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Prevalence and Risk Factors in Kidney Stone Formation: In Southern Region of Rajasthan

Varsha Choudhary*, Jyoti Bhardwaj and P P Singh

Department of Medical Science, Rajasthan Vidhyapeeth University, Rajasthan, India

Corresponding author: Varsha Choudhary, Department of Medical Science, Rajasthan Vidhyapeeth University, Rajasthan, India, E-mail: drjyotiameta@gmail.com

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Abstract

Urolithiasis is a global problem affecting all geographical regions. This study compiles the epidemiology of renal calculi focusing on prevalence, occurance and re-occurance rate in global perspective. Recent studies have reported that the prevalence of urolithiasis has been increasing in the past decades in both developed and developing countries. Risk factors are associated with the formation of urinary calculi can be divided into two main groups, intrinsic or extrinsic factors. The former one includes age, gender, ethnic and familial backgrounds; while the latter group consists of climate and environment, lifestyle and dietary habits, occupation and education level. For this purpose Stone Formers (SF) and controls were selected. Urinary obstruction was observed in none of the patients. TBAR levels in this series are higher in SF. A significant relationship was observed between TBAR *vs*. calcium and TBAR *vs*. oxalate. No difference in calcium, oxalate and TBAR level in SF with and without family history. In conclusion, the data suggested that hypercalciuria, hyperoxaluria, hypocitraturia, hyperuricosuria, hypomagnesuria are important urinary risk factors.

Keywords: Urolithiasis; Risk factor; Prevalence; Stone formers; Hyperoxaluria; Hypercalciuria

About the Study

Recent studies have reported that the prevalence of urolithiasis has been increasing in the past decades in both developed and developing countries [1]. Temporal troughs are clearly visible on the prevalence of urolithiasis, but what concerns us most are:

- Rising or static incidence of nephrolithiasis all over globe, which is linked with improvement in socioeconomic status and remains uncurtailed despite best health education and facilities.
- Its aggressive unkind behavior in urinary tract [2,3].

The most important factors, determining the prevalence, incidence, recurrence rates and constituent of calculi, are climate and dietary habits [4]. Further, grafted upon it are environmental provocations often causing genetical incitements [5]. While both basic and applied researches are glossing over the final solution of this problem, a clinicians is accosted with the immediate problem of management to give relief to patient and long term problem to retard or arrest the recurrence of the disease. Admittedly lithotripsy has given relief against the infliction of surgery but it too has several limitations. For example it can treat the stone of limited size and that it gives no protection against recurrence. Therefore, one has to look for better management care and suitable candidates for chemolysis of calcium oxalate and calcium phosphate stones. Till we arrive at a final destination, it is felt that for a clinician urinary profile remains the best information [5-8]. Since urinary profile is dependent on multiple risk factors, wide fluctuations are reported in different populations. In the present study, therefore, we have examined urinary profile of 180 Stone Formers (SF) and 50 matched controls.

24 hours urine was collected in bottle containing 10 ml chloroform. Standard methods were used for determination of various biochemical parameters including THP and mucoproteins and TBAR [9]. pH was determined In fresh urine samples by pH meter. Student t test was applied to ascertain significance and relationship was evaluated by correlation coefficient (r).

None of the patient had urinary obstruction as was evident by serum creatinine levels. Calcium level tended to be higher in SF but not significant. On the contrary oxalate excretion was higher. Magnesium excretion was lower both SF and controls. Citrate levels were significantly lower in SF. TBAR levels in this series are higher than those reported for western population and that SF tended to excrete it more. When only two factors *viz* calcium and oxalate were taken together in patients, significant relationship was observed between TBAR *vs.* calcium and TBAR *vs* oxalate. Rest of the relationships did not show discernible trend. No difference was observed in calcium, oxalate and TBAR levels in SF without family history and with family history (**Tables 1-3**).

Table 1: Urine chemistry of controls and stone formers (mg/24 hrs).

Parameters	Normal subjects			Stone formers		
	Total (n=50)	Male (n=30)	Female (n=20)	Total (n=180)	Male (n=129)	Female (n=51)
PH	5.97 ± 0.39	6.00 ± 0.32	5.94 ± 0.46	5.89 ± 0.52	5.94 ± 0.40	5.84 ± 0.64
Volume	1289 ± 428	1249 ± 404	1330 ± 452	1342 ± 384	1332 ± 404	1352 ± 364
Creatinine	843 ± 285	966 ± 352	720 ± 217	894 ± 229	1000 ± 245	789 ± 213
Calcium	99 ± 24	107 ± 31	92 ± 17	128 ± 32	141 ± 33	115 ± 31
Oxalate	23 ± 6	24 ± 6	22 ± 5	32 ± 8*	34 ± 8*	30 ± 9*
In phosphorous	483 ± 158	496 ± 176	480 ± 139	554 ± 111	581 ± 136	526 ± 86
Uric acid	392 ± 171	396 ± 154	389 ± 189	424 ± 136	432 ± 101	415 ± 171
THP	67 ± 19	68 ±18	66 ±19	77 ± 24	78 ±22	76 ± 25
Mucoprotein	95 ± 26	97 ± 28	92 ± 24	104 ± 26	114 ± 28	94 ± 24
GAGs	6.5 ± 2.5	6.8 ± 2.9	6.3 ± 2.1	6.3 ± 2.6	6.5 ± 2.9	6.2 ± 2.3
Magnesium	48 ±17	49 ± 17	48 ± 16	41 ± 11	42 ±10	40 ± 11
Citrate	433 ± 151	413 ± 148	453 ± 153	249 ± 84	249 ± 86	250 ± 83*
TBAR(µmol)	0.38 ± 0.10	0.39 ± 0.10	0.37 ± 0.11	0.49 ± 0.18	0.52 ± 0.20	0.47 ± 0.17
Note:*P<0.05						

Table 2: Correlation coefficient between different parameters in various groups.

Parameters	Calcium *vs.* TBAR	Oxalate *vs.* TBAR
N O	0.1744	0.1155
H O	0.0413	0.0867
N Ca	0.0169	0.075
H Ca	0.1744	0.1155
N Mg	0.0196	0.0509
Hy M	0.0215	0.1272
N Cit	0.021	0.0662
Hy Cit	0.1144	0.493
N Ca+N O	0.3152*	0.6543*
H Ca+H O	0.4647*	0.9995*
N Ca+H O	0.0364	0.8941*

H Ca+N O	0.9991*	0.0104
N Ca+N O+N Mg	0.0583	0.1117
H Ca+H O+Hy Mg	0.1512	0.0181
N Ca+N O+N Mg+N Cit	0.0303	0.1026
H Ca+H O+Hy Mg+H Cit	0.2812	0.0978
Note: *p<0.05. Abbreviation: N: Normal; H: Hyper; Hy: Hypo; O: Oxalate; Ca: Calcium; Mg: Magnesium; Cit: Citrate		

Table 3: Urine calcium, oxalate and TBAR excretion in stone formers (mg/24 hrs) with and without family history.

Parameters	SF without family history		SF with family history	
	First episode (111)	Recurrent SF (34)	First episode (28)	Recurrent SF (7)
Calcium	136 ± 38	138 ± 42	152 ± 40	154 ± 34
Oxalate	32 ± 10	34 ± 9	34 ± 9	36 ± 8
TBAR (µmol)	4.9 ± 1.8	4.9 ± 1.9	4.8 ± 1.9	5.1 ± 1.8

Discussion

It is abundantly clear that even today urinalysis is the best source to decipher the etiopathogenesis of urinary calculi except in small percentages of cases with anatomical defects or recurrent massive infection. This is still more useful in developing populations of the world including India where facilities for advance investigations are not available in majority of the health centres. For example, urinary calcium/creatinine can reasonably predict the type of hypercalciuria; persistently raised oxalate levels on low oxalate diet, suggest absorptive oxaluria/renal oxalate leak; high uric acid excretion on vegetarian diet or low animal foods give inkling about its genetical predisposition. Similarly, low magnesium excretion due to defective absorption or low citrate excretion due to defective metabolism or dietary habits can also be reasonably predicted.

None of the SF showed overt or sub-clinical manifestation of hyperparathyroidism in this series. Biochemical investigations including urinary calcium level also suggested the same. Further none of the patients suffered from absorptive hypercalciuria as judged by the criterion laid down [10]. Hyperoxaluria rather than hypercalciuria is major stone risk in this population [11]. This study also confirms the same though the percentage of hyperoxaluric patients is relatively less (20%) than other series. The mean excretion of oxalate was about 50% higher in SF than the controls. Neither of the patients suffered from primary hyperoxaluria as indicated by urinary oxalate levels nor any of them appeared to be suffering from nutritional inadequacy and excess as they were on multivitamin preparation and consume low oxalate diet. They also did not take any additional supplement of vitamin C which could be an important contributor to urinary oxalate. Logically, therefore, it appears that they suffered from renal oxalate leak. Lower urinary magnesium level suggested defective absorption of this element as Indian diet is sufficient in magnesium. Lower citrate intake is very common feature in SF of this population and the same was glaringly evident in this series also. Lower urinary PH is a dominant factor to determine the urinary citrate excretion but importantly no significant difference in PH was visible between SF and controls. At present we unable to offer the exact mechanism of lower citrate excretion and efforts are in progress to explore the causes.

We determined urinary TBAR levels also; surprisingly its excretion is higher in this population than western subjects. The one possibility is dietary contribution to the raised TBAR levels. Its excretion tended to be higher in SF suggesting that SF is exposed to some kind relatively higher oxidant stress. The one strong factor appears to be enlarged oxalate pool in SF because hyperoxaluric patients showed positive correlation between urinary oxalate and TBAR level in this study and in our unpublished data [12]. The rest of correlations as examined in this study and given in table-II do not convey any meaningful conclusion. Oxalate induced oxidant stress has been reported by other workers also [13]. The calcium and oxalate excretion did not show any significant difference between SF with family and without family history which is contrary to many other workers who observe difference in TBAR levels. Comparable data are not available in literature [14-17].

Conclusion

In conclusion our data indicate; that hyperoxaluria, hypomagnesiuria and hypocitraturia are important risk factors; that oxalate excretion poses double risk due to concomitant higher oxidant stress load.

This study also confirms the same though the percentage of hyperoxaluric patients is relatively less (20%) than others of series.

At present we unable to offer the exact mechanism of lower citrate excretion and efforts are in progress to explore the causes.

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