## DEFINITION

1. A urinary calculus or stone consists of a nucleus of organic material around which urinary salts are deposited in concentric layers. These layers are bound together by a matrix of organic matter.

2. Urinary calculus is to be distinguished from calcific deposits in the substance of the kidney at sites of previous inflammation or degeneration, such deposits being designated **nephro-calcinosis**.

3. A **renal calculus** is one which forms in the pelvis or calyces of the kidney.

4. A **vesical calculus** is one which forms in the bladder.

5. Renal calculi may be single or multiple, may remain in the kidney, may become lodged in the ureter or may pass down into the bladder to become a vesical calculus.

## TYPES

6. **Oxalate** calculus consists of calcium oxalate and is popularly known as the **mulberry stone**, being covered with sharp projections. These projections cause the kidney to bleed and altered blood is precipitated on the stone. Such stones are usually single and are extremely hard.

7. **Phosphatic** calculus consists of calcium phosphate but this may be combined with ammonium magnesium phosphate and, rarely, composed of the latter only. In an alkaline urine, it grows rapidly and may fill the renal calyces, taking on their shape and then being known as **Staghorn** calculus. These stones are smooth, soft and crumble easily.

8. **Uric acid and urate** calculus is hard, smooth and, because it is uncommonly found singly, is typically faceted.

9. **Cystine** calculus only occurs in the urinary tract of those with **Cystinuria**, a genetic disorder affecting renal and intestinal handling of lysine, arginine, ornithine and cystine. Such stones are usually soft, multiple, may assume a cast of the renal pelvis and calyces and only appear in acid urine.

10. A combination of any of the above may be found in one calculus.

## CLINICAL MANIFESTATIONS

11. The position, size and mobility of a calculus influence the manifestations.

12. A calculus which is static in the renal pelvis or calyces may not give rise to any symptoms and may only be found co-incidentally on routine radiological examination.

13. A mobile calculus in the renal pelvis may give rise to lumbar pain and haematuria both of which are made worse by exercise or jolting and are relieved by rest.

14. A calculus which enters the ureter may give rise to an attack of renal colic which is characterized by intense pain, usually accompanied by vomiting. If the calculus remains, either temporarily or permanently, in the ureter, repeated intermittent attacks of colic may occur and hydro-ureter, hydro-nephrosis or atrophy of the kidney parenchyma may supervene due to back pressure of urine.

15. Vesical calculus gives rise to increased frequency of micturition and pain in the lower abdomen, particularly on movement and on emptying the bladder.

**AETIOLOGY**

16. The formation of calculi is a complex one and it is surprizing that they do not occur more frequently since some of their constituents are normally present in urine in a concentration in excess of their maximum solubility in water.

17. However, urine contains glycoaminoglycosans, pyrophosphate and citrate, substances which, by forming complexes with the insoluble salts which are stone-forming, may keep those salts in solution in the urine.

18. Thus, any alteration in the urinary concentration of stone-forming constituents can lead to stone formation. Such alteration can occur from -

    18.1 too little urine output.

    18.2 an absolute increase in the amount of a stone constituent excreted over a period of time.

    18.3 a reduction in the concentration of inhibitors of stone formation.

    18.4 stasis in the flow of urine leading to embryonic stones remaining in the kidney for longer than they would otherwise remain if the flow was normal.

19. Factors which can have any of the effects listed at paragraph 18 above are -

    19.1 **Dietetic**. A deficiency of **vitamin A** causes a desquamation of epithelium. The desquamated cells in the kidney form a nidus around which the stone is deposited.

    19.2 **Altered urinary solutes and colloids**. In hot climates, the concentration of solutes will rise. Thus, prolonged residence in hot climates coupled with insufficient fluid intake and excess sweating may cause precipitation of crystalloids from urine containing sufficient colloids to keep them in solution only when the urine is abundant and dilute.

    19.3 **Renal infection**. This favours the formation of calculi especially if the infecting organism is a urea-splitting streptococcus staphylococcus or proteus.

    19.4 **Inadequate urinary drainage**. Any condition which causes an obstruction to the free flow of urine may lead to stone formation.

19.5 **Prolonged immobilisation**. This, whatever the cause, may lead to skeletal decalcification and increased output of calcium in the urine. This, together with the mechanical effects of recumbency on renal drainage, favours the deposition of calcium phosphate stones.

19.6 **Hypercalciuria** may result from -

19.6.1 enhanced absorption (and thus excretion) from dietary sources such as excessive intake of milk and other dairy products, excess intake of vitamin D or from altered vitamin D metabolism in sarcoidosis.

19.6.2 **Hyperparathyroidism**. Although rare, this condition results in a great increase in the elimination of calcium in the urine and thus an increased risk of stone formation. Patients suffering from hyperparathyroidism often present with renal stones.

19.6.3 impaired renal re-absorption due to constitutionally-determined factors. This form is often referred to as **idiopathic hypercalciuria**.

19.6.4 certain studies have shown that there is a clinical relationship between hypertension (both treated and untreated) and urinary calculus formation. These studies have also found that there is hypercalciuria in hypertensive subjects. It has been hypothesized that the hypercalciuria occurs as a "renal leak" - ie failure of re-absorption and that this causes stone formation. However, as has been remarked in the latest study (Capuccio et al), cause and effect relations are not proved by statistical associations and it is possible that, in some of their cases, renal damage caused by stones led to the hypertension rather than the other way around.

19.7 **Idiopathic stone diathesis**. In less than 5% of those with calculi, no physiological abnormality can be detected and the cause for stone formation in these patients remains unknown.

20. Although stone formation is found in association with **renal structural abnormalities** (such as ectopic kidney, polycystic kidney and horseshoe kidney), there is no convincing evidence that the abnormality itself causes stone formation, the expert view being that such stones form secondarily to urinary tract infection (which is common with such abnormalities).

## CONCLUSION

21. Urinary calculi result from precipitation of excess crystalloids in the urine. Factors which influence calculus formation are listed at paragraph 19 above.

## REFERENCES

Mann C V and Russell R C G (Eds). Bailey and Love's Short Practice of Surgery. 21st Ed. 1992. London. Chapman & Hall Medical. p1336-1350.

Pak C Y C. Renal Calculi. In: Wyngaarden J B. Smith L H and Bennett J C (Eds.). Cecil Textbook of Medicine. Philadelphia. W B Saunders Company. 19th Ed. 1992. p603-608.

Cappuccio Francesco P, Strazzullo Pasquale and Mancini Mario. Kidney Stones and hypertension: population study of an independent clinical association. British Medical Journal 1990;300:1234-6.

Davison A M and Lambie A T. Diseases of the kidney and genito-urinary system - Renal and Vesical Calculi and nephrocalcinosis. In: (Eds) Edwards Christopher R W, Bouchier Ian A D. Davidson's Principles and Practice of Medicine. 16th Ed. 1991. Edinburgh. Churchill Livingstone. p594-597.

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