Part-1 Result

Here were only two groups. *i.e.*- **1**- for keratosis and **2**- for pigments. According to the previous result, there had found a better result by **M-2 for keratosis.** And a new medicine was taken **(M-6) for pigments.** The application date of these medicines was **at 6th July 2006.**

***Group wise results were obtained** (on **2nd August 2006**) as follows-

PG-1: Continuing reducement of keratosis and somewhere no evidence of keratosis.

PG-2: No remarkable changes found. One patient had slight change in pigment.

*** Person wise results on 2nd August 2006:**

1. *Patient no. 001, 002 had slight nodules.*
2. *P.no. 008 had no keratosis, but feels slight hardness after washing cloths.*
3. *P.no. 009,010, 012 had no complaints.*
4. *P.no. 014 reducing brown pigments of the chest.*
5. *P. no.003, 004, 014, 016 had no changes on pigments*

ii) Part-2 Result-

The application date of these medicines was at **2nd August 2006** and the results were obtained on **5th September 2006.**

*** Group wise results** are as follows-

PG-1: keratosis reduced in many patients.

PG-2: No pigments in some patients.

*** Person wise result on 5th September 2006 (2nd part):**

1. *Patient no. 001, 002 had satisfactorily reduced the keratosis.*
2. *P.no. 007, 008, 013 had reducing keratosis.*
3. *P.no. 014, 016 had no pigments on chest, palm and legs.*
4. *P. no. 004, 011 had reducing pigments.*

E. Final Scoring of the Results

*** Person wise results on 4th October 2006:**

1. *P. no. 001, 009, 010, 012 had no keratosis.*
2. *P. no. 002, 007, 008, 013 had reducing keratosis.*
3. *P. no. 006 had no change of keratosis.*
4. *P. no. 004,011, 014, 016 had no melanosis.*
5. *P. no. 003 had no change of melanosis.*

3.7 Selection of Highly Effective Medicines

For the first time trial in Sonargaon, there had chosen 7 (seven) medicines which were seem to be better for the treatment of the arsenicosis. All the medicines, we found working on the patients. But tremendous result found in few of them-

A. For keratosis

Three medicines were found effective-

1. ***Calcaria fluoricum*** - 1 (12.5%), out of eight (8) patients,
2. ***Silicea*** - 2 (50%), out of four (4) patients and
3. ***Antimonium crudatum*** – 7 (77.7%), out of nine (9) patients.

B. For Melanosis

Two medicines were found effective-

1. ***Ars. Sulph. Flabum*** – 1 (20%), out of five (5) patients and
2. ***Arsenicum Album*** - 3 (60%), out of five (5) patients.

These were the highly effective medicines in the case of arsenicosis disease treatment.

3.8 Results after Applying Highly Effective Medicines in Sujanagar

In Ahmadpur of Sujanagar upazilla, the total numbers of the patients were 62, among them 34 males and 28 females. 24 patients were recovered out of 43 patients with 3 different medicines. 19 patients were irregular and absent. The tabular formed results are mentioned below:

Table –3.6: Results after applying of medicines on the patients of Ahmadpur

Sl. No.	Medicine Name	Medicine Code	Total number of patients			Recovered patients			No changed			Absent		
			Male	Female	Total	Male	Female	Total	Male	Female	Total	Male	Female	Total
1	Ars. Alb.	M-1	14	12	26	7	6	13	2	1	3	5	5	10
2	Antim Crud.	M-2	7	6	13	3	2	5	2	2	4	2	2	4
3	Cuprum met.	M-7	13	10	23	3	3	6	7	5	12	3	2	5
		TOTAL	34	28	62	13	11	24	11	8	19	10	9	19

a. For *Arsenicum Album*

There were treated 26 patients with the medicine *Arsenicum album*. Here 10 patients were absent and discontinued. 13 (81.25%) patients were recovered and 3(18.75%) patients had no changes of 16 treated patients. By the figure, *Arsenicum album* got 81.25% of recovery (Table-3.6).

b. For *Cuprum Metallicum*

There were treated 23 patients with *Cuprum metallicum*. 5 patients found absent and discontinued in this case. 6(33.3%) patients were recovered and 12(66.7%) patients had no changes of 18 treated patients. According to the figure, *Cuprum metallicum* counted 33.3% of recovery (Table-3.6).

c. For *Antimonium Crudatum*

There were selected 13 patients for *Antimonium crudatum*. There 4 patients were absent and discontinued. 5(55.6%) patients recovered and 5(44.4%) patients had no changes out of 9 treated patients. *Antimonium crudatum* found only 55.6% of recovery (Table-3.6).

3.9 Overall Treatment Results

The general phenomenon is this, **after one month** of applying medicine patients feel comfort mentally, their physical symptoms are disappeared. That means, anxiety, physical weakness, vertigo, palpitation, anorexia, insomnia etc. disappeared.

In the **second month**, softening starts on keratosis, but no changes on melanosis and leucomelanosis (Case no. 1005,1006). Severe cases take several weeks to softening the keratosis. In case of ulceration, in between second month, ulceration stars recovering (Case no.1004). Oedema reduced and again swelled for his food habit and stand for a long time (Case no. 1001).

The **permanent recovery** starts in the **third month.** Softening of keratosis started to disappear (Fig.-3.16). White nodules (changed form of keratosis) started to disappear in this month (Case no. 2012).

Maximum recovery performed in third and **fourth month.** Few cases take more times to recovery (Case no.1001). These are oedema, deep seated ulceration, arsenic pneumonia, arsenic asthma with dyspnoea etc.

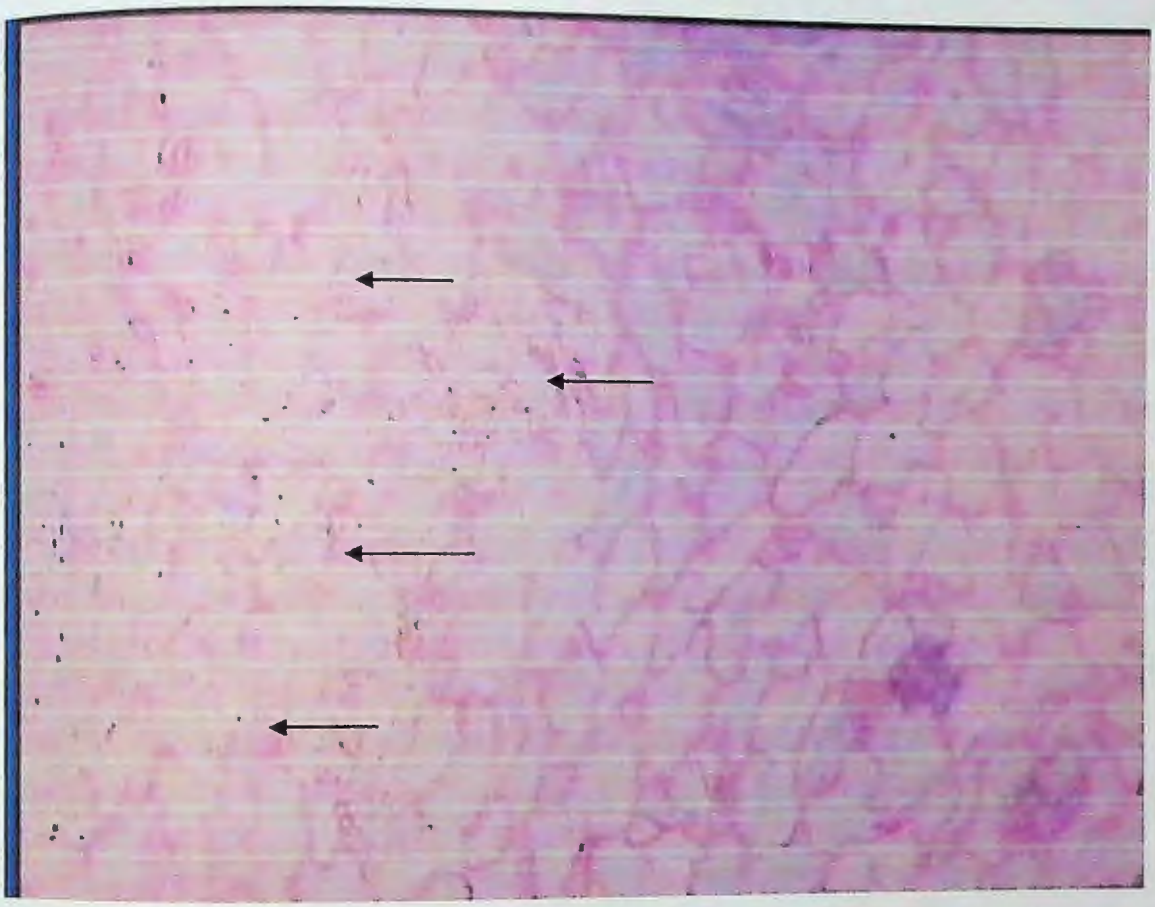


Figure-3.16: Microscopic picture of the palm after recovery of Keratosis (×1000)

(←) indicating the recovered stage of keratin cells

CHAPTER – 4

DISCUSSION

The deadly disease arsenicosis is spreading worldwide and the situation of Bangladesh is so critical that a lot of people will die with arsenicosis in Bangladesh within near future (WHO, 2001). A good number of workers like Heyman *et al.*, 1956; Kyle and Pease, 1965; Hine *et al.*, 1977; Feldman *et al.*, 1979; Blom *et al.*, 1985; Langerkvist *et al.*, 1986; ATSDR 1989; Morton and Caron, 1989, (all in Pouls and Payne, 2008) mentioned that, 'repeat exposure to arsenic compounds have been shown to lead to the development of peripheral neuropathy, encephalopathy, cardiovascular distress, peripheral vascular disease, EEG abnormalities, Raynaud's phenomenon, gangrene of the lower legs ("Black foot disease"), acrocyanosis, increased vasoplastic reactivity in the fingers, kidney and liver damage, hypertension, myocardial infarction, anemia and leukopenia. Other chronic effects of arsenic intoxication are skin abnormalities (darkening of the skin and the appearance of small "corns" or "warts" on the palms, soles, and torso), neurotoxic effects, chronic respiratory diseases (pharyngitits, laryngitis, pulmonary insufficiency), neurological disorders, dementia, cognitive impairment, hearing loss and cardiovascular disease. A significantly higher percentage of spontaneous abortions have been shown in a population living near a copper smelting plant; lower birth weights of babies born to this same population are seen, and an abnormal percentage of male to female births is also apparent, suggesting that arsenic affects babies in utero (Nordstrom *et al.*, 1979). Studies have shown close associations between both inhaled and ingested arsenic and cancer rates. Cancers of the

skin, liver, respiratory tract and gastrointestinal tract are well documented in regards to arsenic exposure (IARC, 1980; Lee-Feldstein, 1989)'.

The above pen-picture inspired the researcher to investigate the arsenic and arsenic situations in two upazilas *viz.* Sonargaon and Sujanagar of Bangladesh. As well as to disclose the status of drinking water and arsenicosis with its relations to age, occupation and socio-economic conditions of the victims, especially with an aim to prevent the consequences of arsenicosis applying the principle of Homoeopathy.

4.1 Victim of Arsenic Poisoning:

4.1.1 Age Relation with Arsenic Poisoning

Development of arsenicosis through arsenic poisoning is influenced by several means of contamination, physiological and socioeconomic conditions of an individual. It has been obviously found that people of middle age group are more vulnerable to arsenicosis. Chen *et al.*, (1988) found that the maximum arsenic affected people were from the age group 30 to 69 years (table-4.1).

Table 4.1: Number of Blackfoot disease patients in the Nonconcurrent cohort from 1968-1983 (Chen *et al.*, 1988)

Age	Patient number	
	Man	Woman
0 to 9	4	1
10-19	6	1
20-29	20	19
30-39	50	63
40-49	96	89
50-59	145	111
60-69	108	59
≥ 70	8	9
Total	437	352

In another study, Mitra *et al.*, (2002) enrolled 150 subjects (75 males and 75 females) by convenience sampling of which 48% patients were young adults.

The present study also revealed that among the total affected people 5.88%, 76.48%, 11.76% and 5.88% belonged to the age groups 0-20, 21-40, 41-60 and 60+ respectively in Sonargaon, and 8.06%, 58.07%, 25.81% and 8.06% belongs to the respective age groups in Sujanagar upazila (Table-3.2).

Sonargaon is situated beside the river Meghna and Sujanagar is on the bank of river Padma. Moreover, these two upazilas are hundreds of kilometers apart from each other. But the arsenic affection pictures on

different age groups were more or less same in these two areas. Mainly the people of middle age group were found much more arsenic affected in both of the upazilas.

4.1.2 Occupation Relation with Arsenic Poisoning

Occupation is very important in arsenicosis patients. Low earning people were the main victim of arsenic poisoning. Low earning people are always suffering from protein deficiency. Daily requirement of protein for each adult is 40-70 grams, which is beyond the capacity of low earning group of people. Milton, *et al.* (2004) in an experimental study found that certain low protein diets resulted in decreased excretion of DMA (Dimethyl arsenic acid) and increased tissue retention of arsenic which in-turn may increase individuals suspicious to chronic arsenic toxicity.

Mitra *et al.*, (2002) studied on 150 patients (75 males and 75 females). They were found in the demographic status, two-thirds (66%) of the patients came from poor socioeconomic class with an average monthly income of less than US$ 60. About 10% did not have any formal education. The social background of the study patients was similar to that usually seen in the hospital.

In relation to our study, the poor people of Haria and Ramgonj village of Sonargaon upazila were really very low earning people in their socioeconomic condition. Maximum males were fish seller (37.5%) and females were mainly housewives (31.25%). More or less same pictures were found in Ahmadpur village of Sujanagar upazila. Most of the males were associated with business (32.3%), such as shopkeepers (22.6%), little

item seller (6.5%), fish seller (1.6%) and rickshaw puller (1.6%) followed by farmers (11.3%). Females were mainly housewives (43.5%). Socio-economically they were earning very little to maintain their family. By all of these circumstances the research has disclosed that the poor socioeconomic condition is one of the vital causes of arsenic poisoning.

4.2 Awareness on Arsenic Contaminated water and Arsenicosis

In Sonargaon, people of Haria and Ramgonj villages were using 3 (three) and 1 (one) shallow tubewells respectively from many years. DPHE and NGO workers noticed them that their tubewells were arsenic contaminated. They had passed 9 (nine) years to sunk new tubewells, due to their poverty. New tubewells were semi-deep (350 ft) type of tubewells. Although public health engineering (DPHE) office had deep tubewells (500 ft) for the people. But these were beyond the capacity of the poor people. Here one thing is important, Choudhury *et al.*, (2004) said that the deep tubewells, which had the depth of 500 ft or more, these had been found to be free from arsenic contamination. It is important that there is a hole on a thick layer of rock in between shallow and deep aquifer. The hole is made by drilling on the thick rocky (silt and clay) layer. As arsenic is heavy metal, it will go down to deep aquifer from the shallow one. In near future, the deep aquifer will be another arsenic contaminated zone.

We have tested their new semi-deep tubewells, 4 (four) in Haria and 1 (one) in Ramgonj village. We found arsenic in every one in below standard levels (0.01 - 0.05 mg/l) and 2 (two) in danger level (0.10 mg/l and 0.30 mg/l). We have suggested them to drink only the low standard leveled

tubewells water. In Sujanagar, there is no use of tubewell water. Danida produces them well water reserve tanks and a generator to pull the water to up tank. Rather than they have river, cannels, ponds and rain water reservoir.

In case of awareness, public has no hurry to test their tubewells water and it is also remarkable that the government has no yearly schedule to test the tubewells water. It is found that an NGO tested the tubewells water of Haria and Ramgonj villages before 9 (nine) years. And at the same time DPHE also tested those tubewells water. After that no one went there to test the water again.

In case of highly contaminated tubewells, people were using water for household works and washing purposes. People and government had no awareness about the wastewater (arsenic rich). People drained them to the fields. Crops were arsenic contaminated by the wastewater in the field. In this situation, purified river water can be acceptable as alternate safe water. In parts of Bangladesh experimental use of river water gave a progressive result instead of ground water. An NGO project for purification of Meghna river water and its supply to the houses of a village by pipeline in Sonargaon upazila is a good attempt to save the mankind from arsenic contamination. In a research, Rasul (2006) had concluded his dissertation on a topic that 'the Padma water will be the best option for the mitigation of water supply problem in Rajshahi city area in present time and future perspectives obviously'.

In case of disease awareness, people did not care about the pigmentation (melansis and lewcomelanosis) on their body. People thought these were like frickle. They had no hurry to show them to the doctors. When patients suffered with keratosis and hyperkeratosis, they were cutting them with a blade. People had no awareness for the prognosis of keratosis. Only ulceration and cancer made them concious. They went to the doctors. Here doctors were interested to notice the case to the higher authority. They had no awareness to cure them. Ultimately patients were dying with cancer.

4.3 Discussion on Analysis of Keratosis

Although in Bangladesh prospect, people are suffering from arsenic induced skin-lesions, such as- melanosis, leucomelanosis, keratosis, hyperkeratosis, dorsum, non-petting oedema, gangrene, skin cancer *etc.* found in Dhaka Community Hospital (Chakraborti *et al.*,1998). In case of keratosis, there were abnormal types of epidermis on the palm. Keratoses are hard, nodulated, oxygen and moisture free protrution like a corn. In comparison with normal one, in a section of a skin, with a stratified squamous epithelium at the surface, showing keratinization and sweat glands and hair follicles in the dermis (Appendix Fig.- 4.1).

The epidermis is the most external part of the skin and it is closely related with the atmospheric environment. Arsenic accumulates in this part of the skin and manipulates keratin layers to produce keratosis. It may be possible that arsenic and fluids come out through the blood vessels to the epidermis by osmosis process and then to thicker peripheral keratin layer (Appendix Figure- 4.2). The radius of arsenic molecule is 1.21Å (Haider,

2008) and the human beings cell pore size is 0.8 Å (Guyton, 1974). The diameter of arsenic molecule is bigger than the cell pore, so arsenic molecule can not enter the cell endometrium (Appendix Figure- 4.3 and 4.4). It remains in the interstitial space of the palm and feet. Due to the keratinization process arsenic molecule come to the keratin layer and form keratosis.

The dead and keratinized cells were remarkably important for our research. The arsenic affected people had keratosis in his/her palm and feet in the keratinized area. Keratosis was nothing, but a dense accumulation of keratin surrounding a hole and its color is black, due to oxygen deficiency. Generally stratum corneums were the flattened plates of the dead keratin substances. But there had found dis-shaped keratins. These dis-shaped keratosis were from a pin head to a giant size in naked eye. These were harder than the normal flattened plates of keratin. The pin headed keratoses were hard and transparent. But a giant keratosis was harder, more dense and blackish in color. Colors were simultaneously deeper from the beginning to a big size of keratosis. Keratosis attached together when they came to very closer to each other. Naturally dead keratinized cells were derived from water and they provide the dust like plaques of keratin that were rubbed off by abrasion. The colonial keratoses were more breakable due to water deficiency, deficit of elasticity and more hardness of the keratins. The cracks start from the outer surface of the skin that was the distal part of the stratum corneum. And it runs to the inner part of keratin. Then the cracks touched dermis. The dermis contains very thin blood vessels. These vessels burst out due to the cracks of the stratum corneum and blood flows to outside. Lastly

infection take place and infection turned to ulcer or/and gangrene and then to epithelial cancer, especially Bowen's disease, squamus cell carcinoma of the skin and basal cell carcinoma of the skin.

For the microscopic examination of keratosis, the surface epithelium of the skin was cut out from the palm. As the thick layer of keratin was present on palm, the microscopic photographs showed the black coloured keratosis with a hole in the centre and surroundings were keratin layers, the stratified epithelium, only the cells of the basal layer lie on the basal lamina. The normal keratin cells had the best access to nutrients and in the most protected position (Fig.- E of 3.8, D of 3.9, C of 3.10).

But in case of keratosis formed in the epidermis (surface epithelium), due to the presence of metallic arsenic. Photographs showed that around arsenic molecules the keratin cells were dis-shaped and all the cells were toward the centre. The metallic arsenic gathers oxygen due to its chemical change. So the cells become black for lack of oxygen. This is natural phenomenon for arsenic and also for the cells.

4.4 Medicinal Action on Keratosis and Melanosis

It is very much true that anti-oxidants are helpful to arsenicosis patients. In many countries vitamin-A, vitamin-E and selenium are applying on the patients. Somewhere found keen improvement with them, *i.e.* - recovering physical weakness and vertigo for few days. But it can not cure it completely.

As the problem is functional, so in our research medicines of chemical source has been considered as priority medicines. Seven (7) medicines were selected, seems to be better according to their clinical symptoms for the trial of arsenicosis curing. These were- *Calcaria fluoricum* (fluoride of lime), *Alumen* (common potash alum), *Antimonium crudatum* (sulphate of antimony), *Silicea* (silicon di-oxide), *Cuprum metallicum* (Metallic copper), *Arsenicum Sulphuricum flabum* (Arsenic tri-sulphate) and *Arsenicum album* (arsenic tri-oxide) itself.

In homoeopathy a medicine may act as molecular level or in energy form. Very low potencies are molecular, and medium and high potencies are beyond Avogadro's number means energy level. Here medium and high potencies (30, 200) of homoeopathic medicines were used to restore health from arsenicosis. Homoeopathy follows the scientific rule 'Similar Repeals Similars'. Potential energy of the matter works here as a medicinal power.

Calcaria fluoricum, Alumen, Antimonium crudatum and *Silicea* have a great action on skin, especially on epidermis and dermis. Hard tumors of epidermis, dermis or muscles are the field of *Calcaria fluoricum*. Indurations of the glands and epithelioma are the main field of *Alumen* and there is indurations resulting from long continued inflammatory irritations. *Antimonium crudatum* has nodulated substances on epidermis, mainly on keratin. It has a great action on corn of palm and feet. Warts and dry gangrenes are also under consideration of this medicine. *Silicea* is a great medicine under the same circumstances. It has a curative action against indurations of tumors, keloid growth, long lasting suppuration and fistulous

tracts, old fistulous ulcers. It has a great effect on the foreign bodies to expel them out (Mathur, 1984). All of these medicinal symptoms are alike to the symptoms to keratosis, *i.e.* - hard nodules on the palm and feet developed due to arsenic accumulation.

In course of the study tanure it was found that *Antimonium Crudatum* and *Silicea* scored 77.7% and 50% in curing keratosis in Sonargaon, where patients were nine (9) and four (4) for each medicine respectively. *Antimonium crudatum* and *Arsenicum album* were scored 55.6% and 81.25% respectively in Sujanagar. *Antimonium crudatum* cured five (5) patients within nine (9) and *Arsenicum album* cured thirteen (13) within sixteen (16) patients.

The medicines softened the hardness of the keratosis. Ultimately nodules dissolved indeed and keratosis disappeared within few months (Fig.-3.16). Finally *Arsenicum album* was found better to remove keratosis.

Arsenicum Sulphuricum Flabum has a great effect on pigmentation of the skin. Brown and white pigments, both are under consideration of this medicine.

In arsenicosis cases, *Arsenicum sulphuricum flabum* worked on melanosis and lucomelanosis, but not so much good. Better results were obtained in case of beginning of melanosis, means scanty pigmentation.

Whereas higher potencies of *Arsenicum album* played much better role in curing both melanosis and keratosis produced as a result of arsenicosis.

Recently, University of Kalyani and Bidhan Chandra Krishi Viswavidyalaya of West Bengal worked together on arsenicosis by homoeopathic medicine *arsenicum album-30* on the basis of the rule 'similia similibus curanter'. They found *arsenicum album-30* reduced tremendously the arsenic concentration from urine and blood of arsenic affected people. They found that "the mean As content in urine and blood of the patients (Group II) before administration of the drug was higher (being 43.75 ppb in urine and 24.13 ppb in blood) than the normal permissible range (3.33-25.55 ppb and 0.3-2.0 ppb, respectively). The content in urine at 7^{th} day after administration of the drug was found to rise up to a strikingly high level (62.30 ppb), but thereafter it steadily declined to reach almost the lower limit of normal range by 45^{th} day (10.78 ppb)" (Khuda-Bukhsh, *et al.*, 2005).

Cuprum metallicum has a great affinity on the skin problem like melanosis, hardness of the skin and rough brown skin with pimples. But presently the medicine was not found to be effective upto the mark of satisfaction.

4.5 Finding Preventive Medicines

The effective medicines, which are using against arsenicosis disease, basically they react chemically against metallic arsenic giving it way to pass out through the barrier in arsenic chain in the body. The medicines are clearing the obstacles and accelerate arsenic expel out from the body easily.

The government initiatives for arsenic control are the provision for arsenic safe water, antioxidents, such as vitamins A, C, E and keratolytic ointment which will help the improvement of skin menifestations of chronic arsenicosis (APSU/DPHE, 2005). According to WHO (2001), 'Control of arsenic is more complex where drinking water is obtained from many individual sources as is common in rural areas. Low arsenic water is only needed for drinking and cooking'.

In these situations of the world and Bangladesh, the treatment and prevention / control of arsenic and arsenicosis, the present research will give a keen light on life saving purposes. In the research, in arsenicosis cases, *Arsenicum album* cures the pigmentation 'melanosis' by low and medium potencies and keratosis cured by high potencies. So it is assumed that at the beginning of arsenicosis formation, medicated *arsenicum album* withstand the formation of disease.

Prevention of consequences of arsenicosis is the main subject of the research, and the arsenicosis following problems are increasing day by day. The consequences of arsenicosis are so many and so dangerous for our lives. If someone enter to secondary / metastatic / necrotic conditions of arsenicosis, it is difficult to return from it. So it is better to prevent it earlier, in preliminary stage, in melanosis and keratosis stage of arsenicosis.

So this is the right time to save the lives from the consequences of arsenic and the medicine *Arsenicum album* is recommendable to be the preventive medicine in withstanding the consequences of arsenicosis disease.

CHAPTER -5

CONCLUSION AND RECOMMENDATIONS

With excessive withdrawal of ground water to meet up the huge need for modern life activities of the ever-increasing population not only in Bangladesh but also throughout the world is facilitating increased concentration of arsenic in it. Addition of arsenic freed from various sources to soil and water and then to crops, vegetables, fish in-turn with drinking water is contaminating the human body. As a result more and more people are suffering from arsenicosis and its consequences like ulceration, oedema, gangrene in lower legs, vascular diseases, cerebro-vascular diseases, kidney and liver damages, hypertention, anemia and unrecoverable cancer. World health organization (WHO) with government and non-government organizations throughout the world is working for mitigation of such deadly environmental problem to protect human lives. In Bangladesh the problem is becoming acute day by day. Accordingly, an attempt was taken to assess the present status of arsenic concentration in drinking water of selected areas with its consequences on human body. Along with these applications of homoeopathic medicines was made to find out the effective ones for preventing those consequences of arsenic contamination.

The study reveals that arsenicosis is prevailing devastatingly in the country keeping positive relation with unsafe water, occupation, socioeconomic conditions and age of the affected people. People of middle age (20 - 60 years), low earning groups suffering from protein deficiency and mental agony are the major victims of arsenicosis. Government and non-

government organizations are working to solve the problems with sporadic efforts and applying some prosaic treatments with Selenium, Vitamin- A, Vitamin- E *etc* of course free of cost producing no successful results.

Application of homoeopathic principles for preventing arsenicosis and its complex consequences during the experimentations was considerably effective measure. It was found that medicines of chemical sources, *Arsenicum album, Antimonium crudatum* and *Silicea* cured 50%, 77.7% and 81.25% of patients treated respectively.

The principal achievement of this research is that arsenic disease protection potential for the arsenic affected area has been investigated and devised. Thus, the research is expected to be use as a guide to facilitate towards appraising the saving life in Bangladesh more comprehensively in future.

Though the research was constrained by time, resource, adequate replications, till then it has open a new avenue for thinking over the problem. In this perspective, this research is only a modest beginning. Further researches should be initiated for refinement of the methods tested in this analysis.

5.4 Recommendations

Arsenicosis disease is the most life hazardous problem for the people of the nation, as well as of the world. It is necessary to prevent the disease by any cost and by any way. The following recommendations may be consider for getting rid of this crucial problem:

(i) To combat the situation, Bangladesh need proper utilization of it's vast surface water and rainwater sources. Proper watershed management is required urgently;

(ii) Awareness should be build up to the community level and regular monitoring of water quality in the study area should be reinforced;

(iii) People must be educated properly;

(iv) Job opportunity for the poor people should be created to improve their socio-economic conditions;

(v) Sufficient protein nutrients should be ensured; and

(vi) Homoeopathic medicine ***arsenicum album*** can give a light on this problem. It is better to do more researches on the subject by Homoeopathy.

5.5 Future Research

The research, based on a small sample from a single area study, has created a mile stone by generating treatment and protecting the people. To increase the wider applicability of the research, a larger analysis all over the country is needed.

REFERENCES

Abernathy, C. 2001. Exposure and Health Effects in United Nations Synthesis Report on Arsenic in Drinking Water, World Health Organization, Geneva. Available: Web: http//www. who.int/ water_sanitation_health/ dwq/en/arsenicum 3.pdf

Adel, M.M. 1998. Biosphere III: The site of Unprecedented Ecocide in the Ganges Basin, National Documentation on the Problems of Arsenic and Farakka, International Farakka Committee, inc. New York, USA, 238pp.

Ali, M.A., Badruzzaman, A.B.M., Jalil, M.A., Hossain, M.D., Ahmed, M. F., Masud, A.A., Kamruzzaman, M. and Rahman, M.A. 2004. Fate of Arsenic Extracted with Groundwater, Fate of Arsenic in The Environment, BUET, 145pp.

APSU/DPHE, 2005. Response to Arsenic Contamination in Bangladesh, Arsenic Policy support Unit/Department of Public Health Engineering, 57pp.

Armstrong, B.K., McNulty, J.C., Levitt, L.J., Williams, K.A. and Hobbs, M. S.T. 1979. Mortality in gold and coal miners in Western Australia with special reference to lung cancer. *Br J Ind Med*, **36:** 199-205.

ATSDR, 1989. Toxicological Profile for Arsenic. Agency for Toxic Substances and Disease Registry, U.S. Public Health Service, Atlanta, GA. ATSDR/TP-88/02

BAMWSP, 2006. Upazila wise Summary Results, National Arsenic Mitigation Information Centre, Web: www.bamwsp.org

Benveniste, J., Davenas, E., Beauvais, F., Amara, J., Oberbaum, M., Robinzon, B., Miadonna, A., Tedeschi, A., Pomeranz, B., Fortner, P., Belon P, Sainte-Laudy, B., and Poitevin, B. 1988. Human Basophil Degranulation Triggered by very Dilute Antiserum against IgE, *Nature,* **333:** 816-818.

BGS, 2000. *Arsenic Contamination of Ground Water in Bangladesh,* Reports of British Geological Survey, web: www.bgs.ac. uk/arsenic/home.html

Blom, S., Lagerkvist, B. and Linderholm, H. 1985. Arsenic exposure to smelter workers: clinical and neurophysiological studies. *Scand. J. Work Environ. Health,* **11**: 265-270.

Brayer, A.F., Callahan, C.M. and Wax, P.M. 1997. Acute Arsenic Poisoning from Ingestion of "Snakes", *Pediatric Emergency Care,* **13**(6): 394-396.

Chakraborti, D., Dhar, R.K., Biswas, B.K., Chowdhury, U.K., Samanta, G., Mandal, B.K., Chowdhury, T.R., Chanda, C.R., Basu, G., Roy, S., Kabir, S. and Quamruzzaman, Q. 1998. *Ground water Arsenic Contamination and Sufferings of the People of Bangladesh,* National Documentation on the Problems of Arsenic and Farakka, International Farakka Com. Inc. New York, USA, 115pp.

Chen, C.J., Chuang, Y.C., Lin, T.M., and Wu, H.Y. 1985. Malignant neoplasm among residents of a black-foot disease endemic area in Taiwan: high-arsenic artesian well water and cancers. *Cancer Res,* **45**: 5895-5899.

Chen, C.J., Chuang, Y.C., You, S.L., Lin, T.M., and Wu, H.Y. 1986. A retrospective Study on malignant Neoplasm of Bladder, lung and Liver in Blackfoot Disease Endemic Area in Taiwan, *Br J Cancer*, **53**(3): 399-405.

Chen, C.J., Wu, M.M., Lee, S.S., Wang, J.D., Cheng, S.H. and Wu, H.Y. 1988. Artherogenicity and carcinogenicity of high-arsenic artesian well water: Multiple risk factors and related malignant neoplasms of Blackfoot disease, *Arteriosclerosis*, **8:** 452-460.

Chen, C.J. and Wang, C.J. 1990. Etiological Correlation between Arsenic level in Well Water and Age-adjusted Mortality from Malignant Neoplasm, *Cancer Res.* **50**: 5470-5474.

Cherry, N., Shaikh, K., McDonald, C., and Chowdhury, Z. 2008. Stillbirth in rural Bangladesh: arsenic exposure and other etiological factors: a report from Gonoshashthaya Kendra, *Bulletin of the World Health Organization*, **86:** 172-177.

Choudhury, M.A. I., Ahmed, M.F. and Ali, M.A. 2004. Influence of Upstream Sediment on Arsenic Contamination of Ground Water in Bangladesh, *Fate of Arsenic in the Environment*, BUET, p-145.

Cullen, N.M., Wolf, L.R. and St.Clair, D. 1995. Pediatric Arsenic Ingestion, *American Journal of Emergency Medicine*, ISSN: 0735-6757, Web: http://www.journals.elsevierhealth.com.

Cuzick, J., Sasieni, P. and Evans, S. 1992. Ingested Arsenic, Keratoses, and Bladder Cancer, *Am. J of Epidemiol.* **136** (4): 417-421.

Engel, R. and Smith, A. 1994. Arsenic in drinking water and mortality from vascular disease: an ecologic analysis in 30 counties in the United States. *Arch Environ Health*, **49**: 418-427.

Feldman, R.G., Niles, C.A., Kelly-Heyes, M., Sax, D.S., Dixon, W.J., Thompson, D.J. and Landau, E. 1979. Peripheral neuropathy in arsenic smelter workers, *Neurology*, **29**: 939-944.

Gur, G.T. 1969. Hematotoxylin Stains, *Biological Staining Methods*, 7th. Ed, p.46.

Guyton, A.C. 1971. *Transport through the cell membrane,* Text Book of Medical Physiology, 4th Ed, p.41

Hahnemann, H. 1833. Translated from the 5th edition by Dudgeon, R.E. 1893, *Organon of Medicine*, (6th ed.), First Indian edition, 1955, Aphorism no-44.

Haider, S.Z. 2008. *The size of Atoms and ions*, Introduction to Modern Inorganic Chemistry, p.107

Haq, M.M. 2002. Panitay Arsenic, Taral A Garal (Arsenic in water, Disharmony in liquid), 56pp.

Heyman, A., Pfeiffer, J.B., Willett, R.W., Taylor, H.M. 1956. Peripheral neuropathy caused by arsenical intoxication. *New England J. Med.* **254**(9): 401-409.

Hindnarsh, J., McLetchie, O.R., Heffernan, L.P.M., Hayne, O.A., Ellenberger, H.A., McCurdy, R.F. and Thiebaux, H.J. 1977. Electromyographic abnormalities in chronic environmental arsenicalism. *Clin Chem and Chem Toxicol of Metals*, **1**: 287-293.

Hine, C.H., Pinto, S.S., and Nelson, K.W. 1977. Medical problems associated with arsenic exposure. *J. Occup. Med.* **19**(6): 391-396.

Hsu, K.H., Froines, J.R. and Chen, C.J. 1997. Studies on arsenic ingestion from drinking water in northeastern Taiwan: chemical speciation and urinary metabolites. In Arsenic Exposure and Health Effects II, CO Abernathy, RL Calderon and WR Chappell, eds. New York, *Chapman and Hall*, 190-209.

IARC (International Agency for Research on Cancer), 1980. Some metals and metallic compounds, IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Some metals and metallic compounds, **23:** pp 39-142.

Jahangir, M. 1998. Molecular Evidence in Homoeopathic Medicines, 10th Homoeopathic congress, Asian Homoeopathic Medical League, pp.235-241.

Jakaria, M. 2003. Background of the Arsenic Problem, The Use of Alternative Safe Water Options to Mitigate the Arsenic Problem in Bangladesh: Community Perspective, Research Monograph Series no. **24**, BRAC, p-5.

Jensen, G.E. and Hansen, M.L. 1998. Occupational arsenic exposure and glycosylated haemoglobin. *Analyst,* **123**(1): 77-80.

Kabir, A. 2005. The Response to Arsenic Contamination in Bangladesh, A position paper, DPHE, P – IX.

Khaleque, S.A. 1982. Collection and preservation of samples, A Text Book of Pathology, 1st ed. 2nd Revised, pp.129-132.

Khuda-Bukhsh, A.R., Pathak, S., Guha, B., Karmaker, S.R., Das, J.K., Banarjee, P., Biswas, J., Mukherjee, P., Bhattacharjee, N., Choudhury, S.C., Banarjee, A., Bhadra, S., Mallick, P., Chakrabarti, J. and Mandal, B. 2005. Can Homeopathic Arsenic Remedy Combat Arsenic Poisoning in Humans Exposed to Groundwater Arsenic Concentration?: A Preliminary Report on First Human Trial, Oxford University Press, pp.537- 543.

Kusiak, R.A., Spinger, J., Ritchie, A.C., and Muller, J. 1991. Carcinoma of the lung in Ontario gold miners, Possible Aetiological Factors, *Br J of Ind Med.* **48**: 808-817.

Kyle, R.A., Pease, G.L. 1965. Hematologic aspects of arsenic intoxication, *New Eng. J. Med*, **273**(1): 18-23.

Lai, M.S., Hsueh, Y.M., Chen, C.J., Shyu, M.P., Chen, S.Y., Kuo, T.L., Wu, M.M. and Tai, T.Y. 1994. Ingested inorganic arsenic and prevalence of diabetes mellitus, *Amer J Epidemiol,* **139**: 484-492.

Langerkvist, B., Linderholm, H., and Nordberg, G.F. 1986. Vasopastic Tendency and Raynaud's Phenomenon in smelter workers exposed to arsenic, *Environ. Res.* **39**: 465-474.

Lawti, Sagun, S. 2006. Diluting the Pain of Arsenic Poisoning in Nepal, 'Progress for Children No.5: A Report Card on Water and Sanitation', UNICEF, Web: www.unicef.org/infobycountry/Nepal.

Lee-Feldstein, A. 1989. A comparison of several measures of exposure to arsenic: matched case control study of copper smelter employees. *Am. J. Epidemiol.* **129**: 112-124.

Lewis, D.R. and Calderon, R. 1998. Drinking water arsenic and non-cancer health effects, *Epidemiol*, **9**: 330-335.

Mathur, R.K.N. 1984. Systematic Materia Medica, 2nd ed. (reprint), 1034pp.

Milton, A.H., Hasan, Z., Shasidullah, S.M., Sharmin, S., Jakaria, M.D., Rahman, M., Dear, K. and Smith, W. 2004. Assciation Between Nutritional Status and Chronic Arsenic Exposure in Bangladesh, *Int. J of Envir. Health Research*, **14** (2): 99-108.

Mitra, A.K., Bose, B.K., Kabir, H., Das, B.K. and Hussain, M. 2002. Arsenic-related Health Problems among Hospital Patients in Southern Bangladesh. *J Health Popul. Nutr*, **20**(3): 198-204.

Morton, W.E. and Caron, G.A. 1989. Encephalopathy: an uncommon manifestation of workplace arsenic poisoning, *Am. J. Ind. Med.* **15**: 1-5.

Ostrosky-Wegman, P., Gonsebatt, M.E., Montero, R., Vega, L., Barba, H., Espinosa, J., Palao, A., Cortinas, C., Garcia-Vargas, G., Del-Razo, L.M., and Cebrian, M. 1991. Lympgocyte proliferation kinetics and genotoxic findings in a pilot study on individuals chronically exposed to arsenic in Maxico. *Mutat Res,* **250:** 477-482.

Paul, A.B. and Kar, D. 2004. Ground water Arsenic in Assam: A Report from Brahmaputra and Barak Velley, Public Health Engineering Department (PHED), Guwhati-6, Assam India, *Environment and Ecology,* CABI Abstract, web: www.cababstractsplus.org/abstracts.

Pouls, M. and Payne, M. 2008. Protect yourself from toxins in our environment, No. 2 - Arsenic, Nutritional Self Defense, 25th annual conference oh heavy metals, University of Michigan. Web: http// www.who.int/nutritionalselfdefense.org.

Rahman, M. 2003. The Bangladesh Arsenic Catastrophe: Clinical Menifestations, *Tropical doctor,* **33**(1): 42-44.

Rahman, M. 2006. International Research on Arsenic Contamination and Health, *J Health Popul. Nutr.,* **24**(2): 123-128.

Rahman, M., Tondel, M., Ahmad, S.A., Chowdhury, I.A., Faruquee, M.H. and Axelson, O. 1999. Hypertension and Arsenic Exposure in Bangladesh, *Hypertension,* **33**: 74-78

Rahman, M., Tondel, M., Ahmad, S.A. and Axelson, O. 1998. Diabetes Mellitus Associated with Arsenic Exposure in Bangladesh, *American Journal of Epidemiology,* **148**(2): 196-203

Rasul, T. 2006. Conclusion and Recommendations, Arsenic Situation in Rajshahi City Area and its Mitigation, *Ph.D. Thesis*, Institute of Environmental Science, University of Rajshahi, Rajshahi, Bangladesh, 124pp.

Reza, S. 2006. *The Essentials of Community Medicine*, 8th ed. 269pp.

Roth, F. 1958. Uber den Bronchialkrebs arsengeschadigter Winzer (On Bronchial carcinoma in arsenic exposed vineyard workers). *Virchows Arch A Pathol Anat Histopathol,* **331**: 119-137.

Smith, A.H., Goyocolea, M., Haque, R. and Biggs, M.L. 1998. Marked Increase in Bladder and Lung Cancer Mortality in a Region of Northern Chille Due to Arsenic in Drinking water, *Am. J of Epidemiol.* **147**(7): 660-699.

SUDP (Sonargaon Upazila Development Profile), Dec.-2004. Quantity of Unions, 83pp.

SYB, 2000. Land Utilization Statistics, 2000 Statistical Year-book of Bangladesh, (21st ed.), 114pp.

Tondel, M., Rahman, M., Magnuson, A., Chowdhury, I.A., Faruquee, M.H. and Ahmad, S.A. 1999. The Relationship of arsenic levels in Drinking water and the Prevalence Rate of Skin Lesions in Bangladesh, *Environ. Health Perspectives*, **107**: 727-729.

Warner, M.L., Moore, L.E., Smith, M.T., Kalman, D.A., Fanning, E. and Smith, A.H. 1994. Increased micronuclei in exfoliated bladder cells of individuals who chronically ingest arsenic contaminated water in Nevada. *Cancer Epidemiol Biomarkers Prevent*, **3**: 583-590.

WHO, 1993. Guidelines for Drinking Water Quality, Geneva, World Health Organization, **1**(2): 188-191.

WHO, 1996. Guidelines for Drinking Water Quality, Geneva, World Health Organization, **2**(2): 156-167.

WHO, 2001. Arsenic in Drinking Water, Fact sheet No 210, Media Centre, World Health Organization , Web: www.who.inf/en/.

Wikipedia, 2009. Homeopathy, Web: http://en.wikipedia/org/wiki/homeopathic_materia_medica.

Wu, M.M., Kuo, T.L., Hwang, Y.H. and Chen, C.J. 1989. Dose-response relation between arsenic concentration in well water and mortality from cancers and vascular disease, *Am J Epidemiol*, **130**: 1123-1132.

Zaldiver, R. 1974. Arsenic contamination of drinking water and foodstuffs causing endemic chronic poisoning, *Beitr Path Bd*, **151**: 384-400.

APPENDICES

Table-1.1: The medicinal substances and their molecular quantity in centesimal scale

Potencies	Medicinal Substances (gm)	Molecular Quantity (in number)
Beginning	1	6.023×10^{23}
1c	1	6.023×10^{23}
2c	1×10^{-2}	6.023×10^{21}
3c	1×10^{-4}	6.023×10^{19}
4c	1×10^{-6}	6.023×10^{17}
5c	1×10^{-8}	6.023×10^{15}
6c	1×10^{-10}	6.023×10^{13}
7c	1×10^{-12}	6.023×10^{11}
8c	1×10^{-14}	6.023×10^{9}
9c	1×10^{-16}	6.023×10^{7}
10c	1×10^{-18}	6.023×10^{5}
11c	1×10^{-20}	6.023×10^{3}
12c	1×10^{-22}	6.023×10
13c	1×10^{-24}	6.023×10^{-2}

Source: Jahangir M, (1998), Molecular Evidence in Homoeopathic Medicine, P-235-241

Table – 2.1 : Union wise population of Sonargaon upazila

Sl.no.	Union/ Pouroshava	Khana	Men	Women	Total
1	Boidderbazar	5246	11500	10950	22450
2	Barodi	5450	14360	12975	27335
3	Jampur	7202	17020	16100	33120
4	Kachpur	5779	15040	13860	28900
5	Mograpara	5500	14430	13200	27630
6	Noaga	3990	10385	9835	20220
7	Pirojpur	5546	14040	13650	27690
8	Sadipur	5336	15400	14500	29900
9	Sombhupura	5235	13440	12935	26375
10	Sonmandi	7715	20130	18600	38730
11	Aminpur (Pouroshava)	3801	11915	11375	23290
	Total	60800	157660	147900	305640

Source: Sonargaon upazila Development profile, (Dec. 2004), Upazila Administration, Sonargaon, Narayangonj, P – 12.

Table-3.1: Family wise Arsenic affected and non-affected peoples of Haria and Ramgonj village, Sonargaon.

Family no.	Total number of family members (Ages)	Number of As Affected members (Age)	Number of As non-affected members
1	6 (55,44,**26**,24.22,20)	1 (26)	5
2	3 (60, 45, 22)	0	3
3	5 (50,**35**,26,**22**,18)	2 (35,22)	3
4	2 (65,35)	0	2
5	6 (**47**,**30**,15,12,9,1.5)	2 (47,30)	4
6	6 (**40**,30,13,10,2,1)	1 (40)	5
7	4 (**35**,30,3,0.5)	1 (35)	3
8	5 (50,35,23,20,16)	0	5
9	3 (35,25,2.5)	0	3
10	6 (50,40,20,18,16,14)	0	6
11	3 (35,25,2)	0	3
12	6 (70,60,30,**28**,25,3)	1 (28)	5
13	4 (45,**35**,**20**,15)	2 (35,20)	2
14	5 (35,30,15,12,2.5)	0	5
15	5 (33,25,8,6,2)	0	5
16	9 (70,35,30,9,6,4,2,2,1)	0	9
17	5 (**68**,**40**,**35**,17,10)	3 (68,40,35)	2
18	4 (**35**,**30**,11,9)	2 (35,30)	2
19	4 (70,45,25,6)	0	4
20	5 (38,30,13,10,8)	0	5
21	4 (27,25,10,6)	0	4
22	6 (57,**45**,27,23,**21**,18)	2 (45,21)	4
22	**106**	**17 (16.04%)**	**89 (83.96%)**

Table-3.2: Family wise arsenic affected and non-affected peoples of Ahmadpur village

Family no.	Total number of Family Members (Ages)	Number of As affected members (age)	Number of As non-affected members
1	4 (**65, 38,** 30, 6)	2 (65, 38)	2
2	3 (**52, 40, 14**)	3 (52, 40, 14)	0
3	4 (**45, 33,** 12, 7)	2 (45, 33)	2
4	5 (**35, 30,** 11, 9, 3)	2 (35, 30)	3
5	4 (**73, 55, 35,** 30)	3 (73, 55, 35)	1
6	5 (**38, 26,** 10, 8, 1.5)	2 (38, 26)	3
7	4 (**40, 32,** 12, 9)	2 (40, 32)	2
8	4 (**37,** 28, 7, 4)	1 (37)	3
9	3 (**30,** 22, 2)	1 (30)	2
10	6 (**62, 55, 27,** 23, 18, 15)	3 (62, 55, 27)	3
11	5 (**55, 50, 22,** 18, 14)	3 (55, 50, 22)	2
12	4 (42, **30,** 6, 2)	1 (30)	3
13	5 (**45, 35,** 8, 6, 2)	2 (45, 35)	3
14	3 (**56, 30,** 12)	2 (56, 30)	1
15	6 (**62, 37, 30,** 10, 7, 4)	3 (62, 37, 30)	3
16	5 (60, **45, 20,** 18, 14)	2 (45, 20)	3
17	5 (**52, 42, 23,** 17, 15)	3 (52, 42, 23)	2
18	4 (**48, 40,** 10, 7)	2 (48, 40)	2
19	3 (**32, 25,** 10)	2 (32, 25)	1
20	5 (**65, 50, 24,** 19, 14)	3 (65,50, 24)	2
21	4 (57, **45, 22,** 16)	2 (45, 22)	2
22	5(**53, 40, 20,** 14, 11)	3 (53, 40, 20)	2
23	4 (**42, 32,** 10, 7)	2 (42, 32)	2
24	3 (**33, 26,** 4)	2 (33, 26)	1
25	3 (**30, 21,** 2)	2 (30, 21)	1
26	4 (**40, 28,** 5, 3)	2 (40, 28)	2
27	4 (**35, 25,** 6, 2)	2 (35, 25)	2
28	6 (**47, 40, 21,** 18, 15, 10)	3 (47, 40, 21)	3
	120	**62 (%)**	**58 ()**

Table-3.3: Particular of arsenic affected patients of Sonargaon upazila

Sl No	Patient's Name	Age	Address	ID No	Occupation	Tube-well use
01	Md. Rafiq F/n: Abul Kashem	26	Harya Badya Para	0512001	Fish seller	Red- many years Green- 5 years
02	Mrs. Rina Akter H/n: M Humayun Kabir	35	,,	0512002	House wife	,,
03	M. Humayun Kabir F/n: Late Noor Mohammad	47	,,	0512003	Fish seller	,,
04	M. Mosharraf Hossain F/n: Mohammad Ali	22	,,	0512004	Fish seller	,,
05	Mrs. Afia Begum H/n: Abdul Ali	35	,,	0512005	House wife	,,
06	M. Hasan F/n: Abdur Rashid	35	,,	0512006	Agriculture	,,
07	Mrs. Sanoara Begum H/n: M. Hasan	30	,,	0512007	House wife	,,
08	M. Munir Hossain F/n: M. Yakub Ali	30	,,	0512008	Fish seller	,,
09	M. Abul Kalam F/n: Abdur Rashid	40	,,	0512009	Labor Painting	,,
10	Mrs. Jhorna Begum H/n: M. Abul Kalam	35	,,	0512010	Service Dairy Firm	,,
11	M. Mahboob Alam F/n: Late Noor Mohammad	40	,,	0512011	Fish seller	,,
12	M. Alamgir F/n: Late Noor Mohammad	35	,,	0512012	Fish seller	,,
13	Mrs. Ayesha Akter H/n: Siraj Mia	40	Ramgonj	0512013	House wife	,,
14	M. Alamgir Hossain F/n: Siraj Mia	21	,,	0512014	Student	,,
15	M. Johurul Islam F/n: Abdul Ali	20	Haria	0512015	Student	,,
16	Mrs. Monoara Begum H/n: Late M Jinnah	40	,,	0512016	House wife Agriculture	,,

Table-3.4: Particulars of the patients (male) of Sujanagar upazila

Sl No	Patient's Name	Age	Address	Occupation	Water in use
1001	M. A. Gafur Sheikh	38	Ahmadpur	Shop Keeper	Rain, Mineral
1002	M. Ismail Sheikh	45	"	Farmer	"
1003	M. Yousuf Sheikh	35	"	Blacksmith	"
1004	Mizanur Rahaman Molla	73	"	Retired	Tube-well
1005	M. Jalal Uddin	25	"	Shop keeper	"
1006	M. Alauddin Molla	38	"	Shop keeper	"
1007	M. Israil Sheikh	40	"	Rickshaw puller	Tank
1008	M. Sirajul	27	"	Little Business	"
1009	M. Islam Sheikh	37	"	Shop keeper	"
1010	M. Mithu	23	"	Work shop	"
1011	M. Abdus Salam	37	"	Work shop	"
1012	M. Borhan Uddin	30	"	Little Bussiness	"
1013	M. Aziz Sheikh	62	"	Fish Seller	"
1014	M. Saman Sheikh	55	"	Farmer	"
1015	M. Sajeeb	14	"	Student	Rain, Mineral
1016	M. Rejaul Islam	50	"	Shop keeper	Tank
1017	M. Nazrul Islam	48	"	Work shop	"
1018	M. Owahidul Haque	53	"	Shop keeper	"
1019	M. Babul	42	"	Shop keeper	"
1020	M. Julhas Molla	33	"	Farmer	"
1021	M. Abbas Molla	30	"	Farmer	"
1022	M. Munir Ahmed	65	"	Little Business	"
1023	M. Akkas Molla	20	"	Little Business	"
1024	M. Shahidul	45	"	Shop keeper	"
1025	M. Sanaul	40	"	Shop keeper	"
1026	M. Alaul	35	"	Shop keeper	"
1027	M. Serajul	21	"	Farmer	"
1028	M. Idris Ali	47	"	Farmer	"
1029	M. Abbas Ali	18	"	Farmar	"
1030	M. Anwar Ali	62	"	Shop keeper	"
1031	M. Alal Molla	22	"	Shop keeper	"
1032	M. Jamal Molla	20	"	Shop keeper	"
1033	M. Karim Molla	24	"	Shop keeper	"
1034	M. Abul Kalam	32	"	Work shop	"

Table-3.5: Particulars of the patient (female) of Sujanagar Upazila

ID No	Patient's Name	Age	Address	Occupation	Water in use
2001	Mrs. Sufia	30	Ahmadpur	Housewife	Rain
2002	Mrs. Hasna	30	"	"	Rain
2003	Mrs. Halima	35	"	"	Tank
2004	Mrs. Rekha	25	"	Teacher	Tank
2005	Mrs. Kamala Begum	35	"	Housewife	Tank
2006	Mrs. Shahnaj Parveen	35	"	"	Tank
2007	Mrs. Manoara Begum	42	"	"	Tank
2008	Mrs. Anoara Begum	40	"	"	Tank
2009	Mrs. Shaheda Begum	65	"	"	Rain, Mineral
2010	Mrs. Saleha Begum	55	"	"	Tank
2011	Mrs. Jesmine	23	"	"	Tank
2012	Mrs. Jayeda	50	"	"	Tank
2013	Mrs. Saheda Begum	48	"	"	Tank
2014	Mrs. Kariman Nesa	40	"	"	Rain, Mineral
2015	Mrs. Bilkis Begum	21	"	"	Tank
2016	Mrs. Salma	25	"	"	"
2017	Mrs. Sajeda	32	"	"	"
2018	Mrs. Kajol	33	"	"	Rain
2019	Mrs. Umme Habiba	56	"	"	"
2020	Mrs. Hasi Begum	23	"	"	Tubewell
2021	Mrs. Jarina Begum	55	"	"	Tubewell
2022	Mrs. Mariam Begum	45	"	"	"
2023	Mrs. Samsun Nahar	26	"	"	"
2024	Mrs. Seerin Begum	22	"	"	Rain, Mineral
2025	Mrs. Khairun Nahar	32	"	"	Tank
2026	Mrs. Meherun Nesa	40	"	"	"
2027	Mrs. Irani Begum	28	"	"	"
2028	Mrs. Ruma Begum	40	"	"	"

FIGURES

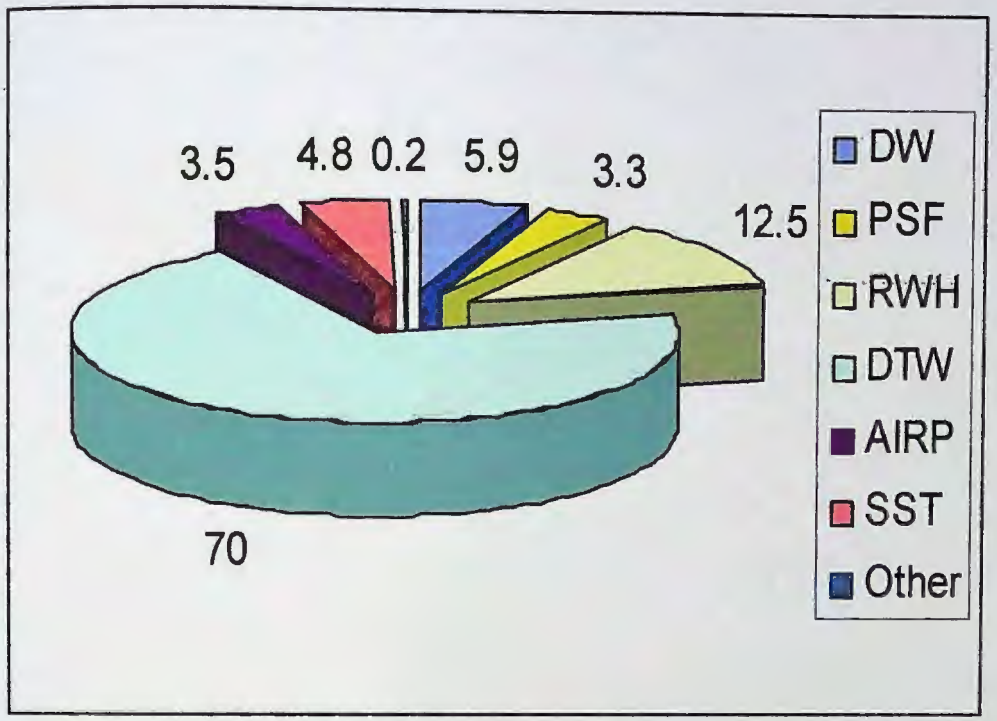


Figure-1.1: Breakdown of options percentage (%) by technology type in arsenic affected upazila

DW = Dug Wells

PSF = Pond Sand Filters

RWH = Rain Water Harvesting

DTW = Deep Tube Wells

AIRP = Arsenic Iron Removal Plants

SST = Shallow Shrouded Tubewells

Others= Piped water supplies, Community based arsenic mitigation technologies, and different types of household level arsenic removal technologies etc.

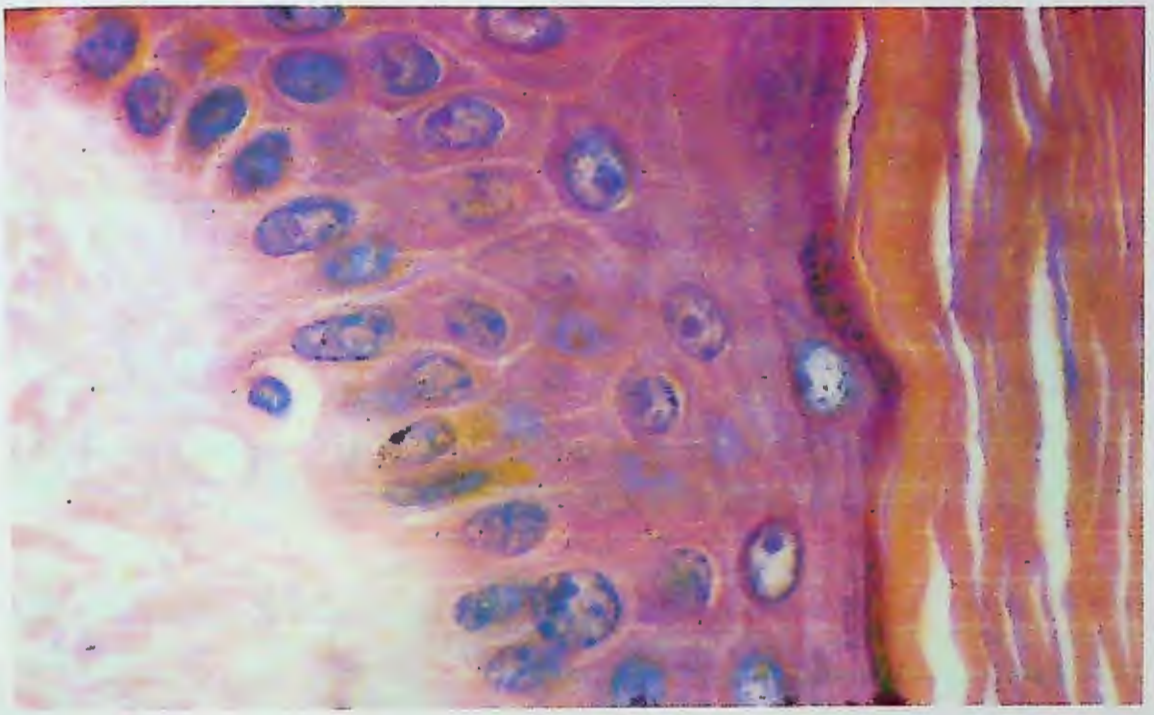


Figure-4. ١: Normal skin: Epidermis, with underlying connective tissue to the left. Magnifi. 0x1375
Big keratin area of the sole of a normal skin,
[**Source:** Rogers, A W, (1983), *Epithelia: the body limits,* Cells and Tissues, P-62]

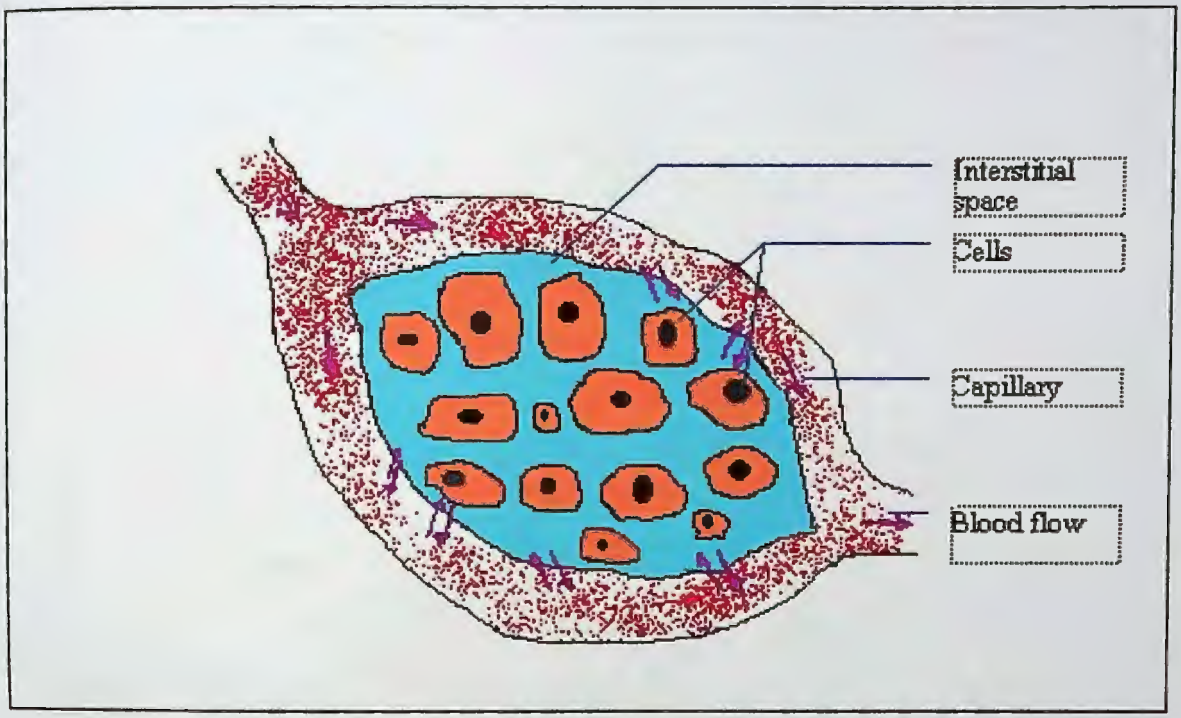


Figure-4 2: Diffusion of fluids through the capillary wall to interstitial spaces and to the cells

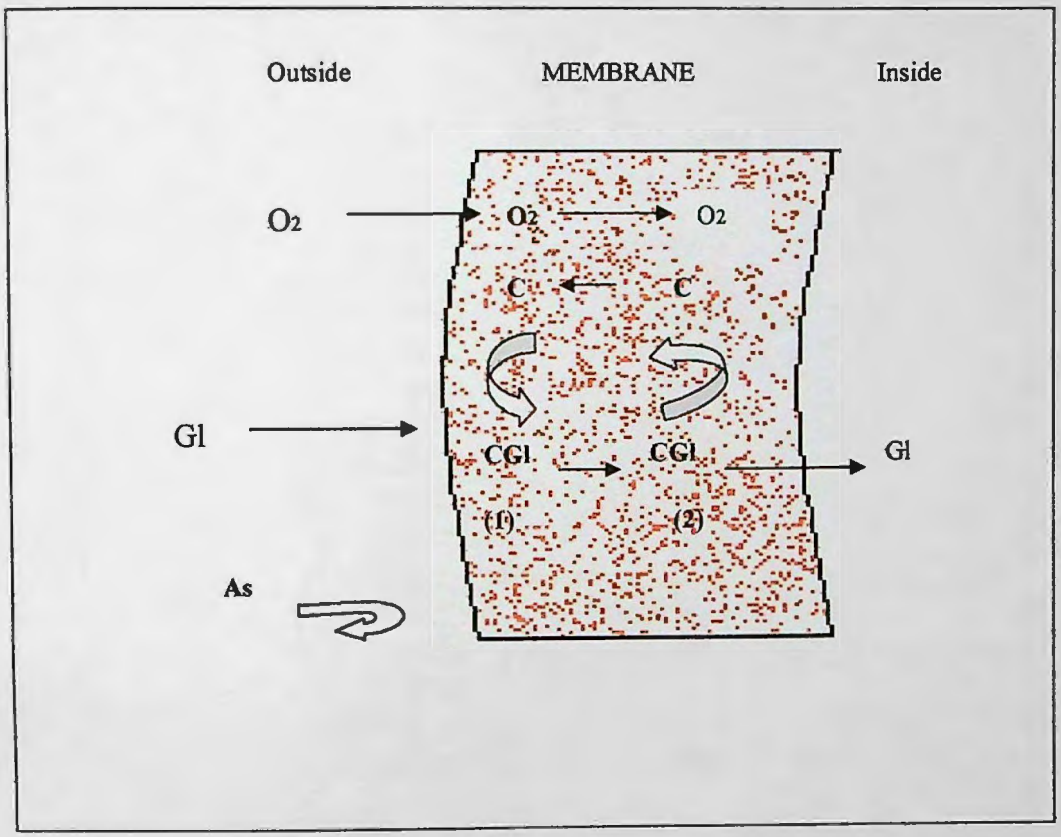


Figure-4.3: Diffusion of substances through the lipid matrix of the membrane

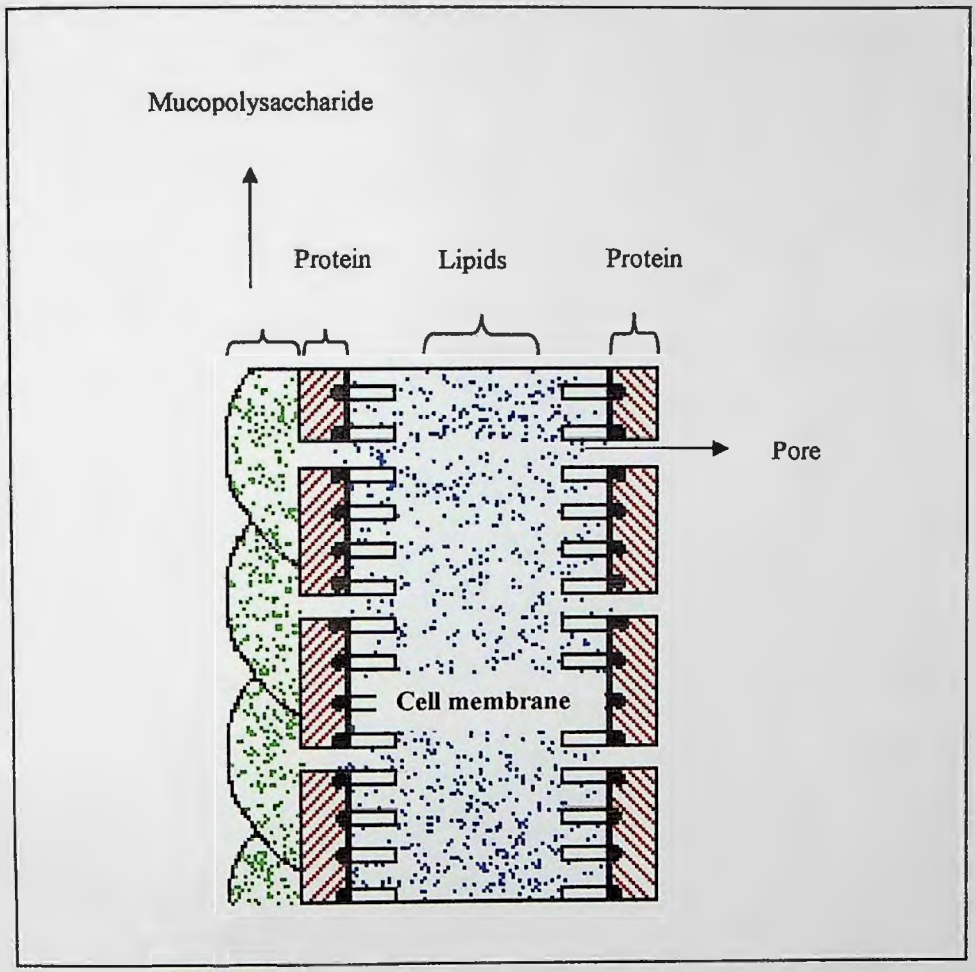


Figure-4.4: Cell membrane of human body.