



ROLE OF DIET, CLIMATE AND INFECTION IN RENAL STONE FORMATION

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ABSTRACT

Urinary stone may be of calcium oxalate, calcium phosphate, uric acid and cysteine. About 70-80% of renal stones in western countries are composed of calcium oxalate alone or mixed with calcium phosphate in men. Study is designed to analyze the renal stones and its relationship with diet and other factors. It was observed that calcium was the major ion that formed salts with phosphate, oxalate, carbonate in most cases of male and female patients. Uric acid calculi were observed only in 1% cases. Microbiological examination showed that *E.coli* and *proteus* were more common as compared to *pseudomonas* and *Klebsilla*. It was observed that diet, hot climate and urinary tract infections due to *E. coli* and *proteus* play an important role in the formation of renal calculi.

Key Words: calcium, renal stone

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INTRODUCTION

Renal calculi or stones can form in urine where it is supersaturated with crystalloid component of the calculus¹. Stone may cause renal damage and this is often progressive; renal function tests show deterioration of kidney functions².

Factors predisposing to stone formation are dehydration, urinary tract infection, alkaline urine, hypercalcemia, hyperuricosuria, hyperoxaluria and lack of urinary inhibitors of crystallization^{3,4}. People living or working in hot climate are liable to become dehydrated and show a greater tendency to form renal stones².

Urinary calculi are crystalloids embedded in a binding substance of mucus and protein with inclusion of bacteria and epithelial cells. Calcium forms insoluble salts with oxalic acid and these salts precipitate as crystals appear in urine. In some persons crystalluria leads to stone formation. The crystals aggregate on the surface of renal papillae, enlarge, some times break away, and migrate to ureter and cause renal colic. It is possible that mucoprotein secreted by distal tubule has a role in causing aggregation of crystals and their attachment to the collection duct⁶.

Urinary stone may be of calcium oxalate, calcium phosphate, uric acid and cysteine. About 70-80% of

renal stones in western countries are composed of calcium oxalate alone or mixed with calcium phosphate in men. It is more common in middle age⁵. Stones in the upper renal tract have been shown to occur in 5-10% of adults in Western Europe and United States⁷.

Different forms of calcium salts are observed like calcium oxalate (precipitate at acidic pH), calcium phosphate (precipitate at pH 6.0-6.5). Less commonly, uric acid which is not very soluble, will crystallize at low pH (5.3) and form stones⁶.

The type of stones formed depends mainly on the composition of urine, which, in turn reflects the type of diet consumed⁸. In countries where there is also a high intake of oxalate from local leaves and vegetables, beverages, nuts and citrus fruits, urinary oxalate is increased and as a result, the ammonium acid urate stones often contain calcium oxalate as well^{3,7}. Hyperoxaluria leads to increased calcium oxalate supersaturation and calcium oxalate stone formation. Excess oxalate can rise from endogenous overproduction as in primary hyperoxaluria or from dietary sources⁹.

About 40% of patients with calcium stones will have hypercalciuria. Urinary calcium level of >300mg/dl in males and 250mg/dl in females is regarded as excessive, when the patient is receiving a diet containing 1 gram of calcium/day. About 10 grams of calcium is filtered by the kidney/day and a small fraction is reabsorbed by renal tubular cell. Increased calcium is due to an increase in intestinal calcium absorption. It also may be due to lack of appropriate renal tubular reabsorption of calcium or loss of calcium from bone or a combination of both¹⁰.

Excessive excretion of uric acid relates to excessive dietary intake of purine (liver, beans, fish, meat)³. This

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study was therefore designed to analyze renal stones and their relationship with diet and other factors.

MATERIALS AND METHODS

Thirty seven male and thirty eight female patients were operated in Surgical Ward of Sir Ganga Ram Hospital Lahore. Their stone analysis included estimation of calcium, phosphate, oxalate, carbonate and uric acid using qualitative means.

RESULTS

Among seventy five cases in surgical unit of Sir Ganga Ram Hospital, Lahore thirty seven were males and thirty eight females. Most of the stones had color from off white to different shades of brown color. Their number varied from 2-4 to numerous with small size. All these stones were received after operation i.e. without using laser technology. It was observed that prevalence rate of stone formation was same in both male and female patients and it was more common in middle age. Most of the patients belonged to middle and low socioeconomic class (data not shown).

Table 1 shows the percentages of calcium, phosphate, oxalate, carbonates and uric acid found in the stone. It was observed that most of the stone mainly consisted of calcium, phosphate and oxalate up to 3% in some 1% in others. It was also observed that in some cases stone consisted of all the ions and these formed the salts of calcium phosphate, calcium oxalate and calcium carbonate (mixed salts). On the other hand some stones only consisted of salts of calcium oxalate and some consisted of calcium phosphate or calcium carbonate. It was observed that the presence of uric acid was not very common in stones.

DISCUSSION

The overall risk of stone formation is considered to be a balance between the degree of supersaturation of urine

with the ionic salts and the inhibitory activity of urine represented by the concentration of acid mucopolysaccharides, pyrophosphate and other inhibitors⁵.

The present study observed stone formation on the basis of gender and no difference was noted. It was observed that most of the patients belonged to middle and lower socioeconomic class and their diet mostly consisted of vegetables with some meat. Use of milk is very common. It was reported by Williams DL⁵ that dietary intake of calcium, oxalate and purine was highly relevant to the enhanced excretion of calcium.

Recently it has been reported by Abrahams HM⁴ that the presence of infectious stones is still a cause of morbidity and mortality. Our study observed that most of the organisms found in renal stones were E. coli and then proteus. It has been reported by Varley H¹¹ that when infection occurs in the urinary tract, urease producing organisms are common and convert urea into ammonium carbonate.

It was observed that most of the renal stones were found in upper urinary tract. Urine volume was below normal and pH was ~ 5.0 (acidic). Reduction in urinary volume and pH was also observed¹². They reported that this reduction in urinary volume and pH may contribute to increased stone formation risk especially of calcium.

The present study observed that the main ion present in renal stones was calcium which was in combination with oxalate, phosphate and carbonate rather than the free form. New avenues of research¹³ are beginning to explore the specific defects that may contribute to hypercalciuria. From such studies, an understanding of the role of certain dietary excesses as contributors to the development of hypercalciuria and, in some cases, attendant bone loss, is being achieved. The contribution of genetics to hypercalciuria has provided a powerful means of identifying genes that contribute to the hypercalciuric phenotype in a number of hypercalciuric conditions.

The incidence of uric acid stones varies between countries and accounts for 5% to 40% of all urinary calculi¹⁴. In this study the incidence rate was only 1%.

Excessive excretion of uric acid relates to excessive dietary intake of purine (liver, beans, fish, nuts). Increased bone resorption and the attendant hypercalciuria and hyperphosphaturia contribute significantly to raising the urinary state of saturation with respect to the calcium salts, namely calcium oxalate, and calcium phosphate. In addition are other environmental and dietary factors^{12,15}.

Table 1: Renal stone analysis of male and female patients.

Parameters	Male (37)	Female (38)
Calcium	22 having 3% 02 having 1% 13 nil	18 having 3% 03 having 1% 07 nil
Phosphate	19 having 3% 02 having 1% 16 nil	19 having 3% 11 having trace 08 nil
Oxalate	18 having 3% 09 having 1% 10 nil	18 having 3% 07 having 2% 13 nil
Carbonates	15 having 3% 07 having 1% 15 nil	18 having 3% 04 having 2% 16 nil
Uric acid	05 having 3% 32 nil	09 having 3% 29 nil



CONCLUSION

It was observed that diet, hot climate and urinary tract infection due to E.coli and proteus play an important role in the formation of renal calculus.

A discussion of other possible causes in association is offered, but it is felt that, in the end, more research into this area is required.

REFERENCES

1. Hess B. Nutritional aspects of stone disease. *Endocrinol Metab Clin North Am* 2002;31(4):1017-30.
2. Whitby LC, Percy-Robb IW, Smith AF. Lecture notes on Clinical Chemistry. 3rd ed. Blackwell Scientific Publication, London, 1987
3. Marshall WJ. Illustrated Text Book of Clinical Chem. 2nd ed. London: Mosby; 1993. p.213
4. Abrahams HM, Stoller ML Infection and urinary stones. *Curr Opin Urol* 2003;13(1):63-7.
5. Williams DL, Marks V. Biochem in Clinical Practice. New Delhi: CBS Publishers and Distribution; 1990 .p.432.
6. Bauer JD. Clinical Laboratory Methods. 9th ed. The CV. ST Loius: Mosby; 1982.p.321.
7. Robertson WG. Renal stones in the tropics. *Semin Nephrol* 2003, 23:77-87
8. Tiselius HG. Medical evaluation of nephrolithiasis. *Endocrinol Metab Clin North Am* 2002;31(4):1031-50.
9. Asplin JR. Hyperoxaluric calcium nephrolithiasis. *Endocrinol Metab Clin North Am* 2002;31(4):927-49.
10. Henry JB. Clinical Diagnosis and Management by Laboratory Methods. 17th ed. Philadelphia: WB Saunders; 1989.p.213.
11. Varley H, Gowenlock AH, Bell M. Practical Clinical Biochem. 5th ed. William Heinmann Medical Books Ltd. 1989.p.43.
12. Zerwekh JE. Nutrition and renal stone disease in space. *Nutrition* 2002;18(10):857-63.
13. Zerwekh JE, Reed-Gitomer BY, Pak CY. Pathogenesis of hypercalciuric nephrolithiasis. *Endocrinol Metab Clin North Am* 2002;31(4):869-84.
14. Shekarriz B, Stoller ML. Uric acid nephrolithiasis: current concepts and controversies. *J Urol* 2002;168 (4): 1307-14.
15. Sorensen CM, Chandhoke PS Hyperuricosuric calcium nephrolithiasis. *Endocrinol Metab Clin North Am.* 2002;31(4):915-25.