URINARY CALCULUS IN THE INDIAN AND AFRICAN IN NATAL

THEUNIS COETZEE, *Department of Surgery, Edendale Hospital, Pietermaritzburg*

Many observers have in the past pointed out the rarity of calculus of the urinary tract as a disease in our African population. In the Non-European Hospital in Johannesburg, Vermooten[1] found records of only 1 case in 91,000 admissions during the period 1922 to 1935. This was an African woman and not a proved case. In Durban, Wise and Kark[2] analysed the records of 483,450 admissions to the African wards of King Edward VIII Hospital during the period 1951 to 1959. They found 7 cases of urinary calculi in locally-born African males. This incidence was compared with that amongst Indian patients, where 12 cases of proved calculi were found in 9,600 admissions. They pointed out that, of the 7 African cases, the calculus could be regarded as primary in only 1 patient; in the other 6 patients it was associated with prolonged recumbency, urinary-tract obstruction, or bilharziasis.

It was therefore considered worth while to review the cases diagnosed as urinary calculus at Edendale Hospital from June 1954 to June 1962 (a period of 8 years), to compare the incidence in Indian and African patients, to

study associated pathology of the urinary tract, and to discuss the possible reasons for rarity of urinary calculus in the African population.

Edendale Hospital Statistics

During the 8-year period 162,449 African patients were admitted to the wards of Edendale Hospital. The number of Indian patients for the same period was 11,549, and the ratio of African to Indian patients 14:1.

A definite diagnosis of urinary calculus was made in 40 patients during this period. Among Indian patients, 19 proved cases of calculus occurred. This represents an incidence of 0·164%. The incidence amongst Africans (21 proved cases) was 0·0129%, and the Indian: African incidence ratio was 12·7:1. Table I summarizes the incidence in the two racial groups and in the two sexes, while Table II shows the racial and sex distribution of the calculi according to their localization at the time of diagnosis. The cases are further subdivided according to whether the calculus could be regarded as primary, or whether predisposing causes were present. In no instance was a diagnosis of hyperparathyroidism established. Local pathology of the urinary tract, however, constituted a frequent predisposing factor. In Table III the cases are grouped according to whether the calculi were considered 'primary' or 'secondary', and the figures are summarized in Table IV. It will be seen that, in the African group, only 3 out of 21 cases were primary calculi, whereas 10 of 19 calculi amongst Indian patients could be so classified. If only primary calculi are considered, the incidence figure for Indians (10 cases) is 0·0865% and for Africans (3 cases) 0·00184%, while the incidence ratio is 47:1. The predominant occurrence of primary calculi in the upper urinary tract and of secondary calculi in the bladder is clearly shown in Table V where renal and ureteric calculi are grouped together under 'upper tract'.

TABLE I. CALCULUS CASES BY RACE AND SEX

	Male	*Female*	*Total*
Indian	15	4	19
African	11	10	21

TABLE II. CASES BY RACE, SEX AND LOCALIZATION

	Kidney		*Ureter*		*Bladder*	
	M	F	M	F	M	F
Indian ..	5	2	7	1	3	1
	7		8		4	
African ..	3	0	1	1	7	9
	3		2		16	

TABLE III. PRIMARY AND SECONDARY CASES BY RACE, SEX AND LOCALIZATION

	Kidney				*Ureter*				*Bladder*			
	Pri.		*Sec.*		*Pri.*		*Sec.*		*Pri.*		*Sec.*	
	M	F	M	F	M	F	M	F	M	F	M	F
Indian ..	2	1	3	1	6	1	1	0	0	0	3	1
African ..	0	0	3	0	1	1	0	0	1	0	6	9

TABLE IV. PRIMARY AND SECONDARY CASES BY RACE AND SEX

		Indian		*African*	
Primary	M	8	10	2	3
	F	2		1	
Secondary	M	7	9	9	18
	F	2		9	
			19		21

TABLE V. PRIMARY AND SECONDARY CASES BY RACE AND LOCALIZATION

	Indian		*African*	
	Pri.	*Sec.*	*Pri.*	*Sec.*
Upper tract	10	5	2	3
Lower tract	0	4	1	15

When the predisposing causes in the urinary tract are studied, the following conditions are found:

Indian Patients (9)

Upper tract (5) — pelvic-ureteric stenosis (3)
ureteric stricture (1)
bladder-neck contracture (1)

Lower tract (4) — bladder-neck contracture (2)
bilharziasis with papillomatous change (1)
long-standing vesico-vaginal fistula (1)

African Patients (18)

Upper tract (3) — chronic epileptic (confined to bed) (1)
prolonged recumbency (1)
malignant ileo-vesical fistula (1)

Lower tract (15) — long-standing vesico-vaginal fistula (5)
squamous carcinoma of bladder (2)
prolonged recumbency (5)
bladder-neck obstruction (2)
carcinoma of cervix (1)

It would appear that infection and obstruction are by far the most important predisposing conditions. Where paraplegia, prolonged recumbency, prolonged bladder drainage, etc. had existed, it was generally the associated urinary infection that constituted the decisive factor. Bilharziasis was not an important associated condition. It is probably true to say that, having in mind the very high incidence of lower urinary-tract infection and the large number of patients confined to bed for long periods on account of tuberculous bone disease and spinal cord injury, the incidence of secondary urinary calculi could be regarded as very low.

The much larger number of cases diagnosed as urinary calculus among our African admissions, as compared with that found in reports from elsewhere, could be explained in two ways. Firstly, with the urbanization of Africans, change in diet, or for other reasons, calculus may have become more frequent than it was 25 years ago. This, however, would not explain the discrepancy between our figures (1954 - 62) and those from King Edward VIII Hospital (1951 - 59), where the Edendale Hospital figures show a nine-fold higher incidence. A second possibility is that a certain number of cases with urinary calculus might have been missed in other studies. This would be quite likely to happen if the hospital's diagnostic index constituted the sole reference. Patients with fracture-dislocation of the spine, vesico-vaginal fistula, etc. are almost always discharged with this primary disease as the only 'final diagnosis', with no mention of the complicating calculus disease. In the series here reported, at least 10 of the 18 secondary calculi occurred in the orthopaedic and gynaecological wards, and records of these cases were available largely on the basis of personal interest in the problem.

The Edendale figures support the impression that urinary calculi are rare in the African population in Natal.

Before going on to discuss the possible factors underlying this low incidence of stone in the African, it might be interesting to review briefly the problem of calculus formation in the urinary tract in broad outline.[3]

Causation of Urinary Calculi in general

As the vast majority of stones in the urinary tract have calcium salts as their main constituent, it is, basically, a failure in the transport of calcium that leads to stone formation. Failure of calcium transport could, in theory, arise under the following circumstances:

A. Excess of calcium to be transported. This is most convincingly seen in hyperparathyroidism, and is probably a factor in prolonged decubitus. However, as many of these patients do not develop urinary calculi, excessive urinary calcium cannot be the main factor.

B. Breakdown of the mechanisms which normally keep calcium in solution. This could operate by:

1. A deficiency of some substance(s) in the urine that normally keep calcium in solution, either by forming a soluble complex with it or by adsorption. The substances that have been suggested are:

(a) Citrates, capable of forming a non-ionized complex with calcium and so avoiding its precipitation. Citrates are destroyed by certain organisms.
(b) Urinary colloids. These absorb crystalloids and so prevent their precipitation. It has been suggested that the urinary colloid excretion might be increased by the administration of hyaluronidase.
(c) Certain organic acids, e.g. glucuronic and ascorbic acids, capable of holding calcium carbonate and phosphate in solution. Glucuronic acid excretion increases after administration of acetylsalicylic acid.
(d) Amino acids. Some patients with urinary calculus show a low excretion of amino acids in the urine. Amino acids may form soluble chelates with calcium.
(e) The presence of urea, and a low-magnesium diet, appear to increase the solubility of calcium.

2. An excess of some substance in the urine that assists the precipitation of calcium salts. An increased excretion of mucoprotein has been demonstrated in patients with urinary calculi. This substance binds calcium to form an insoluble complex. The matrix of calculi appears to consist of muco-polysaccharides, and the increased amount of muco-polysaccharide around and in the cells of the renal tubules suggests the possibility that stone formation may be a disorder of muco-polysaccharide metabolism.

3. A disturbance in the mechanism which normally clears the kidney of precipitated calcium—the plaques of Randall and the microliths described by Anderson and McDonald.

In practice, the following factors have been thought to be important in the formation of urinary calculus, as they are frequently associated:

1. The reaction of the urine. Alteration of the urinary pH (e.g. alkalinization by urea-splitting organisms or by administration of alkalis for peptic ulcer) leads to an alteration in the solubility of various calcium compounds.

2. Infection. This would appear to be of special importance in recurrent calculi.

3. Urinary stasis, with which infection may be associated.

4. Environment and diet—excessive sweating, concentrated urine and excessive calcium content of drinking water. There is no evidence that deficiency of vitamin A is important in stone formation in the human being.

5. Foreign bodies, only occasionally.

In summary it could reasonably be accepted that calculus formation in the urinary tract depends on some or all of the following factors:[4]

(*a*) The failure to maintain a balance between calcium-dissolving and calcium-precipitating factors

(*b*) The matrix substance

(*c*) The volume of urine relative to the amount of calcium to be excreted

(*d*) The calcium content of the diet, recognizing though that this is not directly proportional to the urinary calcium, since calcium absorption in the intestine varies.

Why is Urinary Calculus rare in the African?

In considering, on the basis of the foregoing, the possible reasons for the rarity of stone in the African, the following points might be significant:

1. No evidence of racial immunity or predisposition has been uncovered.

2. The African habitually takes a diet with a high carbohydrate content. Such a diet is associated with a high urinary output and an acid ash residue.

3. The African appears to have a low serum calcium. This may be the result of a smaller dietary intake of calcium, a lower intestinal absorption of calcium (possibly due to intestinal hurry or vitamin-D deficiency), or a low level of serum proteins (on which the serum-calcium level is partly dependent).

4. There may be a low mucoprotein level in the serum of Africans.

5. The African may have a higher urinary excretion of citrates. It has been suggested that this is the result of a high oestrogen level—this being an outcome of liver damage with failure of oestrogen katabolism.

6. Undernutrition, generally, does not appear to be a responsible factor.[5]

Any or all of these factors may operate to make urinary calculus a rare condition in the African. There may be

other factors that have not yet been discovered. Whatever they are, they appear to more than compensate for the predisposing effects of lower urinary-tract infection, urinary obstruction, and enforced prolonged recumbency, so frequent in the African patient.

REFERENCES

1. Vermooten, V. (1937): J. Amer. Med. Assoc., **109**, 857.
2. Wise, R. O. and Kark, A. E. (1961): S. Afr. Med. J., **35**, 47.
3. Jamieson, R. A. and Kay, A. W. (1959): *A Textbook of Surgical Physiology*, Edinburgh: E. & S. Livingstone.
4. McGeown, M. G. and Bull, G. M. (1957): Brit. Med. Bull., **13**, 53.
5. Walker, A. R. P. (1961): S. Afr. Med. J., **35**, 187.