Urol Res (1986) 14:59–62

Urological Research

The Etiology of Urolithiasis in Udaipur (Western Part of India)*

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Accepted: July 10, 1985

Summary. Fifty-two cases of urinary tract calculus disease were investigated for dietary habits, routine chemical and microscopic urinalysis, bacterial culture, quantitative analysis of 24 h urine sample and qualitative analysis of the stones. 54 out of the 56 stones analysed were of mixed type. Magnesium ammonium phosphate was present in 78.2% stones. Dietary habits revealed principal dependence on cereals, lack of animal proteins, consumption of oxalate rich vegetables and widespread consumption of tea. Urinary tract infection was present in 63.7% of the cases. Significant calcium oxalate crystalluria (2+ to 4+) was present in 34.6% of the cases. Hyperoxaluria, hypercalciuria associated with hyperoxaluria-lower excretion of magnesium and citric acid were important urinary risk factors in the local population. These observations strongly suggest the multifactorial etiology of stone disease in this region. Imbalanced nutrition and urinary tract infection were the principal risk factors for urolithiasis in this study.

Key words: Stone formers, Urolithiasis, Stone inhibitors, Hyperoxaluria, Stone analysis.

Introduction

Urinary calculus disease continues to be a problem in many parts of the world. In India upper and lower urinary tract stones occur but the incidence shows wide regional variations [1]. The incidence of urolithiasis is low in the southern part of the country compared to other parts of India. Isolated parts of Rajashan have been labelled as stone areas on the basis of the observations of clinicians, but no epidemiological studies are reported in the literature. We reported a high and progressively increasing incidence of urolithiasis in Udaipur and some other parts of Rajasthan in the western part of India [6, 9]. This was a new observation.

Over the last four years we studied the etiology of urolithiasis in the local population by analysis of single or grouped factors [7, 8, 12, 14, 15].

We have now examined a comprehensive etiological profile of 52 stone formers. The results of this study are reported.

Material and Methods

Fifty-two stone formers, identified radiologically, admitted to surgical wards of the RNT Medical College Hospital were included in this study. The present and past dietary habits were recorded in detail by verbal questionnaire.

All the patients were placed on a standard low oxalate hospital diet (70–160 mg/day) which was continued throughout the period of study. The pH of the first morning sample of urine was estimated thrice by narrow range pH papers (BDH). At the same time urine sample was also collected for routine urinalysis and for bacterial culture.

After the patient had taken the standard diet for 2 days, a 24 h urine sample was collected in a 2.5 l bottle containing toluene preservative. The sample was immediately taken to the laboratory for quantitative analysis.

The quantitative urinalysis was carried out for creatinine [5], calcium [7], oxalic acid [3], uric acid [5], inorganic phosphorus [5], magnesium [5] and sodium and potassium (EEL Flamephotometer, England).

All the patients were subsequently operated upon for the removal of stone, which was washed with distilled water and taken to the laboratory for qualitative analysis by wet chemical analysis [5].

Results

The dietary habits of the 52 stone formers, shown in Table 1, indicate that: (a) Wheat and maize were staple foods (b) consumption of pulses was inadequate (c) none of the pa-

* This paper was presented by Dr. A. K. Pendse at the 24th Biennial Congress of the International College of Surgeons at Manila (Philippines)

Table 1. Dietary habits of 52 stone formers

Food	Frequency of intake				Daily or frequently %
	Daily	Frequently	Occassionally	Rarely/never	
Wheat	28	19	5	–	90.4
Maize	8	33	11	–	78.8
Rice	2	7	19	24	17.3
Pulses	2	46	4	–	92.3
Eggs	–	2	14	36	3.8
Meat/fish/chicken	–	–	11	41	–
Leafy vegetables	–	14	11	27	65.4
Tea/Coffee, 3 cups or less	16	–	2	–	30.8
3 cups	34	–	–	–	65.4

Frequently = 4 or more times per week; occassionally = 1 to 3 times per fortnight; rarely = once in 3 or more weeks

Table 2. Urinalysis findings in 52 stone formers

	Renal (19) %	Ureteric (14) %	Vesical (15) %	Multiple sites (4) %	Total (52) %
Albuminuria	42.1	28.6	33.3	75.0	38.5
Hematuria	36.8	14.3	–	75.0	23.1
Calcium oxalate crystalluria[a]					
Nil	26.3	50.0	73.3	25.0	46.1
1+	21.0	21.4	20.0	–	19.2
2+	42.1	21.4	6.7	75.0	28.8
3+	5.3	7.1	–	–	3.8
4+	5.3	–	–	–	1.9

[a] Moderate to severe (2+ to 4+) calcium oxalate crystalluria – 18 patients (34.6%)

Table 3. Bacterial culture of urine in 52 stone formers

Organism	Renal (19) %	Ureteric (14) %	Vesical (15) %	Multiple site (4) %
S. albus	10.6	28.6	–	25.0
S. faecalis	5.3	–	6.7	–
P. pyocyaneous	5.3	–	–	25.0
E. coli	26.3	14.3	33.3	–
S. albus + S. faecalis	5.3	7.1	6.7	–
S. albus + E. coli	5.3	7.1	2.7	
S. albus + P. pyocyaneous	5.3	–	6.7	–
S. faecalis + E. coli	–	–	–	–
Total	63.1	57.1	80.0	75.0

tients took nonvegetarian food daily and 69.2% cases either took it rarely or never; (d) 65.4% patients took oxalate-rich vegetables frequently but 38.5% had avoided oxalate since the diagnosis of calculus disease and (e) all the patients took tea (or rarely coffee) regularly.

Albuminuria and haematuria were present in 38.5 and 23.1% of the patients respectively (Table 2). Patients with multiple calculi were maximally affected followed by those with renal stones.

Microscopic examination of the urine showed calcium oxalate crystalluria in 53.9% cases; mild (1+) in 19.2 and moderate to severe (2+ or more) in 33.5% of the cases.

Bacterial infection (Table 3) was present in 67.3% of the cases (Table 3).

Table 4. Urinary Ph, 24-h-output and excretion (mg) of creatinine, calcium, oxalic acid, uric acid, inorganic phosphorus, magnesium, sodium and potassium

Site	No.	Volume	pH	Creat-inine	Cal-cium	Oxalic acid	Uric acid	Inorganic phos-phorus	Magne-sium	Sodium	Potas-sium
Renal	19	1,503.9 ±407.1	6.38 ±0.13	890.7 ±98.1	178.7 ±25.3	43.8 ±7.8	307.1 ±41.4	450.6 ±85.01	31.6 ±4.5	2,390.4 ±557.7	1,002.1 ±127.7
Ureteric	14	1,435.7 ±202.9	6.0 ±0.1	811.6 ±88.4	118.2 ±11.3	39.8 ±7.2	340.6 ±91.2	512.8 ±52.8	43.0 ±7.5	3,014.3 ±493.7	897.8 ±159.3
Vesical	15	1,700.0 ±191.1	6.5 ±0.2	967.9 ±117.8	190.0 ±18.1	35.3 ±5.2	367.5 ±66.5	650.0 ±77.7	37.0 ±5.0	3,033.6 ±645.2	777.8 ±99.8
Multiple site	4	1,897.5 ±494.0	6.5 ±0.2	1,096.4 ±142.5	196.3 ±48.7	25.9 ±7.5	513.3 ±213.9	567.9 ±160.7	35.7 ±6.1	2,544.2 ±544.8	693.7 ±212.3

Table 5. Qualitative stone analysis of 56 stones taken out from 52 stone formers

Radical	Renal (23) %	Ureteric (17) %	Vesical (16) %	Total (56) %
Calcium	100.0	100.0	100.0	100.0
Magnesium	78.2	88.2	81.2	82.1
Ammonium	78.2	88.2	81.2	82.1
Carbonate	17.4	–	31.2	16.0
Phosphate	95.7	100.0	87.5	94.6
Oxalate	91.3	94.1	93.7	92.8
Uric acid	39.1	23.5	37.5	33.9
Cystine	4.3	–	–	1.8

The urinary output, pH and 24 h excretion of creatinine, calcium, oxalic acid, uric acid, inorganic phosphorus, magnesium sodium and potassium are given in the Table 4. Urinary output was within normal range in all the subjects. Calcium excretion of > 200 mg/day was present in 25% patients. Oxalic acid excretion of > 50 mg/day was present in 23.0% cases. Magnesium excretion was below normal in all the patients. Sodium and potassium were within normal range. Citric acid was determined in 28 patients only. 22 patients had an excretion rate below 300 mg/24 h.

56 stones were recovered and analysed from the 52 patients (Table 5). Out of these, 54 stones had a mixed composition. Composition of the 2 pure stones, from the same patient, was calcium phosphate (renal) and calcium carbonate (vesical). Calcium was present in all the stones. Oxalate and phosphate were present in 92.8% and 94.6% of the stones respectively.

Discussion

There is a body of evidence in favour of pluricausal nature of urinary calculus disease in developing countries especially the Asian Continent, including India [2, 6]. There is also a number of reports to indicate that the etiology of the disease may differ from one population to the other [10]. Our previous work has supported this view [7, 8, 9]. The present data which provide a comprehensive view of the metabolic profile of 52 stone formers further confirms this belief.

The presence of mixed stones in the 51 patients out of 52, suggests a multifactorial etiology. The presence of magnesium ammonium phosphate in 78.2% stones and of infection in 67.3% patients indicated the importance of bacteriuria as an etiological factor. The significance of nutritional imbalance was indicated by the lack of animal protein, the abundance of cereals, and the frequent consumption of oxalate rich vegetables and the regular consumption of tea.

These patients did not show any signs of obstructive uropathy, hyperparathyrodidism or any other condition which predisposes to calculus disease. None of the patients was taking any drugs implicated in the genesis of urolithiasis.

Crystalluria, present in 67.3% of the patients was another important finding. Moderate to severe (2+ or more) crystalluria in these patients revealed a state of supersaturation in their urine.

Hyperoxaluria of endogenous origin was a common finding. Mild to moderate hyperoxaluria was present in 38.5% patients; 15.4% had excretion rate between 40 and 50 mg/day and 23.1% had an excretion rate of > 50 mg/day.

We consider that an excretion rate of >200 mgCa^{++}/24 h is diagnostic of hypercalciuria in the local population as our unpublished data as well as previous data [1, 13] indicate that calcium intake is quite low here. On the basis of this criterion 25% patients were hypercalciuric. In 13.5% of the patients hypercalciuria was associated with hyperoxaluria. Phosphates, uric acid and pH do not appear to be significant risk factors in stone formation in the local population. It is therefore felt that hypercalciuria could be a risk factor only in a small percentage of cases.

Amongst the urinary stone inhibitor substances magnesium and citric acid appear to be important etiological factors. Magnesium excretion was below normal in all 52 stone formers and citric acid excretion was low in 78.6% of the patients.

Sodium and potassium excretion were generally within normal range suggesting their non-involvement in the stone forming process. Interestingly both reduced [4] as well as the increased [11] excretion of sodium has been suggested to enhance the chances of stone formation.

References

1. Colobawalla BN (1971) Incidence of urolithiasis in India. ICMR Tech Rep Series No 8, pp 42–51
2. Gangwal KC (1971) Current concepts on etiology of urolithiasis. ICMR Tech Rep Series No 8, pp 58–85
3. Hodgkinson A, Williams A (1972) An important colorimetric procedure for urine oxalate. Clin Chem Acta 36:127–132
4. Modlin M (1967) The etiology of renal stone. A new concept arising from studies on a stone free population. Ann R Coll Surg 40:155–158
5. Natelson S (1971) Techniques of clinical chemistry, 3rd ed. Charles C Thomas, USA, p 296, 491, 576 and 741
6. Pendse AK (1985) Urolithiasis in Udaipur and Jodhpur. A comparative study on prevalence and urinary profile. Bull III, Ann Conf Urol Soc India, p 12
7. Pendse AK, Ghosh R, Goyal A, Singh PP (1984) Effect of indigenous drugs on idiopathic hyperoxaluria in stone formers. Asian Med J 27:136–140
8. Pendse AK, Purohit AK, Ghosh R, Goyal A, Singh PP (1984) The effect of ingestion of megadoses of ascorbic acid on urinary oxalate excretion in normal subjects and stone formers. Urolithiasis and related clinical research. Plenum Pub Corp, pp 225 to 228
9. Pendse AK, Srivastava AK, Kumawat JL, Goyal A, Ghosh R, Sharma HS, Singh PP (1984) Urolithiasis in Udaipur (Rajasthan) Indian Med Assoc 82:151–156
10. Pyrah LN (1979) In: Renal calculus. Springer, pp 3–16
11. Silver J, Rubinger D, Friedlaender MM, Popovtzer MM (1983) Sodium dependent idiopathic hypercalciuria in renal stone formers. Lancet II:484–486
12. Singh PP, Pendse AK (1984) Urolithiasis in Rajasthan (Western region of India): Urinary excretion of calcium and oxalate in normal population and stone formers. Urol Res 12:60
13. Singh PP, Kothari LK, Sharma DC, Saxena SN (1972) Nutritional value of foods in relation to oxalic acid content. Ann J Clin Nutr 25:1147–1152
14. Singh PP, Pendse AK, Goyal A, Ghosh R, Shrivsatava AK, Kumawat JL (1983) Blood and urine chemistry of stone formers in local population and evaluation of cystone treatment. Indian Drugs 20:264–280
15. Singh PP, Pendse AK, Jain AK (1984) Urolithiasis in Southern Rajasthan: Contribution of dietary oxalate to urinary oxalate. Urolithiasis and related clinical research. Plenum Pub Corp, pp 77–80
16. Van Reen R, Valyasevi A (1974) Idiopathic bladder stone disease. Public Health Rev 3:57–71
17. Varley H, Gowenlock AH, Maurice B (1980) Practical clinical biochemistry. 5th ed. Heinemann, London p 856

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