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WATER WITH HIGH TDS, RESULTANT CKD (CHRONIC KIDNEY DISORDER) AND IMPOSED NUTRITIONAL AND HEALTH PROBLEMS IN CHHATTISGARH

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Abstract:

Quality of water significantly affects human body, in Chhattisgarh due to presence of abundance quantity of dolomite in soil , the alkalinity of water as well as the calcium content and total dissolved solids is extremely high in comparison to the recommend contents .This water pollution precipitates problem of renal stones (Renal lithiasis) .Thus the state is known as Stone Belt Area. CKD –chronic kidney disease due to renal stones is like a community problem here. Ultimately dialysis is the life saving medical procedure, but repeated dialysis causes hyper-phosphataemia, this is etiological cause of repeated infections and cardiac problems. The study was done on the subjects who resides in the area where the TDS and alkalinity parameters were high, the victims of CKD of this area were randomly selected and their health parameters along with biochemical parameters were collected , the induced problems due to hardness of water and repeated dialysis were assessed and it is concluded that the related health problems imposed dyslipidemias and hyper phosphataemia that are major risk for cardiac health.

Keywords: *quality of water, renal lithiasis, CKD, dialysis*

Introduction:

Water has been vital to man and nature since the beginning of time. The planet earth is abundantly rich in water , which constitute 71.11 of its surface . Besides , being an essential ingredient of animal and plant life . Water made 65% of human body. Out of total water consumed by human

beings , more than 50% of it is consumed for industrial activity and only a small proportion is used for drinking purposes. Ground water is the major source of drinking water in both urban and rural areas .Under ground water contains high amount of ions and salts etc, which are soluble in water and directly affect the quality of water. Only 1% pure water available in pure form, that is present in oceans and iceberg . Rural area people mostly depends on hand pumps for all uses of water- drinking and irrigation. The three basic needs in human life are food, air and water5-6. Good Quality of Drinking water is very necessary for improving the life of people and to prevent from diseases. Water crisis due to Industrialization, Urbanization, Developmental activities and population explosion is serious community problem now a days. Ground water gets polluted due to increased human population, agriculture runoff, domestic sewage, industrial effluents and other activities. Quality of water directly affects the health of human beings and causes various types of diseases. We collected the data regarding the quality of water in the state. The water sample was collected in February 2013, from Rural Area of Bhilai, Chhattisgarh India. Water samples were collected in plastic bottle ,immediately transported to the laboratory of engineering college and avoid any changes in Water quality Parameters. All parameters determined using standard methods. pH of water Sample measured by pH meter using standard solutions; Value of Total Hardness, Temporary Hardness, Permanent Hardness, Calcium and Magnesium of water Sample determined by EDTA method; Total Alkalinity and Value Bicarbonate determined by Acid-Base titration method; Turbidity of water sample measured by Turbidity meter; TDS (Total Dissolved Solid) measured by TDS meter; Dissolved Oxygen determine by Winkler method; BOD also analyzed using BOD incubator.

Water Quality Parameters of ground water sample from study area, and comparison with Permissible values as per IS:10500-1991 S. No. Water Quality Parameters Ground Water Sample Permissible Values (Requirement desirable limit) as per IS: 10500-1991.

Water Quality Parameters	Ground Water Sample	Permissible Values(Requirement desirable limit) as per IS: 10500-1991
1 Taste	Normal	Normal
2 Colour	Transparent	Transparent
3 Temperature	29 °C -	
4 Odour	Unobjectionable	Unobjectionable
5 pH	8.17	6.5 to 8.5
6 Total Hardness	450 mg/l	300 mg/l
7 Turbidity	3NTU	5NTU
8 Calcium Hardness	360 mg/l -	--
9 Magnesium Hardness	90 mg/l -	--
10 Chloride	213 mg/l	250 mg/l
11 Temporary Hardness	180 mg/l -	--
12 Permanent Hardness -	270 mg/l	--
13 Total Alkalinity	400 mg/l	200 mg/l
14 Bicarbonate Alkalinity	400 mg/l -	
15 TDS	1170 ppm	500 ppm
16 DO	24.8 mg/l -	
17 Calcium	144 mg/l	75 mg/l
18 Magnesium	21.87 mg/l	30 mg/l
19 BOD	4.32 mg/l	5 mg/l

The observed value of water quality parameters from Mungali has been mentioned in the given table and the value of water quality parameters compared with standard permissible values as per IS: 10500-1991. The value of total hardness of water was found to be very high 450 mg/l, and causes of human health problems. Due to High value of Hardness various type of disease may be occurred some of these are cardio-vascular disease, but most prominently Kidney stones. The value of pH was found to be 8.17. Turbidity was found to be 3 NTU, Value of TDS also found to be very high 1170 mg/l and causes of some disease –kidney stones and renal failures, coronary heart disease, arteriosclerotic heart disease and cardiovascular disease. Dissolved Oxygen found to be 24.80 mg/l. value of chloride found to be 213 mg/l. Total Alkalinity was found to be 400 mg/l and causes digestion problem and acidification is needed. The value of calcium and magnesium also found to be

high as compare to Indian standard specification for drinking purpose. And BOD found to be 4.32 mg/l.

As Calcium Carbonate and Calcium Bicarbonate (Dolomite) is present in ground water of Chattisgarh in abundance , so kidney stones containing Ca compounds are common health problem here. Earlier considered to be a health problem only in developed countries, 4 out of 5 chronic disease deaths now occur in low and middle-income countries. According to World Health Organization (WHO) Global Burden of Disease Project, disease of the kidney and urinary tract contribute to approximately 850,000 deaths every year of which Chronic Kidney Disease (CKD) is the 12th leading cause of death and 17th leading cause of disability in the world.CKD is associated with increased cardiovascular mortality and a loss of disability-adjusted life years· The global increase in CKD is being driven by the global increase in of diabetes mellitus, hypertension, obesity, and aging.

Recent research suggests that 1 in 10 of the population may have CKD, but it is less common in young adults, being present in 1 in 50 people. In those aged over 75 years, CKD is present in 1 out of 2 people. However, many of the elderly people with CKD may not have 'diseased' kidneys, but have normal ageing of their kidneys. Although severe kidney failure will not occur with normal ageing of the kidneys, there is an increased chance of high blood pressure and heart disease or stroke, so that medical checks will be helpful. Renal statistics, especially the rise in number of CKD and renal failure cases in India has taken an upward surge.

Approximate total burden of CKD is 800 per million populations (pmp). It has been reported that Diabetes mellitus as the cause of CKD was found in 31.2-41% of patients in India. Chattisgarh is Stone Belt area and later-diagnosed renal stones are causes of kidney damage and CKD here. According to state health statistics every day 2-5 deaths are common due to renal failure , absence of dialysis , no renal transplants . Inadequate dialysis- (under dialysis) promote cardiac death by mechanisms as hyperphosphataemia , hypervolaemia, poor control of blood pressure, malnutrition etc. Approximately 60-70% of dietary phosphate, 1000-1500 mg/day, is absorbed in the small intestine. Although vitamin D can enhance

the absorption, especially under conditions of dietary phosphate depletion, intestinal phosphate absorption does not require the presence of active vitamin D. Specifically, high serum phosphate and high dietary phosphate intake do not significantly impair intestinal uptake. The movement of phosphate in and out of bone, the reservoir containing most of the total body phosphate, is generally balanced. Renal excretion of excess dietary phosphate intake ensures maintenance of phosphate homeostasis, maintaining serum phosphate at a level of approximately 3-4 mg/dL in the serum. Phosphate shift from intracellular to extracellular space during dialysis-This pathogenic mechanism alone is an uncommon cause of hyperphosphatemia, but it can exacerbate hyperphosphatemia produced by impaired renal excretion. Clinical situations in which a shift to extracellular space is the major cause of hyperphosphatemia include rhabdomyolysis and tumor lysis. Rarely, extracellular shifts of phosphate occur with insulin deficiency or acute acidosis.

Squeal of hyperphosphatemia- By precipitating calcium, decreasing vitamin D production, and interfering with PTH-mediated bone resorption, hyperphosphatemia can cause life threatening hypocalcaemia; Prolonged hyperphosphatemia promotes soft-tissue calcification, in which an abnormal deposition of calcium phosphate occurs in previously healthy connective tissues, such as cardiac valves, and in solid organs, such as muscles.

Excess free serum phosphate is taken up into vascular smooth muscle via a type 3 sodium-phosphate cotransporter. The increased cellular phosphate activates a gene, *CBFA1*, that triggers a transformation in the vascular cell, causing smooth muscle cells to engage in osteogenesis. Vascular walls become calcified and arteriosclerotic, leading to increased systolic blood pressure, widened pulse pressure, and subsequent left ventricular hypertrophy. Colonoscopy is also creating Problem due to Phosphorous supplementations as Sodium Phosphate before colonoscopy for preparing medium .

The magnitude of the risk is illustrated by the fact that hyperphosphataemic compared with normo-phosphataemic patients have a 52% higher risk of death from coronary artery disease, a 26% higher risk of

sudden death, a 34% higher risk from other cardiac causes and a 39% higher risk of death from cerebro-vascular accidents .

Approximately 60-70% of dietary phosphate, 1000-1500 mg/day, is absorbed in the small intestine. Although vitamin D can enhance the absorption, especially under conditions of dietary phosphate depletion, intestinal phosphate absorption does not require the presence of active vitamin D. Specifically, high serum phosphate and high dietary phosphate intake do not significantly impair intestinal uptake. The movement of phosphate in and out of bone, the reservoir containing most of the total body phosphate, is generally balanced. Renal excretion of excess dietary phosphate intake ensures maintenance of phosphate homeostasis, maintaining serum phosphate at a level of approximately 3-4 mg/dL in the serum. The vast majority of filtered phosphate is reabsorbed by type 2a sodium phosphate cotransporters located on the apical membrane of the renal proximal tubule. The expression of these cotransporters is increased by low dietary phosphate intake and several growth factors to enhance phosphate absorption. The expression is decreased by high dietary phosphate intake, parathyroid hormone (PTH), FGF23, and dopamine. Phosphate absorption in the remainder of the nephron is generally mediated by type 3 sodium phosphate cotransporters. No direct evidence has been found related to the regulation of these transporters in renal cells under physiologic conditions. The absorption in the proximal tubule is regulated such that the final excretion matches the dietary excess in order to maintain-homeostasis.

Etio-pathology of hardness of water & CKD and related health problems-

- The hardness of water is cause of renal stones & kidney failures .
- Due to high alkalinity crystals of Calcium Phosphate are formed and deposited in kidney .
- Later these renal stones block the filtering pores and the condition of renal failure develops.
- This condition is kept under control by repeated dialysis .
- Due to dialysis the serum phosphate level is increased , that of calcium is decreased.

- Parathyroid hormone level is also changed initially.
- The resulted secondary hyper-phosphataemia causes hypercalcaemia.
- This serum-profile with elevated Phosphorous is drastically cardio-damaging.
- Serum phosphate level is associated with cardiovascular risk even in individuals without kidney disease in whom the serum phosphate is within the normal range.
- Studies have shown that acute phosphate loads obtained through dietary ingestion cause endothelial cell dysfunction, manifested as a decrease in flow-mediated dilation, in healthy men. This finding raises the possibility that the prolonged and chronic hyperphosphatemia seen in patients with chronic kidney disease could play a direct role in the enhanced cardiovascular morbidity and mortality seen in these patients.
- This condition is responsible for increased and repeated infections and CVD due to deposition of these ions on cardiac soft tissues.
- Hyperphosphatemia complexes serum calcium, leading to lower-than-normal levels of ionized calcium. The decrease in ionized calcium triggers the release of PTH, resulting in a state of secondary hyperparathyroidism; high phosphate levels alone also stimulate PTH release. The elevated PTH levels lead to a high bone turnover state, resulting in the release of calcium, at the expense of bone, to normalize the serum calcium level.
- High phosphate levels also inhibit 1-alpha hydroxylase, a renal enzyme that produces active vitamin D by adding a hydroxyl group to circulating 25-hydroxycholecalciferol. This inhibition is most likely a result of the hyperphosphatemia-stimulated increase.
- The decrease in active vitamin D results in impaired gastrointestinal absorption of calcium, decreased renal reabsorption of calcium and phosphate, and impaired bone mineralization. Over months to years, bone density decreases. Additionally, the PTH and vitamin D derangements result in abnormal bone architecture. Clinically, the skeletal manifestations of chronic hyperphosphatemia include bone pain and fractures.

- Levels of 25-hydroxyvitamin D and calcitriol are reduced in heart failure, in association with increased serum phosphate but apparently normal serum calcium, after correction for serum albumin. This low vitamin D status is hypothesized to influence cardiac contractility through effects on intracellular calcium and phosphate levels–and has been linked to a modest increase in the risk of myocardial infarction in the general population.

Thus, due to poor quality of water, the resultant CKD finally precipitates Hyperphosphataemia , that causes Cardiac problems with repeated infections.

So, although dialysis is the life saving medical procedure but the life of the patient become miserable due to hyper-phosphataemia. As high serum phosphorous causes high incidences of infections and Cardiac problems. Many previous studies said that the dialysis patients mostly die due to cardiac arrest .

Based on these findings we design a research work to assess the health status of the people who resides in areas , where water is having high amount of TDS , high alkalinity and dissolved calcium . The victims of renal problems of that area who are on dialysis are chosen as subjects for the study and following observations and investigations are made-

Study Area- Mungali , Lormi, Kota region of Bilaspur and Mungali District and from various places from Raipur who are admitted to Dr Subha Dubey's Hospital for dialysis .

Study Time- April 2011- December 2013.

Sample size- 28 patients from the area with where water sample has high TDS , and who resides there more than 10 years with symptoms of CKD were picked up randomly.

Age range- 27-61 years

ESTIMATIONS –The following estimations were either done or if the reports were available , reports were collected.

- Estimation of serum Phosphorous was done in Aakash Patho lab , Bilaspur.

- Baseline phosphate levels were measured in fasting participants with an ammonium molybdate assay on the Olympus AU1000 auto-analyzer (normal range, 2.5 to 4.5 mg/dL).
- We estimated glomerular filtration rate (GFR) using the following equation:

186 × SCr $^{-1.154}$× age (In years) $^{-0.203}$×1.210 (if Male) × 0.742 (if female),

- Where SCr is serum creatinine (in mg/dL). This formula has been shown to have good agreement with iothalamate measurements of GFR. Participants with GFR <60 mL/min/1.73 m^2 body surface area were considered to have chronic kidney disease as per recent guidelines.
- Estimation of serum Calcium was done by 8 Ortho– Cresolphthalein Complexone (OCPC) method. (Abhay 's Patho Lab, Bilaspur)
- Estimation of serum Parathyroid hormone was done by immunoradiometric assay in Apollo Hospital, Bilaspur.
- Estimation of BUN was done by UV Urea method (Kinetic test)
- Estimation of serum Creatinine was done by Jeff's Kinetic method.
- Estimation of serum Vit D status could not be estimated.
- Estimation of Blood pressure was done by Auscultator method
- C-reactive protein was estimated by Kinetic method.

OBSERVATION-

- Serum Phosphorous -3.8-7.6 mg/dL
- Serum Calcium -8.7-10.2 mg/dL
- Serum Parathyroid -167 ng/ L
- BUN -32 mg/dL
- Serum Creatinine -3.9 mg/dL
- Blood Pressure- 197/101 mm of Hg
- Ca 2 x P complex - 72 mg /d L
- C-reactive protein –3.1.mg/L

AGE GROUP	NO. OF SUBJECTS	SERUM CALCIUM VALUE mg/ dL
27-30	4	8.5
30-35	5	10.4
35-40	5	9.6
40-45	3	10.3
45-50	4	8.3
50-55	3	9.4
55-61	4	10.5

AGE GROUP	NO. OF SUBJECTS	SERUM BLOOD PRESSURE VALUE mg/ dL
27-30	4	98/133
30-35	5	101/142
35-40	5	99/131
40-45	3	109/142
45-50	4	118/137
50-55	3	123/146
55-61	4	132/143

AGE GROUP	NO. OF SUBJECTS	SERUM C-REACTIVE PROTEIN VALUE mg/ dL
27-30	4	3.1
30-35	5	2.3
35-40	5	2.6
40-45	3	3.1
45-50	4	3.5
50-55	3	3.3
55-61	4	3.2

AGE GROUP	NO. OF SUBJECTS	SERUM PHOSPHOROUS VALUE mg/ dL
27-30	4	3.6
30-35	5	6.7
35-40	5	7.1
40-45	3	7.3
45-50	4	6.9
50-55	3	4.6
55-61	4	7.4

AGE GROUP	NO. OF SUBJECTS	SERUM CREATININE VALUE mg/ dL
27-30	4	2.4
30-35	5	3.1
35-40	5	3.1
40-45	3	2.3
45-50	4	3.0
50-55	3	3.4
55-61	4	7.4

AGE GROUP	NO. OF SUBJECTS	BUN mg / DL
27-30	4	29.6
30-35	5	30.5
35-40	5	33.1
40-45	3	34.6
45-50	4	28.7
50-55	3	32.4
55-61	4	30.1

Estimations of factors related to CVD-

Specific Factors Related to CKD	Percentage of patients affected
Anaemia	96.7
Hypocalcaemia(<8.1mg/dl)	57.3
Hyper-phosphataemia (>5.5 mg/dl)	55
Product of Ca and P(> 5.5 mg^2/dl^2)	23
C-reactive Protein	78

Association between CKD specific Factors and CV Events

Specific Factors related to CKD	CV Events		
	Developed (n=28) %	Non-developed (n=28) %	P value
Raised LDL(>100mg/dl)	(43.3)	(48.0)	0.417
Triglyceride(>150mg/dl)	(59.3)	(55.3)	0.484
Lower HDL(<40mg/dl)	(78.0)	(85.3)	0.101
Hypocalcaemia(<8.1 mg/dl)	(54.0)	(60.7)	0.243
Hyper-phosphataemia (>5.5mg/dl)	(52.0)	(58.0)	0.296
Product of Ca & P(>55mg mg^2/dl^2)	(22.7)	(23.3)	0.891
C-reactive Protein (positive)	(80.0)	(76.0)	0.347

Figures in the parentheses denotes corresponding percentage.

Association between Traditional Risk Factors and CV Events-

Traditional Risk Factors	Cardiovascular Events		
	Developed (n=28)	Non-developed (n=28)	P value
Smoker Non-smoker	(26.7) (73.3)	(27.3) (72.7)	0.897
BMI- Under weight & normal Overweight & obese	(78.7) (21.3)	(83.3) (18.7)	0.564
Anaemia(< 12 g/dl)- Present- Absent-	(97.3) (2.7)	(96.0) (4.0)	0.185
Hypertension- Present- Absent-	(84.7) (15.3)	(82.0) (18.0)	0.535

Diabetes- Present- Absent-	(29.3) (70.7)	(18.0) (82.0)	0.021

Figures in the parentheses denotes corresponding percentage.

The Serum Lipid Profile of the studied Victims-

Age group	No. of Participants	Total Cholesterol	Triglyceride	HDL	LDL
27-40	4	2.02±0.33	0.95±0.29	0.29±0.06	1.46±0.24
41-44	5	2.07±0.25	1.63±0.38	0.27±0.08	1.49±0.19
45-48	5	1.50±0.35	1.11±0.39	0.31±0.07	0.98±0.31
49-52	3	1.83±0.26	1.24±0.37	0.30±0.09	1.29±0.22
53-56	4	1.88±0.25	1.87±0.28	0.34±0.07	1.17±0.19
57-60	3	1.76±0.22	1.76±0.46	0.27±0.05	1.13±0.17
60+	4	1.89±0.17	1.91±0.42	0.28±0.02	1.23±0.16

(Values expressed as $\overline{x}$ mg.ml^{-1} serum and are presented as Mean value ± Standard Deviation).

MEAN, SD & 't' VALUES OF LIPID PROFILE OF VICTIMS IN COMPARISON WITH HEALTHY CONTROLS

Factors Of Lipid Profile	(mean ± Sd)		Change in percent-age value	(df = 130) t value
	Affected (n=28)	Not affected (n=28)		
Cholesterol(mg/ml)	1.25 (±0.08)	1.85 (±0.17)	□ □ □ □ □	17.63*,**
Triglyceride(mg/ml)	0.98 (±0.20)	1.49 (±0.36)	□ □ 53%	18.68*,**
HDL (mg/ml)	0.59 (±0.18)	0.29 (±0.02)	□ □ 51%	24.29,*,**
LDL (mg/ml)	0.54 (±0.16)	1.26 (±0.17)	□ □ 131%	49.00,*,**

* P<0.05 level, **P<0.01 level. SD Values showed in parenthesis.

Serum Profile Comparison

Lipid Constituents	(Mean ± SD)		Change in percent-age value
	Not affected	affected	
Phosphorous (mg/ml)	2.6 (±0.19) mg/dL	6.62 (±0.24)	154.61%
Calcium (mg/ml)	7.53 (±0.17) mg/dL	11.81 (±0.18)	56.83%
PAH (mg/ml)	47 (±0.04) pg/mL	77 (±0.04)	□ □ □ □ □ □ %
BUN(mg/ml)	6.63 (±0.21) mg/dL	9.92 (±0.25)	49.62%

Conclusions-

- We found hardness of water is significant cause of kidney damage and dialysis dependency in the study.
- In the area where the hardness and alkalinity of water were high , 49% of the studied patients belonged to that area.
- we found that lower levels of estimated GFR were associated with higher levels of serum phosphate when kidney function was impaired. (r = 1.056)
- High BP and Hyper-phosphataemia has very significant co-relation(r= 0.943)
- 47% of the chronic dialysis patients have significantly high level of serum Phosphorous .
- 63% of patients with high serum Phosphorous have arrhythmia , hypertension .
- Only 72% of these patients undergone ECG and 28% have undergone Eco-cardio-graphy , a significant number of them have shown calcium-phosphorous deposited cardiac tissues.
- Serum phosphorus mean was reported 6.2 mg/dL. Ten percent of patients had levels greater than 9 mg/dL and 30% had serum phosphorus levels greater than 7 mg/dL. The relative risk of death for those with a serum phosphorus greater than 6.5 mg/dL was 1.27 relative to those with a serum phosphorus of 2.4 to 6.5 mg/dL.
- Dyslipidemia is present in significant number of patients .

Thus, we can draw a conclusion from the study that there is significant relation between the quality of water and renal health of human, the poor quality of water specially with increased hardness and alkalinity is disastrous for human kidneys ,the medical procedures like dialysis precipitates hyper-phosphataemia like condition that induce life threatening CVD .

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