Arsenic Contamination of Groundwater and Its Clinical Impact on Inhabitants in Central East India

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Abstract

We have worked on the As concentrations present in the groundwater samples in the Rajnandgaon region of India, as well as the clinical assessment of the impact of Arsenicosis on the human population. Out of twenty groundwater samples from eight villages, arsenic was found to be above the permissible limit in eighteen samples. The people in the affected area were found suffering from diffused and spotted melanosis, diffused and nodular keratosis, leucomelanosis, hepatomegaly, and splenomegaly. The hematological parameters, viz. low Hemoglobin, high ESR, and low PCV, with RBC, WBC, and Platelet counts adversely altered and increased Prothrombin time. The change in biochemical parameters reflects the toxic effect of prolonged exposure to arsenic viz. – low Serum Glucose, high LDL, HDL, and TG, decreased Total Protein, Albumin, and Globulin. Total Bilirubin, Creatinine, Urea, and SGPT were at higher levels, but the activity of Alkaline phosphatase was found lower. The study reveals the severe health impact on humans, especially on the liver, kidney, and skin caused by arsenic toxicity in Kaurikasa village and adjoining area.

Introduction

Arsenic is a common element that is found in soil, air, water, and also in living organisms. In terms of abundance, it is ranked 20th in the earth's crust, and seawater, and the 14th most abundant element in the human body, it is the 12th most abundant element. The element is carcinogenic to human beings, and there is very little evidence available for arsenic-induced carcinogenicity in animals.

Essential normal wellsprings of arsenic incorporate deposits of minerals, hydro, geothermal, and volcanic activities. The leading anthropogenic roots of arsenic lie in wood additives, pesticides, mechanical uses, mining, and industrial purification processes. The most harmful type of arsenic is arsine gas, trailed by arsenite and arsenate, which are the species typically found in groundwater. Up to this point, the estimation of total element concentrations was considered to be adequate for clinical and ecological contemplations. Even though the total elemental determination gives valuable and basic logical data for scientific information, the degree of individual species is common of more relevant. For instance, the precise determination of speciation of the toxic species is more significant in the setting of environmental and toxicological parameters as compared to the determination of total elemental concentration. Knowledge about speciation is crucial as the bioavailability and toxicity. Different arsenic compounds have varied toxicity levels. For instance, the inorganic arsenic species, As(III) and As(V) are more potent toxic than their pentavalent methylated counterparts. Arsenic is one of the significant components in huge numbers of sea kelp and the degree of toxicity depends upon the type of arsenic species present. (Environmental Health Criteria; 224 2001).

Arsenic is the most toxic environment-derived metal and the most potent source of arsenic toxicity in humans is its contamination in the drinking water derived from natural underground sources rather than from the agricultural, mining, or smelting processes. (Matschullat et al. 2000).

The working group of the World Health Organization (WHO) characterized Arsenicosis as an "interminable wellbeing condition emerging from lengthened arsenic ingestion (at least more than six months) for over a safe dose, which is generally characterized by trademark skin lesions, with or without affecting the internal organs" (WHO Regional Office for South-East Asia, 2003). The most extreme allowable breaking point of arsenic levels in groundwater according to WHO proposals are 10 µg/L, yet India and Bangladesh have supported <50 µg/L as the acknowledged level in arsenic-defiled zones, where elective hotspots for drinking water are not accessible (Smedley and Kinniburgh 2002). Arsenic exists in the form of a natural contaminant and is widely distributed in the biosphere as arsenite (As^{3+}) and arsenate (As^{5+}) (Hei et al. 2004). Thus Arsenate contamination of drinking water is a grave ecological issue around the globe, and large numbers of populations are at risk (Chowdhury et al. 2000).

The toxicological and human health effects caused by arsenic exposure is known for centuries. Yet, there are still many questions that are unanswered, particularly concerning the mechanisms of action of arsenic and the factors that may affect susceptibility to the damaging effects of these elements and their compounds. The toxicity of inorganic arsenic (As) relies upon its valence state (-3, +3, or +5), and also on the physical and chemical properties of the compound in which it is present. The trivalent (As^{+3}) compounds are usually more toxic than it's pentavalent (As^{+5}) form. More water-soluble compounds are usually more toxic and more likely to have systemic effects than the less soluble compounds and are more likely to cause chronic pulmonary effects if inhaled. In this connection, the arsine gas (AsH_3) is the most toxic inorganic compound. Generally, humans are more sensitive than laboratory animals to the toxic effects of inorganic arsenic. In this connection, the rodents exhibited a critical impact in the form of immuno-suppression and hepato-renal dysfunction, whereas in humans, the primary target areas of arsine gas are the skin, vascular system, and peripheral nervous system. Water-soluble inorganic arsenic compounds are assimilated through the G.I. tract (<90%) and lungs, dispersed predominantly to the liver, kidney, lungs, spleen, aorta, and skin; and excreted chiefly in the urine at a higher rate of 80% in 61 hours following oral dose (U.S. EPA 1984; ATSDR 1989;

Crecelius 1977). The pentavalent arsenic form is reduced to the trivalent form and then it is subjected to methylation in the liver to less toxic methylarsinic acid forms (ATSDR, 1989).

The world's largest arsenic-contaminated area is the Bengal Delta Plain (BDP). In this BDP, Bangladesh and West Bengal in India are the worst affected territories in the world. In 42 districts of Southern Bangladesh and nine adjacent districts of West Bengal, India, 79.0 million and 42.7 million people respectively are manifested to groundwater with arsenic level, way above the WHO's maximum permissible limit of 50µg/L (Choudhury et al. 2000). The source of arsenic is geological in origin, which primarily contaminates the aquifers that provide water for over one million tube wells (Nickson et al. 1998). In some of the tube wells of West Bengal, India, the arsenic contamination is very high (3400 µg/L) (Guha et al. 1998).

Sarkar in 1983 has reported uneven distribution of arsenic in central India. This region is a part of the Lower Proterozoic age formations popularly known as the Bailaidla group and is characterized by typical rock compositions of Phyllitic shales and haematitic quartzes. The is region located at Dalli – Rajhara, 25 km east of Kaurikasa, consists of Banded Haematite or Magnetite quartzes (BHQ) and iron ore mines. The western sector of the area shows broad volcanism, and the rocks of the Nandgaon bunch comprised of lower Bijli Rhylites and Pitepani Volcanics. This volcanic stage was trailed by the emplacement of Dongargarh granite rocks and the intrusion of meta-dolerite dykes and quartz veins. The arsenic-disinfested zone, which shapes the eastern outskirts of the Dongargarh granite batholiths, comprises metabasalt and metarhyolites. Along these lines, the source of arsenic in Kaurikasa territory is topographically influenced by the Nandgaon Orogeny (Pandey et al. 2002).

In Central India, the contamination of arsenic in the groundwater was first reported by Pandey et al. in 1999 in the village Kaurikasa of Chhattisgarh State. In this communication, we report the levels of total arsenic (and speciation) in groundwater in Kaurikasa and six adjoining villages of Ambagarh Chowki (Rajnandgaon) of Chhattisgarh state in Central East India (Fig. 1), and for the first time its toxicological and health impact on the human population.

Materials And Methods

The contaminated area in Chhattisgarh

Rajnandgaon district is situated in the southeastern part of India and Chhattisgarh state. The region lies between 20˚70' and 22˚29' N latitude and 81˚29' and 88˚29' E longitudes. The total area of the region is 6396.28 km^2 out of which 2987 km^2 is forest area. The climate is tropical, and the average rainfall is 1275 mm. Sheonath is the key river of the Rajanadgaon district; it is a feeder of the Mahanadi River, the greatest waterway of Central east India. The river starts at Garhchiroli in Maharashtra crossing over more than 2000 km and streams northeastern way (Pandey et al. 2002).

Almost the entire district depends on tube wells and dug wells for drinking water. The tube wells are fitted with either hand pumps or power pumps. In the Ambagarh Chowki block (20.777689° N, 80.747795° E), there are about 600 hand pumps installed. Overall there are about 1200 pumps/tube wells installed in the district. The contamination of arsenic in the groundwater of Kaurikasa village of Ambagarh Chowki of district Rajnandgaon was reported by Pandey et al. (1999), for the first time.

Sampling of Groundwater

Twenty groundwater test samples were gathered from the hand siphons, tube wells, and open wells from Kaurikasa and connecting villages. Water is drawn for the first 5 min. was disposed of and afterward cleaned plastic sampling bottle was filled to the top with the test samples. The sampling bottles were at first washed with non-ionic detergent followed by an acid wash and then washed with clean lab water finally rinsed with distilled water. Two types of control test samples were collected i.e. duplicate samples and equipment blanks. The reason for the duplicated tests was to ensure the preciseness of samplings and analysis. The duplicate test samples were gathered for screening and laboratory analyses. After filtration of the examples with 0.45 µm membrane filters, a quickly concentrated HNO_3 of pH < 2 was added to preserve the samples for further arsenic analyses. Arsenic was estimated by Varian Cary 300 Atomic Absorption Spectrophotometer utilizing Carry Win UV programming software following the method prescribed by the American Public Health Association (APHA) (Greenberg et al. 1992). For the arsenic speciation examination, the standard arrangement was obtained from Sigma, USA. As (III) was directly analyzed by a generation of hydride at pH 5-7 and afterward As (V) was estimated at PH <1 by utilizing a pre-reductant, *viz*. potassium iodide-ascorbic acid technique (Francesconi et al. 1994).

Symptomatic, hematological, and biochemical tests of the affected population

We carried a survey for the symptomatic study of the human population from the arsenic-contaminated Kaurikasa village of Ambagarh Chowki area of Rajnandagaon district of Chhattisgarh India availing the assistance of a physician. Prior permission from the Institutional Ethics Committee of our institute, Government V.Y.T.PG. Autonomous College, Durg, Chhattisgarh, India, was also availed before conducting the study (IEC/GVYTPGAC/07/DURG, DT 18.10.2014). Blood was collected from the population showing symptoms of Arsenicosis with their kind consent and with the help of the medical technician. Volunteers were told not to take any food in the morning before drawing blood. Blood samples were drawn by sterile disposable syringe and needle and collected in a sterile vial containing EDTA. The hematological examination was carried by assessing the blood Hemoglobin by Sahli's method with the help of a hemometer (Marienfield, Germany). The Total Red Blood Corpuscles (RBC), White Blood Corpuscles (WBC), and Platelet count were determined by the improved Neubauer Hemocytometer method. The Packed Cell Volume (PCV) was estimated by the Macrohematocrite method and while Erythrocyte Sedimentation Rate by the Wintrobe method. The biochemical examination was carried out by photometric determination for serum Glucose. At 505 nm, total cholesterol at 546 nm, Low-Density Lipoprotein, and High-Density Lipoprotein at 340 nm, triglyceride at 546 nm, total Protein at 546 nm, Creatinine at 510 nm, Serum Glutamate Oxaloacetate Transaminase at 340 nm, Serum Glutamate Pyruvate Transaminase at 340 nm and Alkaline Phosphate at 420 nm. The enzymatic kit for the above experiments was obtained from Merck Specialties Private Limited, Mumbai (India). Autoanalyser of Merck (Microlab 300) was used for the above estimations.

Results And Discussion

The extent of underground water contamination

In the present study, the arsenic contamination in water was evaluated using the groundwater samples collected from various adjoining villages of Kaurikasa of Rajnandgaon district in Chhattisgarh. Twenty water samples were collected from eight villages, including Kaurikasa. Table 1 summarises the obtained results. Total arsenic was above the permissible limit in eighteen samples of five villages. The geogenic distribution of arsenic was found variable in the region, which might be due to the variable geochemical system. An examination of the results shows that the water sample from Kaurikasa village recorded (AS III-980±126.08µg/L; AS V- 1220±120.06) was the highest of all samples, which could be due to the greater depth of the borewell compared to the handpump samples from the same village. In the descending order, the arsenic levels (in µg/L) in groundwater samples were Bharritola (ASIII-432±28.6 and 232±15.3; AS V 604±36.06 and 386±24.02), Kuretola (ASIII-212+14.8; AS V- 348±21.06), Mangatola (ASIII-160±9.8; AS V- 230±36.02), and Tumrikasa (ASIII-150±9.6; AS V-216±16.04). Out of three samples collected from Murhetola, two samples were within permissible limits ASIII43±0.06; ASV-78±6.07 µg/L and ASCII-47±4.02; ASV-84±5.18 µg/L, respectively (Table-01). The arsenic level in groundwater samples in Kaurikasa and surrounding villages is far above the limit of 10 µg/L recommended by WHO for drinking water (WHO, 1999).

Symptomatic features of Arsenicosis among the population of Kaurikasa

The survey was carried in association with a physician among the population of Kaurikasa village who confirmed the highest degree of Arsenicosis. The study revealed a prevalence of various symptoms of Arsenicosis among people, as described below:

1. Diffused type of melanosis with darkening of the skin in various parts of the body, especially on the palm.
2. Spotted melanosis of face, hand, limb, back, and chest.
3. Patches of melanosis on the tongue, gum, and lips
4. Diffused and nodular keratosis on the palm and sole.
5. Leucomelanosis on several parts of the body.
6. Hepatomegaly, splenomegaly and ascitis.
7. Non-pitting swelling.
8. General weakness, sleeplessness, breathlessness, and indigestion are common complaints from the people in the affected area (Fig 1a, b, c & d).

Hematological alterations among the population suffering from Arsenicosis

Based on prevailing symptoms, twenty people (12–62 years) were selected for hematological and biochemical evaluation from Kaurikasa village. The Hemoglobin percentage was found below the normal range in nineteen samples and RBC count was found low in thirteen samples. WBC was lower in eleven samples, but ESR was found high (>20 mm/h) in sixteen samples in comparison to their normal range. PCV and platelet count were lower in twelve than their standard limit, but prothrombin time was elevated in eighteen samples relative to their standard limit. (Fig. -3)

Belon et al. (2006) reported a low count of RBC, WBC, and low estimation of PCV,
Hemoglobin, but a high level of ESR among the population in the groundwater arsenic-contaminated village of Ghetugachhi (Chakdha, West Bengal, India). Soignet et al. in 2001 have also observed the mitostatic effect of arsenic trioxide, especially for WBC in patients with relapsed acute promyelocytic leukemia (APL). Winski et al. in 1997 have established hemolysis and destruction in Hemoglobin in human blood exposed to arsine, and they found an alteration in the volume control of cells due to leakage of potassium and influx of sodium which increased hematocrit. Blair et al. (1990) investigated the impact of arsine gas exposure on male and female mice for 5, 15, and 90 days. They reported moderate hemolytic anemia, evident by a decrease in erythrocyte counts, hematocrits, and hemoglobin concentration. Tripathi et al. (2003) have reported a significant fall in hemoglobin concentration and PCV along with a decrease in total erythrocyte count in a *Clarias batrachus* after exposure to sodium arsenite and observed the disturbed hemopoiesis, erythrocyte membrane disruption, impaired iron uptake by RBC, and hemolysis due to arsenic toxicity. The Agency of Toxic substances and Disease Registry (ATSDR), Atlanta in 2000, in a case study of 35 years human of chronic Arsenicosis reported the occurrence of macrocytic anemia, bone marrow depression, a low percentage of hematocrit, leucopenia, and thrombocytopenia. Wilkinson et al. (1975) also reported thrombocytopenia in the human population suffered from chronic exposure to arsenic. In China, the use of arsenic for the treatment of leukemia is an age-old practice. In an incident in China in 2004, over a hundred workers ingested extensive levels of arsenic due to an accident caused by pipeline leakage in a copper smelting factory. After toxicological evaluation, Yuanyuan Xu et al. found 69.2% of sufferers were Leucopenic within 15 days. Similarly, in Taiwan, 926 workers of semiconductor plants were investigated by Luo et al. (1995) of which 30% of male photolithography workers were found Leucopenic than 5-6% in control male workers. Our findings for the twenty people exposed to arsenic-contaminated groundwater are almost in concurrence with the earlier reports from different parts of the world.

Biochemical alterations among the population suffering from Arsenicosis

The biochemical alterations noticed in the twenty blood samples were found significant. The serum glucose was below the normal range in ten samples (Fig. 4). Among lipid profiles the total cholesterol, LDL and HDL were
found above the normal range in eight, eighteen, and thirteen samples respectively, but the TG was above the normal range in all twenty samples (Fig. 5). Protein metabolism was also disturbed. The
total protein, albumin, and globulin levels were found below the normal range in nineteen, twenty, and sixteen samples, respectively (Fig. 6). The total bilirubin and creatinine were higher than the normal range in fifteen and sixteen samples, respectively (Fig. 7). The serum urea level was above the standard limit in all twenty samples (Fig. 8). SGOT was within the normal limit in all twenty samples, but SGPT was above the normal range in all twenty samples. Alkaline phosphatase was below the normal range in all twenty samples (Fig. 9). Pal et al. in 2004 have reported a hypoglycemic effect in male Wistar rats, similar to our findings. Mukherjee et al. (2004), Navas-Acien et al. (2006), and Zierold et al. (2002) have also reported hyperglycemia under arsenic poisoning. Acute arsenic toxicity, including its effect on glucose metabolism, is generally associated with its reactivity towards the thiol (SH) group (Aphoshian 1989; HRC 1999). Under acute conditions, arsenite inhibits the enzyme pyruvate and alpha-ketoglutarate dehydrogenase (Aposphian 1989) which is necessary for glucogenesis and glycolysis processes. Krebs in early 1993 also described that arsenic interferes with pyruvic acid metabolism. Arsenate, on the other hand, can mimic phosphate during energy transfer pathways of phosphorylation and it is also involved in oxidative phosphorylation uncoupling (Kennedy and Lahninger 1949). However, it is unlikely that these toxic effects of acute arsenic exposure take
place as a result of chronic exposure to environmentally relevant doses (Tseng 2004). Arsenic could influence alteration in sugar metabolism by other mechanisms, including oxidative stress, inflammation, apoptosis, and the nonspecific mechanism that has been involved in the pathogenesis of type-2 diabetes. Arsenic exposure can augment the production of reactive oxygen species (Chen et al. 1998, Tseng 2004, Wang et al. 1996), impede the activity of crucial antioxidant enzymes such as glutathione reductase, glutathione-S-transferase, glutathione peroxidase, and glucose-6-phosphate dehydrogenases (Maiti and Chatterjee 2000; Santra et al. 2000) and also can induce lipid peroxidation (Santra et al. 2000). In studies from Taiwan, the increase in arsenic levels in human blood is correlated with the increased level of reactive oxygen species and with increasing levels of antioxidant capacity of plasma (Wu et al. 2001). Overall the experimental and epidemiological evidence at present is insufficient to confirm the arsenic-induced hyperglycemia or hypoglycemia.

In the present study, we found raised levels of total cholesterol, HDL, LDL, and TG in a majority of samples. In a study conducted in Bangladesh in the arsenic-contaminated region, overall 45% of residents were reported with a low range of total cholesterol, 54% with low range of HDL, and 20% with low range of LDL but triglyceride was above the normal range of 47% population (Nabi et al. 2005). Several studies suggest no significant changes in lipid profile due to arsenic toxicity (Want et al. 2006; Petia et al. in 2003; Mahaffey et al. 1977). Protein metabolism was also found disturbed in the present study (Fig. 6). In a study where arsenic contamination in drinking water was established, the total protein in serum was found higher in 43% population (Nabi et al. 2005). In an experiment
conducted on the pigs, the total protein concentration was found to decrease than the normal range (Wang et al. 2006). The oxidative stress ensuing from arsenic toxicity causes damage to sulfur-containing enzymes and other proteins. This phenomenon ends up in the form of inactivation of protein, defective cross-linkages, and protein

denaturation (Serhan et al. 1991). Serum albumin and a small fraction of globulin are synthesized in the liver, and the serum protein is afflicted both quantitatively and qualitatively in liver disorder. In any disease-causing hepato-cellular damage, the concentration of serum albumin decreases. The dynamically changing levels of serum albumin, therefore, are a valuable indicator of severity, progress, and prognosis in hepatic diseases. In the present study, liver cell damage is attributed due to arsenic toxicity.

The elevated levels of bilirubin were observed in the current study. In plasma, bilirubin is found as indirect reacting bilirubin which is insoluble in water. The direct reacting esterified bilirubin is water-soluble. At the end of the life, span erythrocytes are destroyed in the reticuloendothelial system and liberate hemoglobin. The globulin is separated from Hemoglobin, and the porphyrin ring is opened. The released iron part goes into the iron store and may be used further for hemoglobin synthesis. Green color biliverdin forms first from the non-iron-containing residue of Hemoglobin (i.e. Protoporthyrin). Biliverdin gets reduced to yellow-colored bilirubin. Generally, total serum bilirubin is found to increase in case of hepato-cellular damages (toxic hepatopathy neoplasm, etc.), obstructions in intra and extrahepatic biliary tract, intravascular, and extravascular hemolysis processes. Excessive elevation of direct bilirubin is seen during cholestasis and late in the course of chronic liver diseases. In the present finding, an elevated level of bilirubin in 16 samples out of 20 suggests liver toxicity among the population due to Arsenicosis.

The elevated level of SGPT in all 20 samples also suggests liver cell damage. Steven et al. in 2001 and Wang et al. in 2006 have also reported an increased concentration of SGPT under Arsenicosis. While serum urea was elevated in all the 20 samples, the creatinine level was high in 16 samples. Belon et al. also reported a high concentration of serum urea and creatinine in a study on 20 males and 19 females affected by groundwater arsenicosis in village Dasdiya of West Bengal, India. Elevated levels of urea are found in pre-renal, renal, and post-renal conditions associated with high serum creatinine in any renal functional impairment. Thus the current finding of both high urea and creatinine levels indicates renal dysfunction among the population due to Arsenicosis.

In the present study, alkaline phosphatase activity was found lowered in all twenty samples. Mahaffey et al. (1977) and Mehranjani et al. (2006), have also reported the decreased activity of alkaline phosphatase under Arsenicosis, but Mazumdar et al. (1998) found increased activity in 51.3% of patients suffering from Arsenicosis in West Bengal, India. Thus our results endorse the findings of Mehranjani et al., that under the influence of Arsenicosis, the activity of alkaline phosphatase is impaired.

Table– 1- Distribution of arsenic in Kaurikasa and nearby villages of Rajnandgaon district of Chhattisgarh, India (Number of sample replicates = 10)

Sample Number	Village	Source	Depth of water tapping points (Ft.)	Estimated Concentration of As III (µg/L) [Mean±SD]	Estimated Concentration of As V (µg/L) [Mean±SD]
1.	Murhetola	Hand Pump	280	57.00±4.03	106.00±8.04
2.	Murhetola	Hand Pump	260	43.00±3.06	78.00±6.07
3.	Murhetola	Hand Pump	262	47.00±4.02	84.00±5.18
4.	Devasur	Hand Pump	268	55.00±4.06	92.00±6.06
5.	Devasur	Hand Pump	276	55.00±4.06	88.00±6.04
6.	Tumrikala	Hand Pump	262	62.00±4.8	98.00±7.06
7.	Tumrikala	Hand Pump	350	150.00±9.6	216.00±16.04
8.	Kuretola	Hand Pump	364	212.00±14.8	348.00±21.06
9.	Bharritola	Hand Pump	372	232.00±15.3	386.00±24.02
10.	Bharritola	Hand Pump	400	432.00±28.6	604.00±36.06
11.	Kaurikasa	Borewell	480	980.00±126.08	1220.00±120.06
12.	Kaurikasa	Hand Pump	275	55.00±4.07	86.00±6.04
13.	Kaurikasa	Hand Pump	340	73.00±18.03	124.00±16.08
14.	Dongargaon	Well	320	54.00±4.05	96.00±8.02
15.	Dongargaon	Hand Pump	300	59.00±4.6	102.00±8.08
16.	Dongargaon	Hand Pump	270	54.00±4.04	88.00±6.06
17.	Dongargaon	Hand Pump	262	57.00±4.04	84.00±5.06
18.	Dongargaon	Hand Pump	260	54.00±4.03	80.00±4.08
19.	Dongargaon	Hand Pump	275	62.00±4.6	92.00±6.02

Conclusion

Both the symptomatic and clinical features of Arsenicosis were found apparent among the population of the study area due to chronic exposure to arsenic through drinking water. All major hematological parameters show people were adversely affected due to Arsenicosis, especially the destruction of RBC and Platelet cells. Similarly, the biochemical evaluation has established liver dysfunction, renal dysfunction, and abnormal glucose metabolism among the population. Thus in the residents in the Ambagarh Chowki area are suffering from serious health problems due to geogenic Arsenicosis through contaminated groundwater. Although the Government has already taken initiatives to ensure the supply of arsenic-free water to the population still, the residual arsenic is posing a severe health problem.

Declarations

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AUTHOR CONTRIBUTION:

AK, Conceptualized and supervised work; ST & NM, have performed experiments and prepared the manuscript; SBJ, suggested and reviewed the manuscript.

CONFLICT OF INTEREST:

The authors declare that they have no conflict of interest.

DATA AVAILABILITY STATEMENTS:

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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Figures

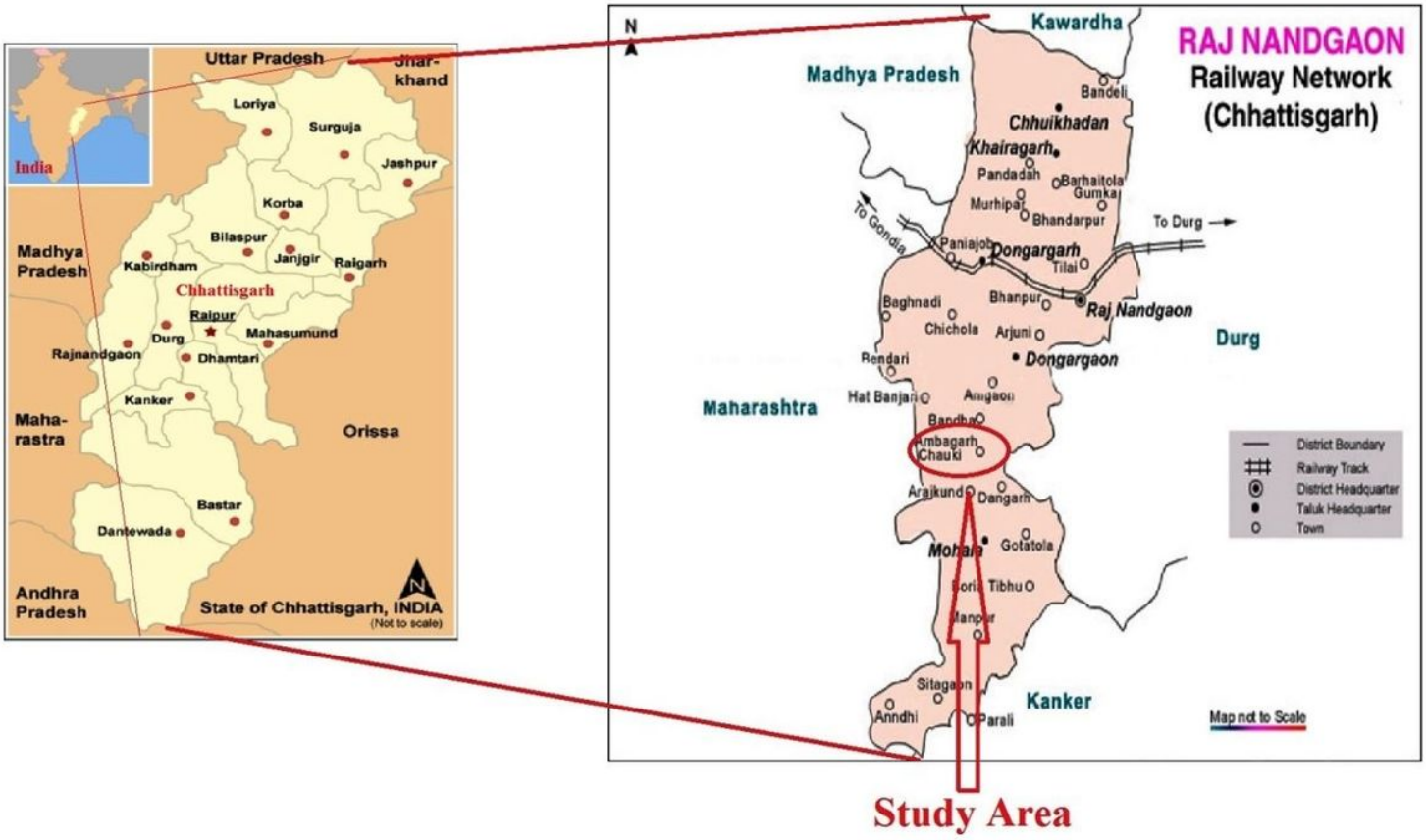

Figure 1

Figure Showing the study area of Ambgarh Chowki of Chhattisgarh State, India.

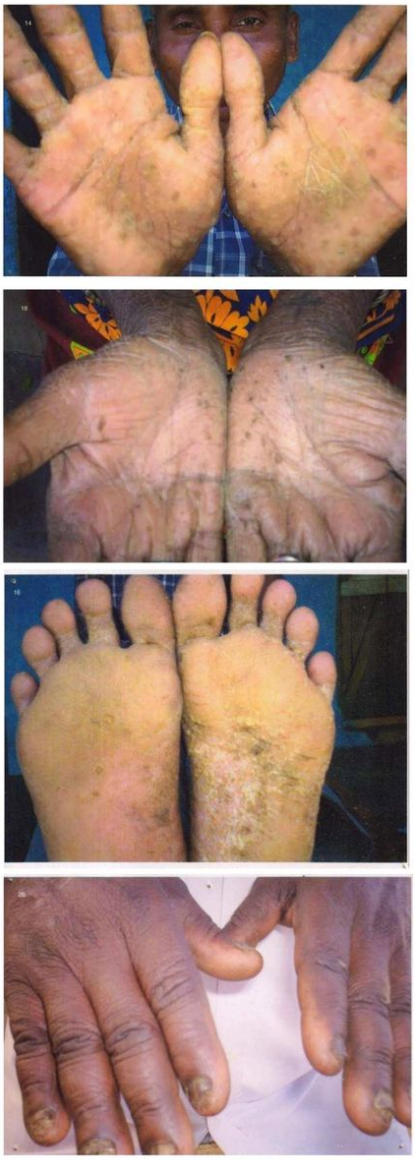

Figure 2

a. Showing both Hypo and Hypermelanosis with keratosis in a person suffering from Arsenicosis. b Showing Leucomelanosis in a person suffering from Arsenicosis. c. Showing Diffused and Nodular Kerstosis in a person suffering from Arsenicosis. d. Showing Nail degeneration in a person suffering from Arsenicosis.

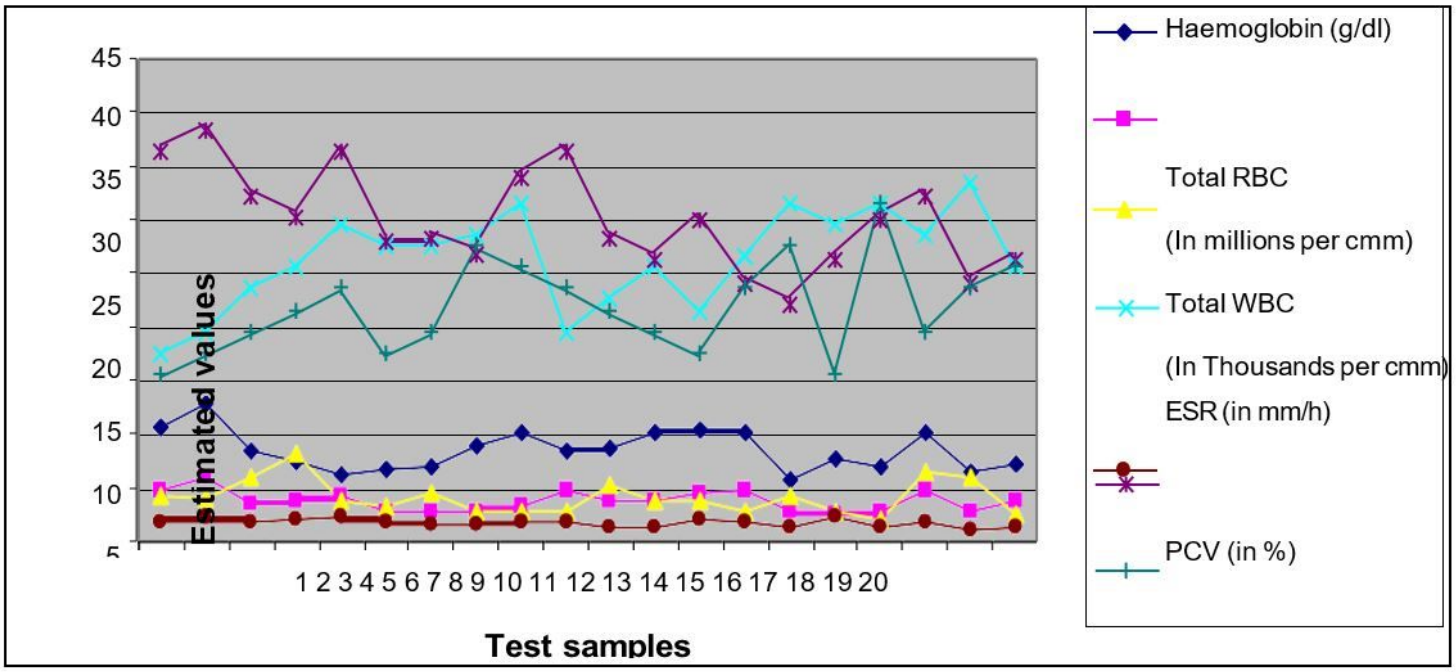

Figure 3

Normal range: HB- = 13 -18 g/dl, = 12-16 g/dl, Total RBC- = 4.5-6.0 m/cmm, = 4.0-4.5 m/cmm, Total WBC = 4-10 103/cmm, ESR- = 0-15 mm/h, = 0-20 mm/h, PCV = 42-52 % & = 36-48%, Platelets = 250-500 103/cmm, Prothrombin time =14-16 sec. Showing alterations in hematological parameters of the human population affected by Arsenicosis.

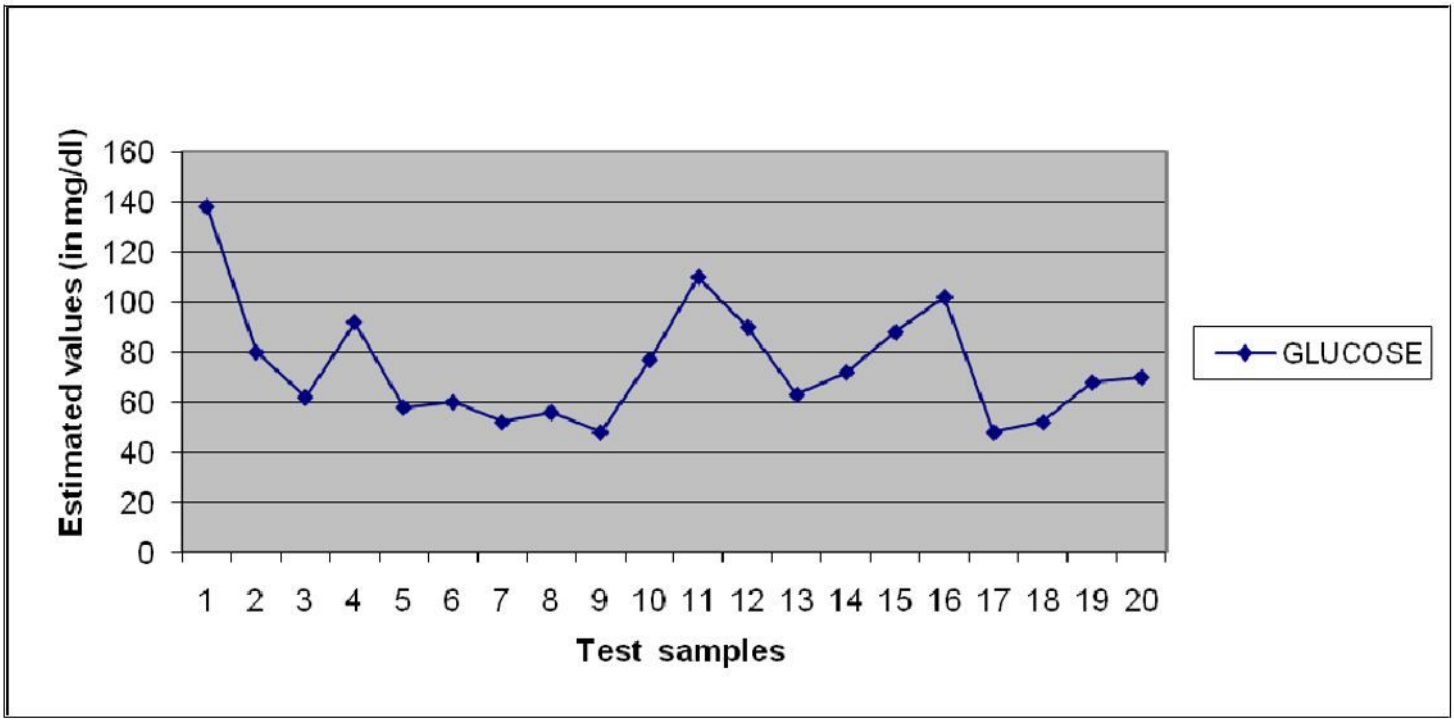

Figure 4

Normal range – Glucose-70-100 mg/dl Showing alterations in glucose level of the human population affected by Arsenicosis.

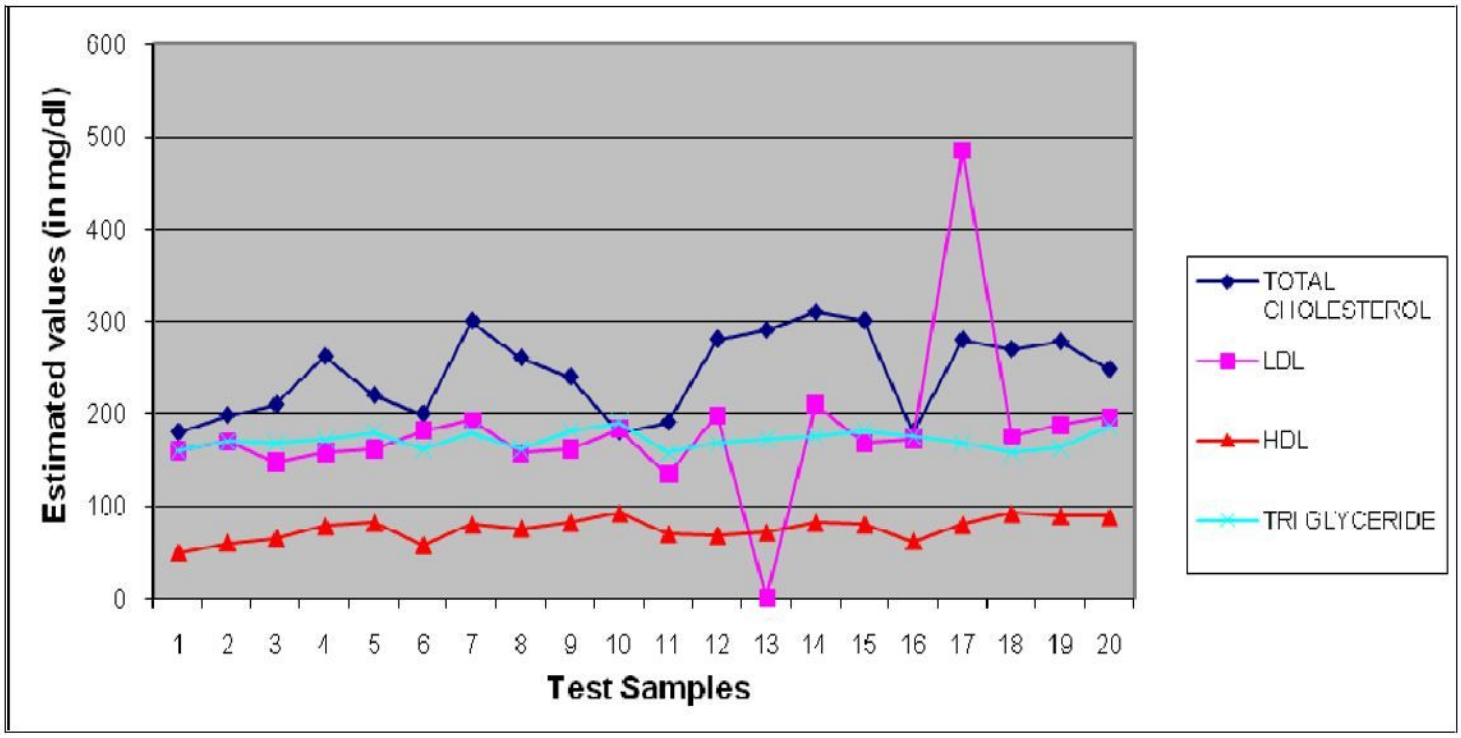

Figure 5

Normal range- Total Cholesterol- 150-225mg/dl, HDL - 30-60mg/dl, LDL – 80-150mg/dl , Triglyceride- 75-150mg/dl. Showing alterations in the lipid profile of the human population affected by Arsenicosis.

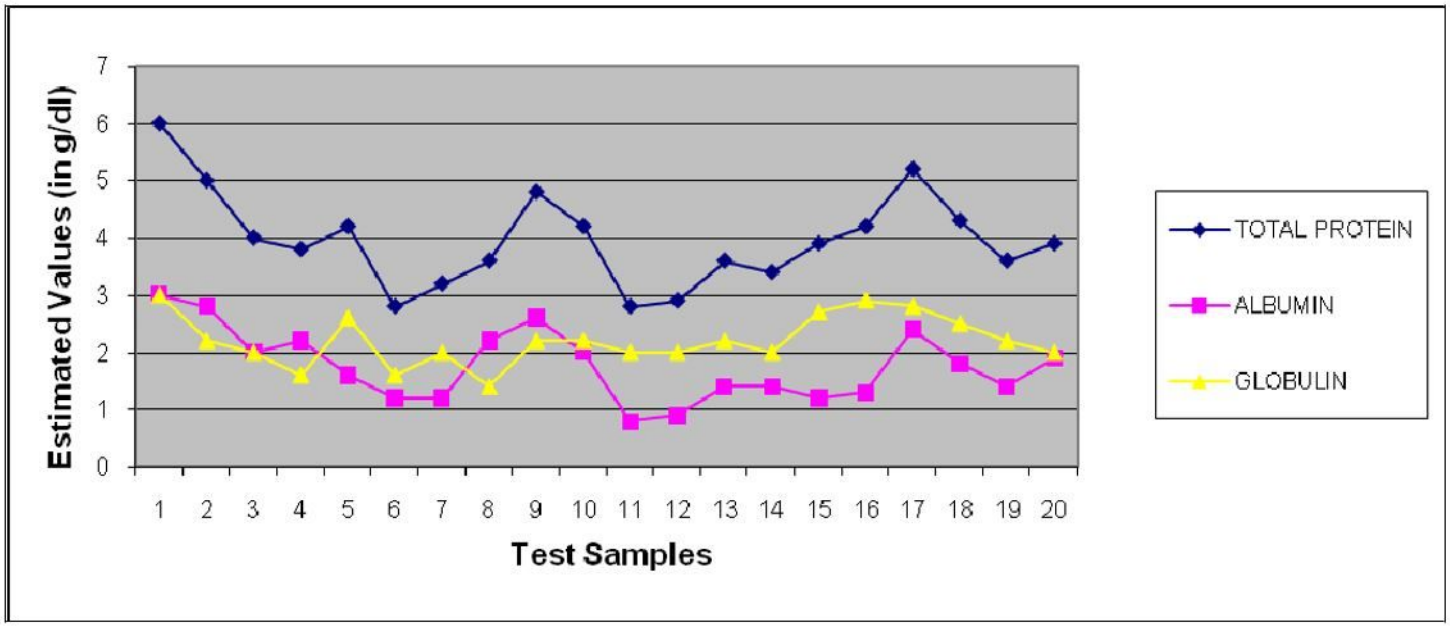

Figure 6

Normal range- Total protein- 6-8g/dl, Albumin- 3.5-5 g/dl Gloobulin- 2.5-3.5 g/dl. Showing alterations in the protein profile of the human population affected by Arsenicosis.

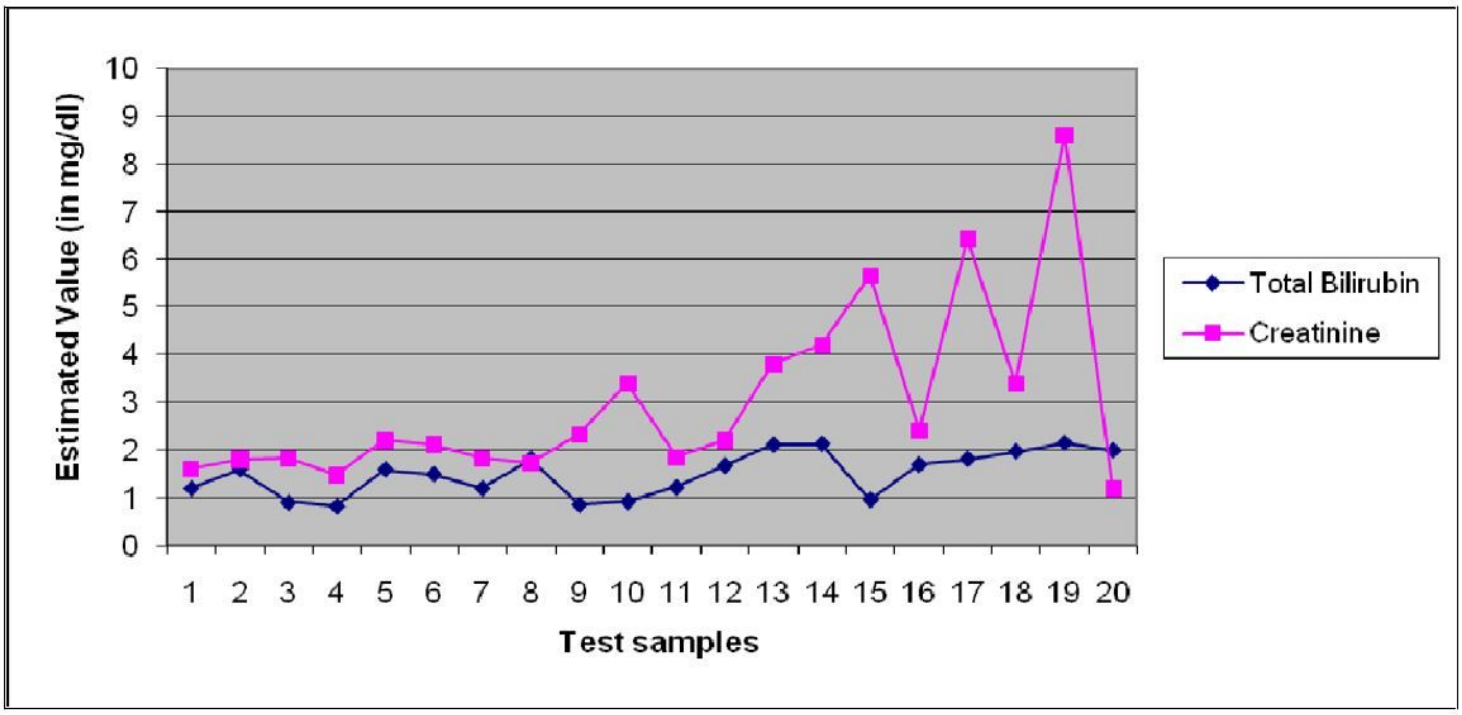

Figure 7

Normal range – Total Bilirubin- 0.2-1 mg/dl , Creatinine- 0.5-1.5 mg/dl. Showing alterations in creatinine and total bilirubin of the human population affected by Arsenicosis.

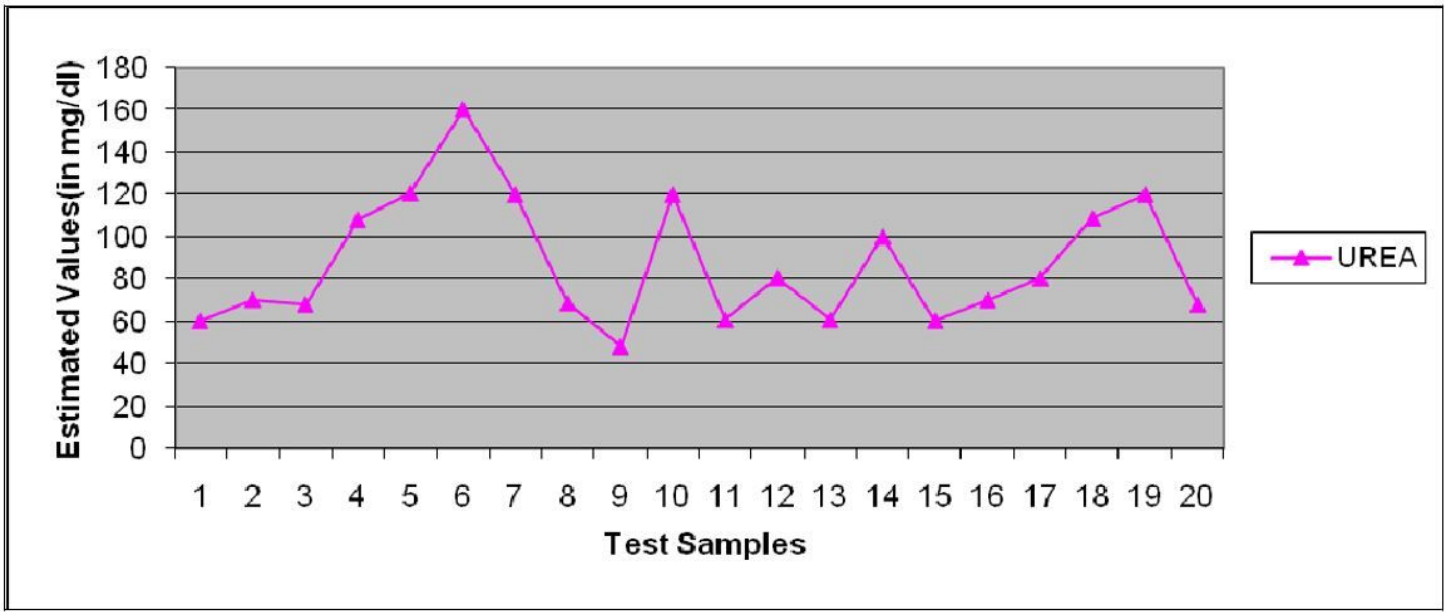

Figure 8

Normal range – Urea – 15-40 mg/dl. Showing alterations in the urea level of the human population affected by Arsenicosis.

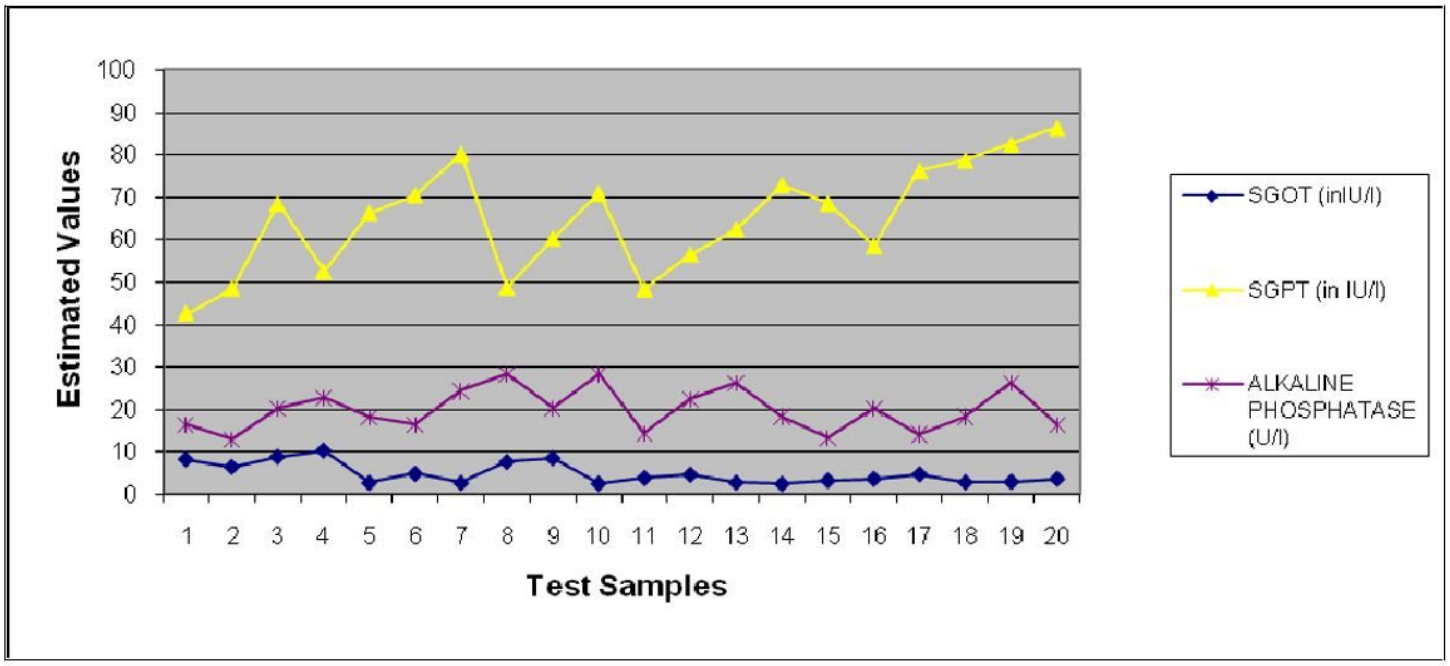

Figure 9

Normal range – SGOT- 5-45 IU/l , SGPT- 5-40 IU/l , Alkaline Phosphatase- 3-13 U/l Showing alterations in the lipid profile of the human population affected by Arsenicosis.