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Review

Demographic Diversity of Food Intake and Prevalence of Kidney Stone Diseases in Indian Continent

Manalee Guha [1#†], Hritwick Banerjee [2†], Pubali Mitra [1] and Madhusudan Das [1,*]

[1] Department of Zoology, University of Calcutta, 35 Ballygunge Circular Road, Kolkata 700019, West Bengal, India; manalee.guha@gmail.com

[2] Department of Biomedical Engineering, Faculty of Engineering, 4 Engineering Drive 3, National University of Singapore, Singapore 117583, Singapore; biehb@nus.edu.sg

* Correspondence: guha.manalee@gmail.com; Tel.: +91-94744-15095

† These authors contributed equally to this work.

Present Address: School of Biotechnology, Jawaharlal Nehru University (JNU), New Delhi, Delhi 110067, India

Abstract: Food intake plays a pivotal role of human growth, which necessarily contributes 45% of global economy and wellbeing in general. Consumption of balanced food is elementary for overall good health while a shift of equilibrium can lead to malnutrition, prenatal death, obesity, osteoporosis and bone fractures, coronary heart diseases (CHD), idiopathic hypercalciuria, diabetes and many more. Though CHD, osteoporosis, malnutrition, obesity are being classified thoroughly in the literature, there are fragmented findings in the regime of kidney stone diseases (KSD) and the correlation with food intake therein. KSD associated with hematuria and renal failure poses an increasing threat to the healthcare and global economy while its emergence of Indian populations is being affected with multi-factorial urological disorder resulting from several factors. In this realm, epidemiological, biochemical, macroeconomic situations been portrayed when food intake is also a paramount importance which rarely been forecasted. Hence, in this article we will be reviewing the corollary connotation with diverse food consumption and the efficacy it plays in KSD extrapolating in Indian context.

Keywords: food intake; food diversity; kidney stone disease; social epidemiology

1. Introduction

Kidney stone diseases (KSD) and associated research became rampant since calcium based kidney stone and its correlation with genetic predisposition analysis been introduced with convincing outcomes. To illustrate more, in USA itself, there was a sharp rise of KSD nearly 200% since 1964 through 1972 [1]. In the same way, European countries unlike Scotland (3.83% in 1977 to 3.5% in 1987), Germany, Spain, and Italy also have shown an increasing trend in KSD prevalence over the last decades [2-5]. Along with these geographical boundaries, Japan and some parts in Iran, USA etc. soon started investing money in conducting research to forecast KSD with generic pattern for the age population both for men and women [6-8]. While conducting studies, it found out that, KSD is considered to be prevalent with nearly 35% of the controlled group affected with hypercalcaemic nephrolithiasis disorder [9]. Along with this, reports holistically demonstrated that monozygotic twins (32.4%) have approximately 15% high frequency rate in comparison to dizygotic twins (17.3%; $P<0.001$) [10]. To extrapolate more in this paradigm, reports aptly depicted in Canadian context that even though the presence of dent disease and hypophosphatemic rickets with hypercalciuria, still a firm corollary established with the ancestors and genetic pattern with the family history [11]. Furthermore, to conclude the hypothesis they have counteract with several genetic analysis namely encoding VDR, calcium sensing receptor (CaSR), 25(OH)D 1α-hydroxylase, osteocalcin, uromodulin, and osteopontin etc [12]. On the other hand, studies portraying gender and age as

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principle parameters revealed, Iran and USA as peak KSD prevalent locations for 40-49 age group, while Japanese women shown the same in 50-59 age groups [1]. Data disclosed a similar pattern in Japan and USA for male group in 40-49 years of age regime, while Iran followed with a different trend [13]. This initial investigation lead to a conclusion that, it is increasingly unscientific to extrapolate KSD pattern based on age and sex only indifferent geographic locations. So, the researchers were challenged to come up with a set of new parameters to fine-tune more realistic solution in the regime of KSD and its prevalence therein.

Diet is an integral part of renal accumulation and thus filtration, which in turn will affect absorption and bodily homeostasis for renal stone occurrence [14-15]. The epidemiology differs in accordance with different geographical regions and social construct. In this context, food habit was proposed to be one of the major risk factor in renal stone formation as a form of epidemiology to urine composition [16-17]. Food pattern is one of the major factors for renal stone formation and stone material deposition can be manageable by regulating food intake. In the context of Indian food, there are collection of many tastes and flavours of food with diversity yet identity. From the colour rich food of Rajasthan, to the spicy food of Punjab, to the slightly sweet with oil based Gujarat food, to the southern little sour tasted sea foods, all are found in this land of paradise. Though several communities in the country are vegetarian, there is still a large range of non-vegetarian recipes with full of richness. Increase in urinary calcium excretion is highly related to high consumption of animal protein with reduction of urinary pH and citrate excretion which are the ultimatum of stone formation. Due to this vast food pattern in Indian soil, we believe there is a paramount interest to describe through this review article the nutritional aspect causing renal stone formation and the immediate effects therein.

2. Diverse Food Habits in India

The era of rapid globalization and packaged market products has shifted the equilibrium of nutritional scenario in India. Urban areas of Indian population has seen to absorb more packaged foods into their dietary meals thus overpowered bodily mass and causing premature obesity. In this realm, Indian cooking vastly reflects thousand years of history leading to diversity of flavours and thus innumerable regional unique dishes found herein [18]. Diversified food habit ranges parts of India with different geographical area span from Rajasthan desert to Madhya Pradesh forest or Maharashtra seashore to Jammu & Kashmir Mountain [18]. For the ease of description, Indian cooking is classified principally into the North Indian, East Indian, West Indian and South Indian cuisine based on the similarity and differences in the food habits. North Indian cuisines are distinguished by its unusually high use of dairy products and the prevalence of flat breads like roti and paratha, baked in clay ovens, over rice dishes. Even though parts of Western India like Gujarat and Rajasthan are predominantly vegetarian, the remaining cuisines have their fair share of meat or animal protein owing its origin to the Muslim incursion in India. South India can boast of a simply bewildering range of regional cuisine. With cuisine ranging from the rich northern style Mughlai cooking of the pre-dominantly Muslim dominant Hyderabad to the simple vegetarian dishes of Tamil Nadu, from the seafood, kebabs and puris of Maharashtra to the strong Portuguese influenced cuisine of Goa, from the coconut based recipes of Kerala and the Malabar fish dishes to the unique cuisine of a small diminishing Franco-Indian populate in Pondicherry, South Indian food habit encompasses a wide spectrum of culinary choices [18]. Socio-demographic, macroeconomic and alike life style in the coastal part of Karnataka and Kerala supports the fact of close proximity results in dietary habits and food style. Transition from South Indian to Eastern Indian food habit encompasses the cuisines from the states of West Bengal and Orissa to the North eastern states. The staple food of this region is largely depends on rice and wheat. East Indian food habit has a fair balance of vegetarian and non-vegetarian dishes with fish curries being the cornerstone of its non-vegetarian platter. Steaming and frying are the popular methods of cooking. So to quantify the diversity and correlate Indian food

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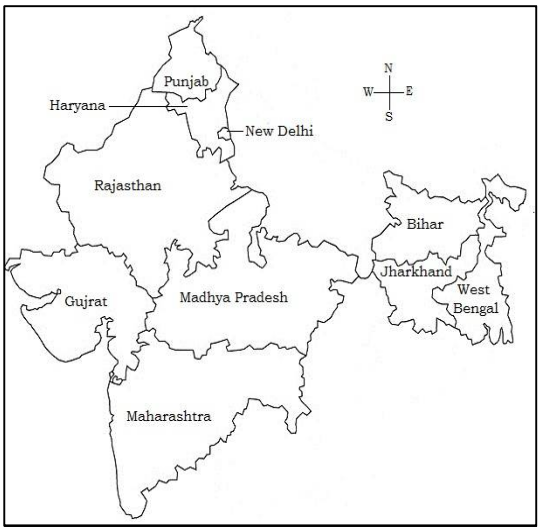
style with KSD, this review article investigates to curtail the prevalence of KSD cases which affect directly or indirectly with food consumption therein.

2.1. Stone Forming Area in India

In the context of India, KSD is very much prevalent with an expectancy of 12% in a total population as reported prone to urinary stones [19]. Out of this 12%, 50% of the population are severely affected with renal damage and thus even lead to loss of kidneys [19]. Unlike the South India where a few reported percentages affected from Urolithiasis, in North Indian there is a steep 15% of the population inside the regime of KSD [20]. So in the prospect of kidney stone belt that affected by KSD in India, a proper corollary needed to be established [20]. This stone beltoccupies areas of Maharashtra, Gujarat, Rajasthan, Punjab, Haryana, Delhi, Madhya Pradesh, Bihar and West Bengal (Fig.1). In these regions, the frequency of prevalence and recurrence rate of renal stone is high in the most of the members of a family.

(A) **(B)**



Stone belt States	Protein Consumption (gm/day/capita)*	
	Rural Area	Non rural Area
Rajasthan	71.4	63.7
Haryana	70.7	64.5
Punjab	67.2	64.4
Madhya Pradesh	62.7	59
Maharashtra	60.2	58.8
Bihar	57.6	62.4
Gujrat	56	56.4
Jharkhand	53.6	61.7
Bihar	57.6	62.4
West Bengal	53.3	55.5

Figure 1. Stone belt Area: (A) Major Kidney stone prevalent states of India continents, (B) Animal protein consumption per gram per day per capita that lead to KSD (*Ministry of Statistics and Programme Implementation 2012)

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2.2. *Food Habits with Stone Formation*

Indian food habit has been widely recognized risk factor of kidney stone formation [21]. An increase in calcium excretion after a load of protein was stated by many studies [22, 23]. The increase amount of consumption of animal product lead to higher calcium, oxalate and phosphorous in the urinary track [24, 25, 26]. These are the reason of stone formation initially in the form of insoluble calcium oxalate or calcium phosphate crystals. Proteins also increase uric acid generation which may end up in stone formation [25, 26]. High carbohydrate and lipid consumption have been shown similar changes [27]. Presence of high amounts of salt in the fast food especially in industrialized countries causes higher calcium in the kidneys [28]. Contrary of this, low calcium diet is suggested a risk factor as it increases intestinal absorption of oxalate [29]. Citric acid, potassium and magnesium act as negative regulator of stone synthesis [25]. For this reason inadequate intake of fruits, vegetables are considered as a risk factor for stone synthesis although some oxalate rich fruits like berries, chikoos and vegetables like tomatoes, spinach, beets are still there [30,31]. Vitamin C, when administered in higher quantities into the human body, is reported to have caused kidney stones in some cases [32]. It is inferred that Vitamin C gets converted to oxalates [33]. But some study reported that the oxalate excretion is little related to the dietary intake [34]. There are number of comparative studies between stone former and healthy control dietary habits [27, 29]. But results are contradictory. Most of these studies support the relation between food habit and kidney stones whereas opposite result is also there [35]. So the development and the progression of the disease are not so clear till now. In this regime, Table 1 corresponds to the reports conducted in different geographical study population to strengthening the correlation of food intake and its impact in KSD.

Table 1. Impact of food content and prevalence of KSD in some different zones.

Food Content	Impact on Stone Formation	Studied Zone	Reference
Dietary oxalate	Intestinal hyper absorption of oxalate, increased urinary oxalate excretion	Western part of India	Pendse et al., 1986 [36]
		Germany	Hesse et al., 1993 [2]; Siener et al.,2003 [37]
		North Carolina, USA	Holmes et al., 2001 [38]
		Italy	Meschi et al., 2004 [31]
		Boston	Taylor and Curhan, 2007 [39]
Dietary ascorbic acid	Increases urinary oxalate excretion	New York	Urivetzky et al., 1992 [40]
		Italy	Trinchieri et al., 1998 [41]
		Washington	Massey et al., 2005 [42]
		Sweden	Thomas et al., 2013 [32]
		Boston	Ferraro et al., 2016 [43]

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High dietary calcium	Reduces calcium oxalate stone formation	France	Bataille et al., 1983 [60]
		Boston	Curhan et al., 1993 [35]
		Germany	Siener et al.,2003 [37]
High intake of carbonated beverage	Increases urinary oxalate	Boston	Curhan et al., 1997 [45]
		Women of Omaha	Heaney and Rafferty, 2001 [46]
		Netherland	Asselman and Verkoelen, 2008 [47]
		Boston	Taylor et al., 2009 [15]
		North Carolina	Saldana et al., 2007 [48]
Protein rich diet	Increases acid load in kidney , increases risk of stone formation	Boston	Curhan et al., 1997 [45]
		Chicago, USA	Reddy et al., 2002 [49]
	Reduce the body's ability to absorb calcium	Switzerland	Nguyen et al., 2001 [81]
	Increases urinary calcium	Italy	Borghi et al., 2002 [14]
High intake of sodium	Increases urinary calcium	Northern India	Awasthi and Malhotra, 2013 [51]
		Post-menopausal women of Korea	Park et al., 2014 [52]

2.2.1. Protein:

High intake of protein especially animal protein is responsible for the relatively high prevalence of stones [23, 49, 53]. Animal protein containing purines are precursors of uric acid stones [54]. Amino acids like glycine, tyrosine and tryptophan convert into oxalate which is a very common component of kidney stone [55]. It also causes renal acid excretion, increased calcium reabsorption and increased renal reabsorption of citrate which ultimately leading to kidney stone formation [49]. Thus protein has a well contribution in making a bridge between calcium (increased calcium) and uric acid (decreased citrate) stone by its activity. While a balanced amount of protein intake is required to ignite metabolism, more consumption on the other hand increase the burden in kidney and liver [56]. To support the fact of protein consumption and associated kidney stone, reports have shown to increase three times in meat consumption in developed nation than developing ones like Asia in between 1970 to 1990 [57].

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153 ***2.2.2. Calcium Rich Food:***

154 Nishiura (2002) demonstrated a comparative study between the control individuals with stone
155 former individuals in respect of oral consumption of calcium diet with urinary excretion. In stone
156 formers there is dependency of urinary calcium excretion on diet whereas in controls there is
157 variation in calcium excretion with diet [34]. On the contrary, in a study it was shown that lower
158 calcium intake have a higher risk of stone formation than higher calcium intake [58]. Calcium rich
159 cereals like ragi, rajma, soyabeans or dairy products are main ingredients of regular diet in India
160 especially in stone belt region [18]. Conversely, the intake of calcium supplements outside of meals
161 causes an increased risk of stone formation in patients taking more calcium supplements [34].
162 Calcium intake in the outside of meal results a different effect than calcium intake with other
163 nutrients [45].

164 ***2.2.3. Carbohydrate Rich Food:***

165 Calcium stone formers exhibit an enhanced urinary calcium excretion to dietary content
166 containing high carbohydrates in comparison to healthy controls [59]. Carbohydrates reduce
167 reabsorption of calcium at the level of the distal tubule, but subsequent studies have shown that
168 glucose in a high concentration can enhance the intestinal absorption of calcium [60]. In addition
169 fructose increases the urinary excretion of calcium and oxalate, both of which are important risk
170 factors for calcium stones. Low urinary pH which is a major reason behind uric acid formation is the
171 trailing step of insulin resistance due to excessive fructose intake [61]. It was reported that uric acid
172 synthesis upregulated due to a single carbohydrate component fructose [62]. In India north and east
173 region contain maximum sucrose content in everyday life [63].

174 ***2.2.4. Sodium, Potassium:***

175 Modification of sodium by cutting down the daily intake of salt is advised for reducing kidney
176 stone recurrence [64]. Changes in the composition of urine i.e. increased calcium or decreased citrate
177 is attributed by increased sodium in diet [14]. It is shown that sodium can greatly affect urinary
178 excretion of calcium i.e. 25 mmol/day increases in urinary sodium causes an increase of 0.6 mmol/
179 day in urinary calcium [65]. Sodium and calcium excretion in the urine are well correlated shown in
180 some studies [52, 66]. Potassium also regulates value of urinary calcium in the body [67]. In one study
181 it was reported that in healthy subjects' diet with normal sodium quantity, dietary potassium
182 deprivation is associated to an increase in urinary calcium excretion [68]. The regular salty food
183 pattern is one of the reasons behind kidney stone formation in India [69, 70]. In contrast sodium and
184 potassium also increase urinary volume and pH which is required in cystine lithiasis [71].

185 ***2.2.5. Oxalate Rich Food:***

186 The dietary oxalate and the metabolism of vitamin C both originate Oxalate [38-40]. Urinary
187 oxalate excretion derives from metabolism but 10-50% comes from dietary oxalate [38]. In the
188 Western countries intake of oxalate ranges 100-300 mg/day and approximately 5-10% of the total is
189 absorbed in the intestine [72]. Intestinal absorption depends on the form in which it is consumed i.e.
190 soluble or insoluble and on its interaction with other food materials [73]. The main sources of dietary
191 oxalate are relatively few: spinach (45%), potatoes (10%), cold cereal (4%), nuts, coffee and tea account
192 for about 70% of all dietary oxalate [30, 31]. Although some study claimed that renal stone formers
193 consume more oxalate than healthy controls, but there are some studies where the oxalate quantity
194 of the diets of renal calcium stone does not only reason for renal stone formation [34]. Here in Table
195 2, we compiled diverse food intake in different Indian regions and its impact in KSD therein.

196

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197 Table 2. Different geographic regions of Indian population and their food habits.

198

Indian Part	Food*	Protein %	Calcium %	Carbohydrates %	Sodium-Potassium %	Oxalate %	Remark
Central	Mughlai	10-18	7	20-56	1	-	Protein
	Mushroom	6	1	-	9	-	
	Bamboo shoots	5	1	1	15	-	
	Pickle	-	-	-	50	-	
East	Fish	44	1	-	2-10	-	Protein and Carbohydrate
	Meat	52	0	-	2-12	-	
	Egg	26	5	-	3-5	-	
	Rice	5	1	9	1	-	
	Potato	4	1	10-20	6-12	1	
	Tomato	1	1	1	-	1	
	Spinach	5	9	1	3-15	1	
	Chives	6	9	1	2-8	-	
	Dairy	3	8	1	2-4	-	
North	Kidney bean	48	14	20	20-40	-	Protein and Carbohydrate
	Wheat	28	3	23	12	-	
	Corn	18	-	24	1-8	-	
	Mughlai	10-18	7	20-56	1	-	
	Paratha-Saag	10	11	30	1	-	
	Tomato	1	1	1	2-5	1	

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	Legume	10	2	4	0-6	1	
	Dairy	3	8	1	2-4	-	
West	Seafish	30-40	-	-	2-10	-	Protein
	Crabs	36	9	-	7-15	-	
	Nut	40	11	7	11	1	
	Rice	5	1	20-28	-	-	
	Coconut	6	13	-	1	-	
	Sweets	3	8	1	2-4	-	
South	Dosa/Idli	3	6	23	-	-	Protein and Carbohydrate
	Grains	26	10	14	6-15	-	
	Fish	44	1	-	2-10	-	
	Meat	52	0	-	2-12	-	
	Coconut	6	13	-	1	-	
	Pickle	-	-	-	50	-	

*Some most common food consumed by different parts of India

3. Mechanism of Different Types of Stones According to Food Habits

3.1 Impact of Food in the Mechanism of Stone Formation

Kidney stones are named according to the names of the crystals which make up the hard part of the stones: Calcium Oxalate, Calcium phosphate, Uric Acid, Cysteine, Struvite. In India, calcium oxalate and calcium phosphate stones are predominant whereas report of uric acid and cystine stone is very few [74]. Struvite stones are not in consideration as it is formed by bacteria kind ammonia in urinary tract infections and generally not found in Indian population in much extent [21].

3.1.1 Calcium Stone

Calcium is the major element of about 80-90% of all urinary stones [75]. They are usually made of calcium oxalate or calcium phosphate or mixed of them detected in chemical or infrared spectrometric analysis [76]. Calcium phosphate may solidify in the renal interstitium and later on papillary surface along with Calcium oxalate [77]. Many studies reported the derivation of 10-50% of the urinary oxalate from diet like dark-green leafy vegetables, spinach, beets, beans, cereals, dietary

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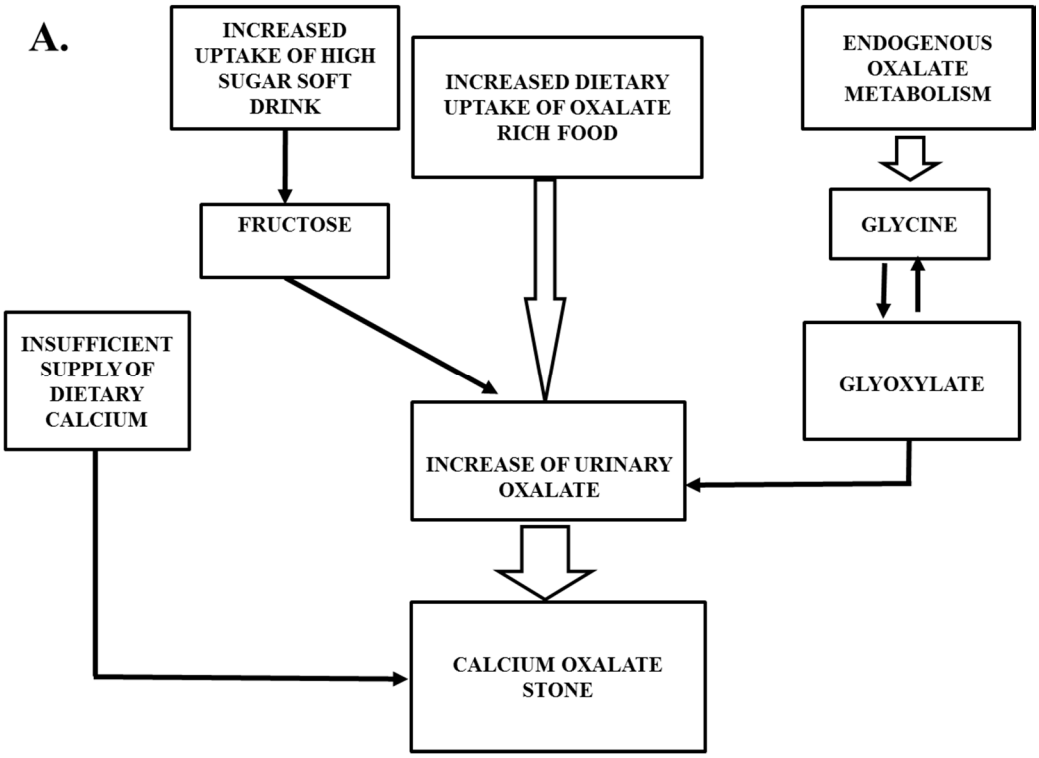
ascorbic acid, glycine rich food like animal proteins, chocolate, black tea etc. [2,38-40,73,78] Protein breakdown product glycine in a metabolic pathway oxidised to glyoxylate which is the precursor of oxalate, a major stone component [79]. In a study it was shown overconsumption of animal protein create observable increased rate of urinary calcium (23%) and oxalate (24%) [80]. High fructose consumption from soft drinks is associated with an increased risk of hypercalciuria, hyperoxaluria and hyperuricosuria [39, 81]. In an experiment higher urinary calcium excretion occurred in rats fed high-fructose diets compared to rats fed high-starch diets [82, 83]. An insufficient supply of dietary calcium is also a notable risk factor for both calcium oxalate and phosphate stone formation [34, 45]. High salt intake has been associated with elevated urinary calcium excretion by reducing tubular reabsorption which is an output of free particle model on crystallization [84]. This high concentrations of calcium in the urine combines with oxalate and phosphorus to form stones (Figure 2).



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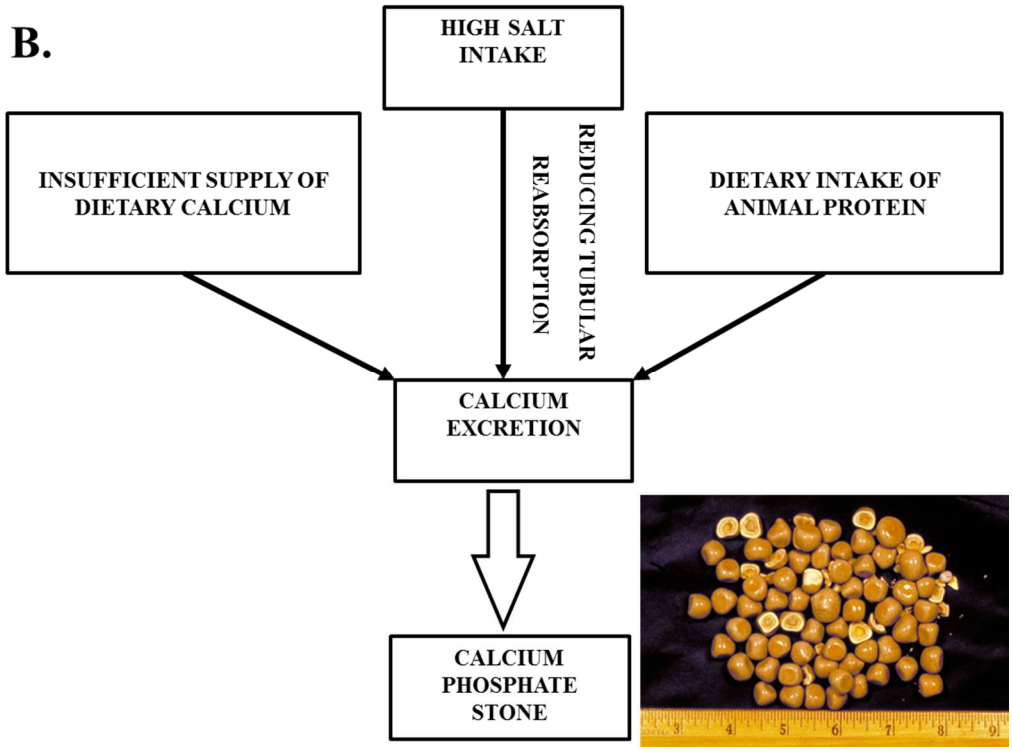

Figure 2. Calcium stone formation with food habits (A) calcium oxalate stone formation (B) calcium phosphate stone aggregation.

KSD is mostly accompanied with hypercalciuria nearly 30–60% due to high intestinal calcium absorption [85]. In hypercalciuria, calcium stimulates supersaturation of mineral crystallization and makes obstacle of stone inhibitory factors (citrate, GAGs) by binding with them [86]. Other events like bone resorption, renal leakage take a positive part in implicating hypercalciuria manifestation [87]. By reducing calcium intake, heights of calcium excretion are manageable at a certain level [88]. So, dietary management should be required for the regulation of hypercalciuria. A diet based study demonstrated that lower intake of animal proteins and salts with optimum calcium intake have great impact on reducing the chances of stone recurrence [52, 66]. Overtaking salts and proteins increase urinary calcium excretion nearly 23% and the outcome of it kidney stone formation [52]. Fructose also has a significant role on it and it was noticed in rat model experiment with elevated urinary calcium excretion [89].

3.1.2 Uric Acid

Uric acid stones record of nearly 5-10% of urinary stones [90]. Low urine pH is very much familiar to this type of stones [22]. Urinary uric acid solubility decreases approximately 185 mg/dL when urine pH dropped from 7 to 5. At higher pH 95% of uric acid is in its soluble urate form and at lower pH solubility is decreasing in most of the uric acid [91]. Excess uric acid excretion through urine, hyperuricosuria is caused by purine rich diet, precursor of uric acid (Figure 3) [90]. Foods containing high protein especially animal protein like poultry things eggs, meat, seafish and some plants products like seeds, nuts are the highest source of purines [92]. It was reported that daily increase of animal protein significantly increased 48% in the uric acid excretion [93]. Another factor sweet drinks containing high fructose level are in relation with increased risk for renal stones. In one study it was shown that over consumption of fructose results in a rapid rise of serum uric acid through increased purine synthesis [89]. In the metabolism pathway fructose breaks into insonine and xanthine with the help of energy driver ATP. This xanthine ultimately promotes uric acid formation by using ADP as a substrate [94].

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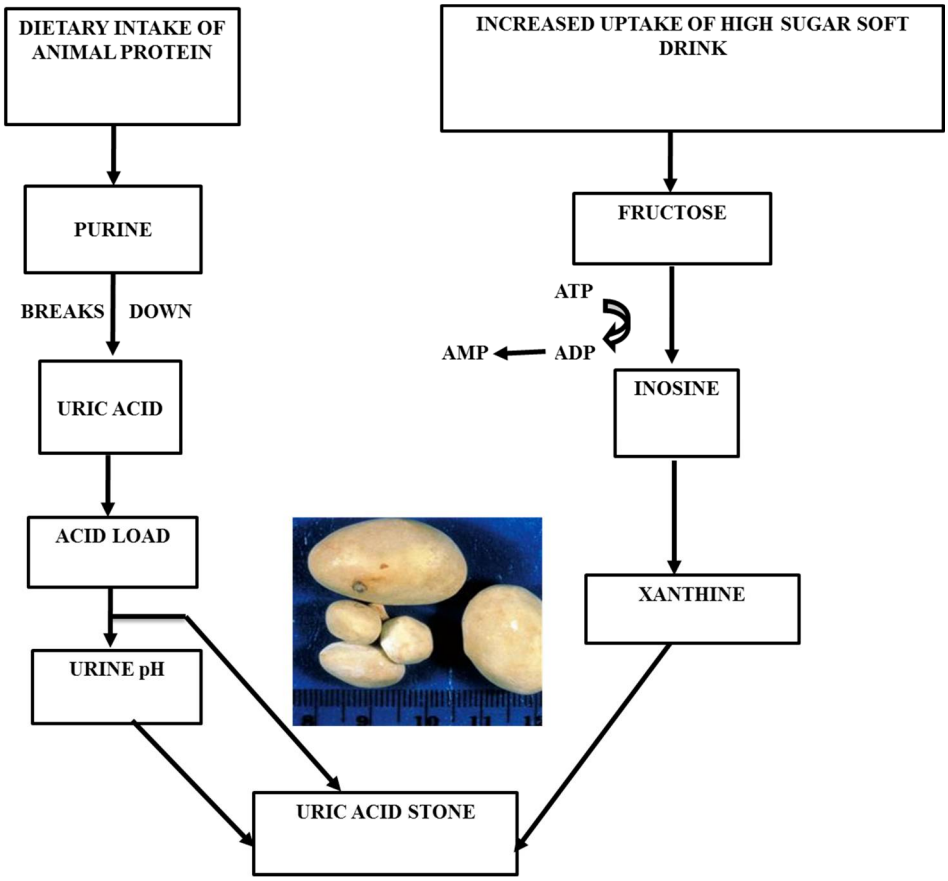

Figure 3. Uric acid stone formation with food habits.

3.1.3 Cystine Stone

Cystine stones are very rare nearly 1–2% of urinary caliculi [95]. These are formed in those people who have tendency of excessive cystine leakage from the kidneys into the urine [96]. Maintaining cystine concentration in urine below 200 to 300mg/L is the best medical care this type of stone [97]. High liquid substance is required for producing at least 3 litres volume urine during a day to decrease the high level of urinary cysteine [98]. In addition potassium citrate is usually taken as a drug for reducing renal acidosis [99]. In food, animal proteins containing methionine like meat break down into cystines which increase urinary cystine level in the body [100]. In a study it was shown that urinary cysteine excretion reduced to 34% by consuming very low protein diet nearly 20g/day [101]. Overtaking sodium has high impact in this disease manifestation by promoting excretion of cysteines (Figure 4) [102].

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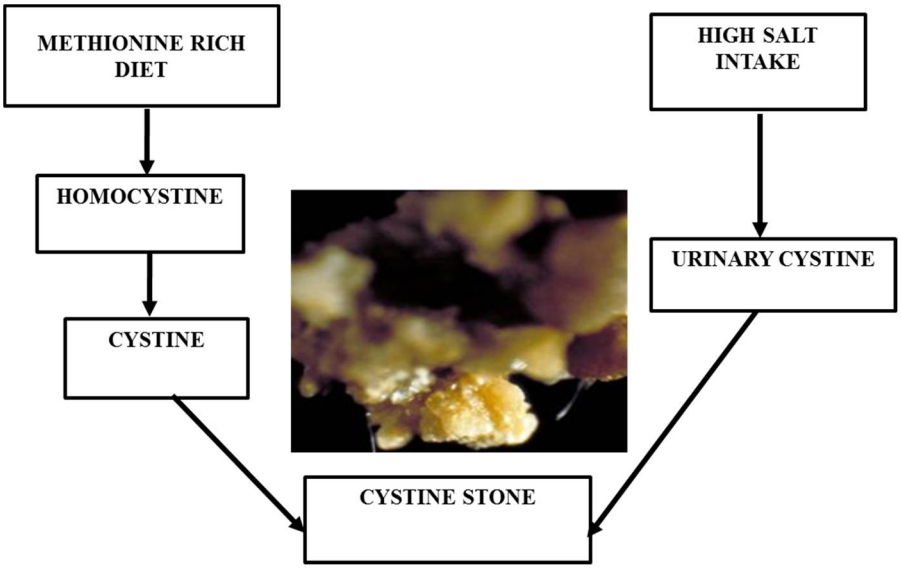

Figure 4. Cystine stone formation with food habits.

4. Food Diversity and Nutritional Effects In Indian Population:

Over nourishment and undernourishment are both great burdens for society. At one point over taking protein, carbohydrate, oxalate rich foods enhance the occurrence of renal stone [23, 59, 61, 62, 72]. Whereas raised protein break down and protein undernutrition are familiar in chronic kidney disease (CKD) patients [103]. So the vital factor protein has an observable influence on these two kidney disease by its presence or absence. Several studies have suggested CKD is a recognized issue among stone formers [104-106]. Renal stone is a result of an impaired kidney function and an important clinical parameter serum creatinine level should be monitored in the follow up [107]. It is reported in a population study from US that elevated serum creatinine levels cause nearly 25-44% increased risk for CKD in stone formers [108]. Protein rich diet may increase serum creatinine levels, which has an impact on the changes of glomerular filtration rate [109]. Interestingly there are multiple reports on protein–energy malnutrition which occur during the CKD especially in the mature stages(3-5) and the risks of mortality is high due to the occurrence of protein malnutrition at the time of dialysis [110]. Many studies have also shown that CKD patients have much resting energy expenditure in comparison to normal individuals and during dialysis this expenditure is increased more [111,112]. For this reason they require more energy given food especially protein. In a conclusion food management is very important tool for maintaining the health of kidney.

In Indian scenario, where mostly there are people under the poverty line, income plays a major role in determining the status of life and food intake in general [101]. This poor socioeconomic position is associated with chronic malnutrition since it inhibits purchase of essential nutritious foods for growth and development as the price rates are not increasing with income proportionately [113]. So, there are many indirect pathways which constitute poor healthcare, malnutrition, abstemious food intake that leads to a CKD and many others (Figure 5) [114]. In prenatal cases where calcium intake and nutritional levels need to properly maintained are frequently been underprivileged. In this section, we have given an introductory causality model that very much exists in India in the realm of nutrition, food intake and their impact in human lives.

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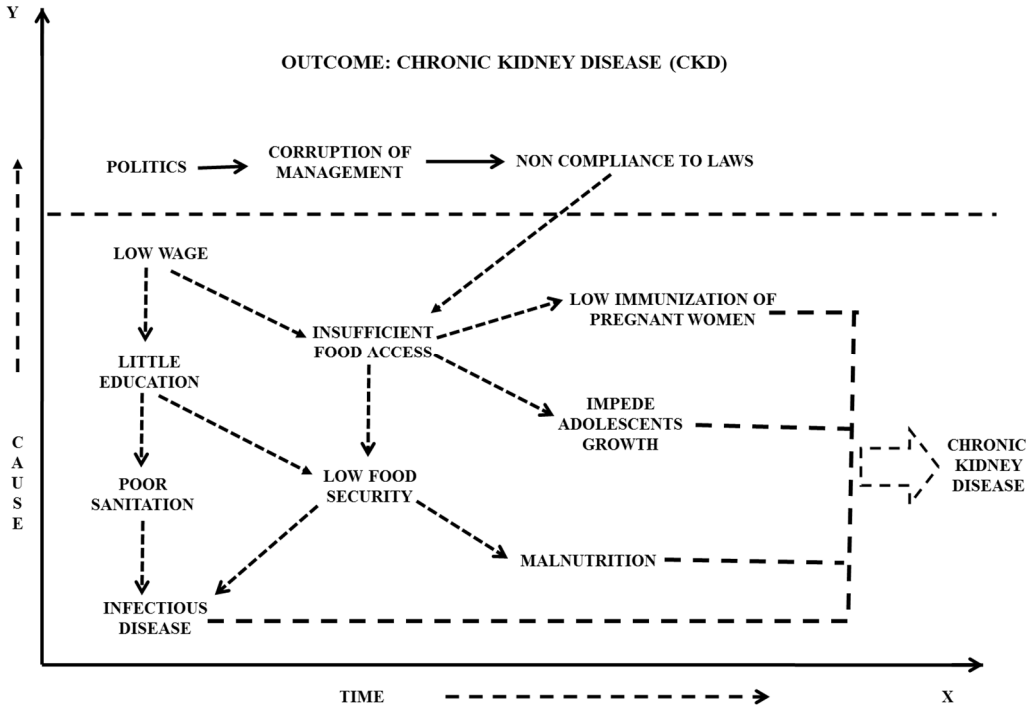

Figure 5. Diagrammatic representation of a causal conceptual model in Indian scenario. An example where societal factors like economy, political view, education effects health and wellbeing of poorer class of Indian population.

5. Concluding Remarks

KSD is a rising concern, major healthcare burden and associated with hematuria, renal failure. The risk of renal stone varies from 1-5% in Asia, 5-9% in Europe, 10-15% in USA and 20-25% in the middle-east [115]. Dietary therapy can be one of the promising solutions to minimize the cases of recurring kidney stone formation and hence better quality of life. So, an awareness of health concern and optimized food therapy can potentially curtail the cost of hospitalization and enhance compliance in general. Vis-à-vis to dietary control, insufficient understanding of molecular, genetic basis of pathogenic mechanisms remains a critical barrier to early detection, treatment. Mostly stone formation attributes two mechanisms– 1) renal calcium leak, excessive absorption, bone resorption/formation imbalance 2) mineralization. Dietary factors have been widely recognized one of the prime risk factors of kidney stone formation [59]. On the other hand, parathyroid hormone primarily modulates calcium balance. It increases calcium excretion in kidney. VDR regulates calcium homeostasis by affecting bone resorption, calcium absorption. CLDN-14, tight junction protein, decreases Ca2+ permeability whereas MGP regulates calcification [76]. SPP1 prevent renal stone formation by decreasing aggregation of crystals and binding to renal epithelial cells [116]. These are the reason of stone formation initially in the form of insoluble calcium oxalate or calcium phosphate crystals. Most of these genetically inspired reports support the relation between food habit and kidney stones whereas stratified contradictory reports are also there. So conclusively, there is a high demand to understand better the correlation between food intake and CKD and hence quantified researches, aligned case studies need to establish in near future.

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