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Reduction of calcium excretion in the stone-forming kidney in unilateral ureteral obstruction

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Summary. Thirteen urolithiasis patients with unilateral obstructive uropathy were treated with percutaneous nephrostomy (PCN) either for urinary diversion, endopyelotomy, nephrolithtotmy or chemolysis. After percutaneous nephrostomy, the indvidual urine volume, creatinine clearance (Ccr), urinary absolute and fractional excretions of sodium, potassium, calcium, magnesium and inorganic phosphate were measured separately in timed urine collections from a pigtail catheter and from the urethra. The data showed that Ccr and the absolute urinary excretions of sodium, potassium, calcium, magnesium and inorganic phosphate were significantly lower in the PCN kidney immediately or 2 days after relief of obstruction. The ratio of total urinary calcium excretion to urinary creatinine excretion in the obstructed kidney was significantly greater than that in the contralateral kidney. The fractional excretions of calcium and magnesium increased as renal function decreased. The results showed that when the total Ccr is below normal, the apparent excretion of urinary calcium will be underestimated. However, when the total Ccr of patients is within normal range, hypercalciuria may be detected adequately and thus favors early implementation of an appropriate therapeutic strategy.

Key words: Urolthiasis – Obstructive uropathy – Percutaneous nephrostomy – Hypercalciuria – Creatinine clearance

Hypercalciuria is one of the important factors predisposing to stone formation [4, 6, 8, 9, 14]. Thus, an accurate diagnosis and prompt treatment of hypercalciuria may be helpful for prevention of urinary calculi recurrence [10]. However, calcium excretion is substantially influenced by kidney function [1, 3, 11, 12]. When they are first seen by a urologist, some patients already exhibit urinary calculi with ipsilateral renal damage, although their global renal function may still be within the normal range. This suggests that the total urinary output of calcium may not reflect accurately the excretory function of the individual, affected kidney. Clinically, in order to eliminate the confounding effect of surgery, urinary tract infection, and obstructive uropathy on urinary calcium excretion, urolithiasis patients with these complications are usually temporarily excluded from the metabolic evaluation [13]. Thus, little information is available about the actual relationship between obstructive uropathy and urinary calcium excretion and the pathophysiology of renal tubular handling of electrolytes after release of chronic unilateral ureteral obstruction in man. In the present study, we aimed at assessing the effect of obstructive uropathy on calcium, magnesium, and phosphate excretion in the stone-forming kidney and in determining the relationship between the separate renal function and calcium excretion in these patients.

Materials and methods

The present study included 13 patients (9 men and 4 women), ranging in age from 17 to 82 years (average: 48.2). They had all presented with recurrent urinary calculi. Five patients had been operated on before this admission. The radiological investigations included excretory urography and ultrasonography of kidneys in all 13 cases and retrograde pyelography in 8 of the total cases. Ten patients had severe hydronephrosis and three had moderate hydronephrosis (Table 1). The degree of hydronephrosis was defined by IVP and ultrasonography as previously described [5]: severe: sac-like collecting system and markedly thin parenchyma; moderate: calices ballooned outward and papillae barely visible. Before any type of surgical intervention, each patient underwent metabolic evaluation which included SMA-20 blood chemistry, serum magnesium, parathromone and osteocalcin levels. None of our patients had primary hyperparathyroidism, hyper- or hypocalcemia, vitamin D deficiency or intoxication, or any other obvious complications that might influence renal calcium excretion. After completion of the measurements, sonoguided percutanoues nephrostomies (PCN) using a pigtail catheter were performed routinely on the kidney with the calculi either for urinary diversion, endopyelotomy or percutaneous nephrolithotomy. During the PCN procedure, antegrade pyelography was conducted in each patient, and the results were consistent with IVP and kidney sonography.

Table 1. General data

Patient	Sex/age	Diagnosis/stone size (mm)	Hydronephrosis[a]	Serum Cr/BUN (mg/dl)
1	F/53	Ureteral stone L/3, rt (10×10)	Severe	1.9/24
2	M/46	UPJ stone, lt (45×36)	Severe	1.1/10
3	F/19	UPJ stone, rt	Severe	0.7/11
4	F/17	Staghorn renal stone, rt	Moderate	1.7/15
5	M/82	Ureteral stone U/3, lt (20×20)	Severe	2.1/27
6	M/32	UPJ stenosis and stone, lt (15×10)	Severe	1.1/19
7	M/38	UPJ stone, rt (30×30)	Moderate	1.2/20
8	M/59	UPJ stenosis and Renal stone, lt (15×10)	Severe	1.3/14
9	M/42	UPJ stone, rt (25×25)	Moderate	1.4/16
10	M/63	Ureteral stone U/3, rt (15×10)	Severe	1.2/20
11	F/50	Ureteral stone U/3, lt (10×10)	Severe	1.8/24
12	M/44	Ureteal stone U/3, rt (20×25)	Severe	1.8/22
13	M/82	UPJ stone, lt (30×25)	Severe	1.8/28

[a] Hydronephrosis was diagnosed by IVP and sonography; Cr, Creatinine; L/3, lower third; UPJ, ureteropelvic junction; U/3, upper third

The study was performed in two parts. In the first part, the 24-h urine samples of 11 patients were collected 2 days after PCN separately from the pigtail catheter and the urethra to determine the individual kidney excretions of urine volume, calcium, magnesium, inorganic phosphate, creatinine clearance (Ccr), calcium/creatinine ratio and fractional excretion of calcium, magnesium and inorganic phosphate. In the second part, the urine samples of two patients were collected for the measurement of individual urine volume, Ccr, absolute and fractional excretion of sodium, potassium, calcium, magnesium, and inorganic phosphate, on 2, 4, 6, 8 and 24 h after PCN. All urine samples were stored in an acid-washed plastic bottle containing a small amount of concentrated hydrochloric acid as a preservative.

The creatinine concentrations of plasma and urine were measured by the Rate Jaffe reaction (ASTRA 8, Beckman Instrument, USA). The urinary sodium and potassium were measured by ASTRA 8 Autoanalyzer (Beckman Instrument, USA). The urinary calcium, magnesium and inorganic phosphate were measured by colorimetric titration (Paramax Autoanalyzer, Baxter, USA). The urinary oxalate was determined by the Sigma commercial colorimetric kit. Calculation of Ccr was based on the clearance equation: $Ccr = Ucr \cdot V/Pcr$. The serum parathromone were detected with N-tact PTH IRMA kits (INCSTAR Corp., Stillwater, Minnesota) for intact PTH. Serum osteocalcin was measured by INCSTAR ^{125}I Osteocalcin RIA kits.

Data are presented as mean ± SD. The Pearson product-moment correlation method was used to determine the correlation coefficient (r value). Differences between groups or kidneys were analyzed statistically by Student's t-test.

Results

Among the 12 stone specimens available, 5 were whewellite, 6 were a whewellite and carbonate apatite mixture, 1 was a uric acid and whewellite mixture. Patient 11 was diagnosed as having a uric acid stone. Only THAM chemolysis via PCN was used; no stone specimen was obtained in this patient.

Table 2 shows the serum concentrations of calcium, phosphorus, magnesium, parathromone and osteocalcin,

Table 2. Serum calcium, uric acid, phosphorus, magnesium, parathromone, osteocalcin and total urinary oxalate excretion in 13 patients

Patient	Ca (mg/dl)	UA (mg/dl)	P (mg/dl)	Mg (mg/dl)	I-PTH (pg/ml)	Osteocalcin (ng/ml)	Oxalate (mg/24 h)
1	10.0	8.1	4.2	1.9	18.07	3.03	20
2	10.3	5.5	3.5	2.1	24.12	2.19	18
3	10.0	4.6	4.5	2.0	–	–	7
4	9.2	6.0	4.0	2.1	18.42	2.75	16
5	8.4	8.9	3.2	2.0	26.50	1.51	17
6	9.7	7.1	2.9	1.8	20.85	3.30	21
7	9.2	7.0	2.7	1.9	–	2.66	29
8	9.4	9.0	2.2	2.1	18.56	3.26	12
9	9.8	8.4	3.2	2.2	–	–	15
10	10.0	7.8	2.4	2.0	16.75	2.04	20
11	10.1	4.5	4.3	2.0	21.26	4.45	14
12	9.0	6.0	4.5	2.1	17.53	3.45	15
13	9.2	6.8	4.0	2.0	20.46	2.52	21
Mean ± SD	9.6 ± 0.5	6.9 ± 1.5	3.5 ± 0.8	2.0 ± 0.1	20.25 ± 3.09	2.83 ± 0.80	17.3 ± 5.3
Normal range	8.4–10.6	1.5–7.2	2.1–4.7	1.7–2.9	< 35.3	1.8–6.6	< 50

Ca, Calcium; UA, uric acid; P, phosphorus; Mg, magnesium; I-PTH, intact parathyroid hormone

Table 3. Individual Ccr and daily urinary excretions of calcium, magnesium and inorganic phosphate in 11 patients

Patient no.	Kidney	Ccr (ml/min)	Calcium (mg/24 h)	Ca/Crea ratio	Fe_{Ca} (%)	Magnesium (mg/24 h)	FE_{Mg} (%)	Pi (mg/24 h)	FE_{Pi} (%)
1	NPCN	35.90	36.0	0.04	0.70	68.4	6.96	151.2	6.96
	PCN	5.56	28.0	0.21	3.50	19.2	12.62	47.2	14.04
2	NPCN	71.78	102.0	0.09	0.96	42.0	1.93	282.0	7.79
	PCN	11.99	20.0	0.11	1.12	31.3	8.63	65.0	10.76
3	NPCN	43.35	92.0	0.19	1.47	44.0	3.52	152.0	5.41
	PCN	22.62	45.0	0.20	1.38	35.0	5.37	105.0	7.16
4	NPCN	20.88	55.2	0.11	2.00	55.2	8.74	185.0	15.38
	PCN	3.70	13.5	0.15	2.75	16.5	14.75	21.5	10.09
5	NPCN	31.55	63.6	0.05	1.67	79.9	8.79	257.5	17.71
	PCN	26.05	52.8	0.07	1.68	88.0	11.73	184.8	15.40
6	NPCN	62.37	132.0	0.13	1.52	132.0	8.17	270.0	10.37
	PCN	21.75	109.0	0.22	3.59	77.0	13.66	125.0	13.76
7	NPCN	81.67	115.2	0.08	1.06	51.2	2.29	394.0	12.41
	PCN	35.42	118.8	0.19	2.53	46.8	4.83	210.0	15.25
8	NPCN	39.53	82.0	0.11	1.53	–	–	420.0	33.54
	PCN	20.51	60.0	0.16	2.16	–	–	234.0	36.01
9	NPCN	71.12	218.1	0.15	2.17	190.3	8.45	835.0	25.48
	PCN	20.32	70.6	0.17	2.46	41.2	6.40	700.0	74.76
10	NPCN	64.35	216.0	0.19	2.33	92.8	5.01	408.0	18.35
	PCN	19.10	71.0	0.22	2.58	77.0	14.00	78.0	11.82
11	NPCN	25.46	42.0	0.06	1.13	68.0	9.27	136.0	8.63
	PCN	9.18	32.7	0.14	2.45	37.2	14.07	50.0	8.80
Mean ±SD	NPCN	49.8 ± 21.0	104.9 ± 62.9	0.10 ± 0.05	1.51 ± 0.51	82.4 ± 46.5	6.3 ± 2.9	317.3 ± 201.3	14.7 ± 8.7
	PCN	17.4 ± 9.4***	56.5 ± 34.3*	0.17 ± 0.05**	2.40 ± 0.78**	46.9 ± 25.2*	10.6 ± 3.9**	165.5 ± 190.8***	19.8 ± 19.8

PCN, Obstructed kidney; NPCN, contralateral kidney; Ca/Crea ratio, 24 h urinary calcium/24 h urinary creatinine excretion ratio; $FE_{Ca, Mg, Pi}$, fractional excretion of calcium, magnesium and inorganic phosphate; Pi, inorganic phosphate
* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$

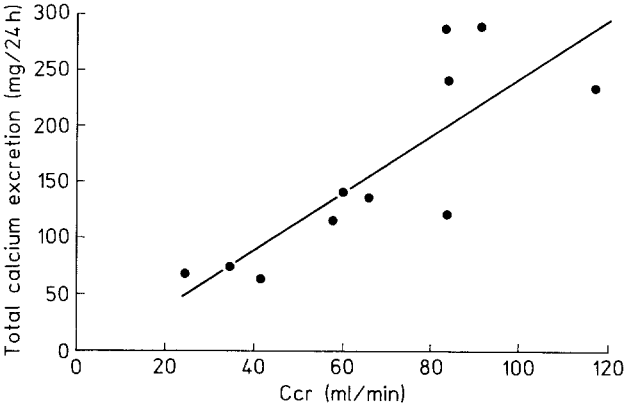

Fig. 1. Relationship between daily urinary calcium excretion and total Ccr in urolithiasis patients. $Y = 2.55X - 10.98$; $r = 0.82$; $P < 0.01$

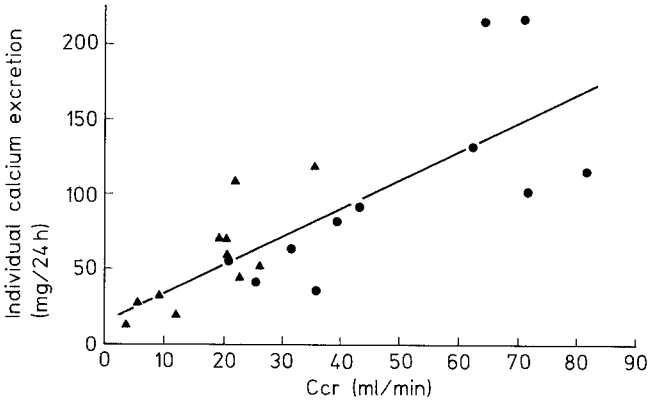

Fig. 2. Relationship between individual renal calcium excretion and individual Ccr in urolithiasis patients. $Y = 1.9X + 16.36$; $r = 0.78$; $P < 0.005$; ▲, PCN; ●, non-PCN

and total urinary oxalate excretion in 13 patients. All of these parameters were within the normal range. Five patients had hyperuricemia.

Comparison of creatinine clearance and daily urinary absolute and fractional excretions of calcium, magnesium and inorganic phosphate between the non-PCN and PCN kidneys of 11 patients are shown in Table 3. Endogenous creatinine clearance and urinary absolute excretions of calcium, magnesium and inorganic phosphate were significantly lower in the PCN (obstructed) kidneys than those in the contralateral kidneys. However, the total urinary calcium excretion normalized by total urinary creatinine

Table 4. The acute renal excretory response to percutaneous nephrostomy

	Duration (hours) following relief of unilateral ureteral obstruction									
	PCN					NPCN				
	2	4	6	8	24	2	4	6	8	24
Case 1										
$\dot{V}$ (ml)	50	40	50	50	700	240	240	220	250	2,500
Ccr (ml/min)	13.2	10.4	16.8	9.8	7.8	38.5	40.0	37.2	39.6	37.4
$U_{Na} \cdot \dot{V}$ (mmol/min) ($\times$ 1,000)	1.4	2.0	1.5	1.0	1.7	103.5	25.0	47.9	36.7	36.0
FE_{Na} (%)	0.06	0.16	0.17	0.09	0.14	0.86	0.29	0.42	0.39	0.37
$U_K \cdot \dot{V}$ (mmol/min) ($\times$ 1,000)	22.2	0.4	0.3	0.3	0.3	12.3	3.0	5.6	4.3	3.9
FE_K (%)	32.6	1.07	0.91	0.75	0.78	3.31	1.15	1.62	1.51	1.31
$U_{Ca} \cdot \dot{V}$ (mg/min)	0.03	0.02	0.03	0.02	0.01	0.06	0.05	0.05	0.06	0.06
FE_{Ca} (%)	2.2	2.4	1.8	1.8	1.7	1.7	1.4	1.3	1.7	1.8
$U_{Mg} \cdot \dot{V}$ (mg/min)	0.03	0.02	0.03	0.02	0.01	0.07	0.08	0.07	0.07	0.06
FE_{Mg} (%)	9.8	9.9	8.3	7.7	7.8	8.7	9.5	8.2	8.5	7.7
$U_{Pi} \cdot \dot{V}$ (mg/min)	0.10	0.09	0.09	0.08	0.07	0.38	0.39	0.38	0.39	0.38
FE_{Pi} (%)	16.2	18.9	20.7	19.5	20.9	21.9	21.7	21.2	20.8	24.5
Case 2										
$\dot{V}$ (ml)	140	80	60	60	800	460	200	250	200	2,160
Ccr (ml/min)	15.8	8.7	6.4	7.8	8.3	86.3	60.7	80.8	66.9	69.2
$U_{Na} \cdot \dot{V}$ (mmol/min) ($\times$ 100)	4.8	0.3	3.8	2.7	2.0	19.6	4.4	5.0	5.6	6.3
FE_{Na} (%)	2.59	1.85	1.61	1.97	2.09	3.64	0.79	0.95	1.10	1.19
$U_K \cdot \dot{V}$ (mmol/min) ($\times$ 100)	1.2	1.1	1.5	0.9	1.0	8.2	2.6	3.1	3.5	2.4
FE_K (%)	20.3	23.5	19.8	19.8	19.4	47.3	14.4	18.6	19.8	14.4
$U_{Ca} \cdot \dot{V}$ (mg/min)	0.04	0.02	0.02	0.02	0.02	0.16	0.15	0.17	0.17	0.15
FE_{Ca} (%)	3.0	2.2	2.6	2.8	2.6	2.0	2.6	2.2	2.7	2.2
$U_{Mg} \cdot \dot{V}$ (mg/min)	0.06	0.07	0.03	0.06	0.05	0.16	0.06	0.13	0.08	0.10
FE_{Mg} (%)	16.4	36.5	20.2	30.7	30.0	9.3	5.1	7.7	5.6	6.7
$U_{Pi} \cdot \dot{V}$ (mg/min)	0.03	0.01	0.01	0.01	0.01	0.39	0.39	0.41	0.44	0.42
FE_{Pi} (%)	4.8	2.1	2.6	2.1	3.0	11.2	16.1	12.6	16.3	14.0

PCN, Obstructed kidney; NPCN, contralateral kidney; $\dot{V}$, urine volume; Ccr, creatinine clearance; $U_{Na} \cdot \dot{V}$, absolute sodium excretion; $U_K \cdot \dot{V}$, absolute potassium excretion; $U_{Ca} \cdot \dot{V}$, absolute calcium excretion; $U_{Mg} \cdot \dot{V}$, absolute magnesium excretion; $U_{Pi} \cdot \dot{V}$, absolute inorganic phosphate excretion; $FE_{Na, K, Ca, Mg, Pi}$, fractional excretion of sodium, potassium, calcium, magnesium and inorganic phosphate

excretion in the PCN kidney was significantly greater than that in the non-PCN kidney. Except for inorganic phosphate, urinary fractional excretion of calcium and magnesium in the obstructed kidneys was increased significantly.

The relationships between creatinine clearance and urinary excretion of calcium are depicted in Figs. 1 and 2. A significant linear correlation was noted between total Ccr and daily total calcium excretion ($r = 0.82$, $P < 0.01$). The total urinary calcium excretions of three patients with total Ccr below 45 ml/min were less than 75 mg/day (64, 68.7, 74.7 mg/day, respectively). As shown in Fig. 2, there was also a significant linear correlation between the individual Ccr and 24-h urinary calcium excretion ($r = 0.78$, $P < 0.005$).

Figure 3 compares the fractional excretion of calcium among patients grouped on the basis of their creatinine clearance. The fractional excretion of filtered calcium (FE_{Ca}) increased as renal function decreased, and it became significantly higher when Ccr was below 20 ml/min. In addition, the individual creatinine clearance was also significantly correlated with the daily excretions of magnesium ($r = 0.55$, $P < 0.01$) and inorganic phosphate ($r = 0.60$, $P < 0.01$).

In order to evaluate the acute renal excretory response to percutaneous nephrostomy, bilateral urine samples were collected immediately after PCN and the data are summarized in Table 4. The creatinine clearance (Ccr) was consistently lower in the obstructed kidney than the contralateral kidney in both patients. The Ccr of PCN

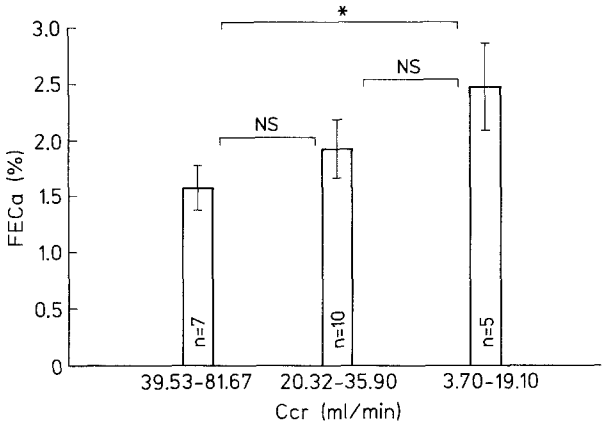

Fig. 3. Fractional calcium excretion profiles of nephrolithiasis patients with different levels of renal function. FE_{Ca} = fractional excretion of calcium. NS = Not significant. * $P < 0.05$

kidney decreased gradually and reached a steady level 24 h after PCN. There was no significant change in Ccr in the contralateral kidney. The absolute excretions of sodium, potassium, calcium, magnesium and phosphate were lower in obstructed kidneys than in the contralateral kidneys. However, the fractional excretions of sodium and potassium of obstructed kidney were higher than the contralateral in one case (case 2), but the effect was opposite in the other. The fractional excretions of calcium and magnesium appeared higher in obstructed kidneys. In contrast, the fractional excretion of phosphate was significantly lower in the PCN kidneys than that in the contralateral kidneys. The serum sodium, potassium, calcium, magnesium, phosphorus and creatinine were unchanged during the 24-h observation period in these two patients.

Discussion

Theories have been advanced to account for the formation of urinary tract stones. The most commonly mentioned and an attractive explanation is the hyperexcretion-crystallization hypothesis. Hypercalciuria has been considered one of the important factors predisposing to calcium stone formation [4, 6, 8, 9, 14]. However, in patients with advanced renal failure the daily total excretion of calcium was decreased [1, 3, 11, 12], indicating that renal function affects calcium excretion. The present study provides an approach to measuring individual Ccr and calcium excretion from each kidney for evalution of the effect of obstructive uropathy on calcium excretion. The results are in accordance with the previous observations that impaired renal function reduces urinary excretion of calcium. Furthermore, we found a highly significant correlation between Ccr and total or individual urinary calcium excretion.

As shown in Table 3, the stone-forming (PCN) kidneys of our patients had less total urinary calcium excretion than the contralateral kidneys. The reduced absolute calcium excretion in obstructed kidney corresponds to a reduced Ccr. This finding seems to contradict the hyperexcretion-crystallization theory, which would suggest that stone-forming kidneys have a higher excretion rate of calcium and other ions. However, if urinary calculi formation results in ureteral or renal obstruction and leads to impaired renal function, the urinary calcium excretion may not appropriately reflect the excretory capability of the affected kidney at the preobstructed stage. On the other hand, it is noteworthy that when the individual urinary calcium excretion is normalized by respective creatinine excretion, the calcium/creatinine ratio in PCN kidney is greater than that of the non-PCN kidney. It is unclear whether or not the altered calcium/creatinine ratio indicates that the stone-forming (PCN) kidney originally had more calcium excretion than the non-stone forming kidney and subsequently resulted in ipsilateral stone formation. Our previous animal experiments showed that when one kidney was obstructed, the contralateral kidney compensated by excreting more calcium, sodium and potassium, leading to little change in the total excretions of urinary calcium, sodium and potassium (unpublished data). In this study, five non-PCN kidneys excreted a great amount of calcium when their creatinine clearances were greater than 60 ml/min (Table 3, Fig. 2). If urolithiasis patients have the same mechanism to handle renal calcium as rats with ureteral obstruction, we might conclude that in patients with a normal or near-normal Ccr, the total excretion of 24-h urinary calcium will not be affected by obstructive uropathy regardless of the presence of unilateral obstructive uropathy. On the contrary, if the Ccr is below normal, the total urinary calcium excretion will be underestimated due to impaired global renal function. This is of significance because simultaneous measurements of Ccr and calcium excretion may then be used to screen the urolithiasis patients for therapeutic strategy. If the Ccr is within the normal range, the amount of calcium excretion may be reliable and the metabolic evaluation of urolithiasis patient should not be delayed in order to proceed to early and adequate treatment for hypercalciuria. On the other hand, once one kidney is damaged, the other normal kidney may excrete more lithogenic substances than in the usual condition. Whether this compensatory hyperexcretion is a risk factor causing new urinary calculi formation in normal kidneys remains uncertain. A prospective and long-term follow-up study seems to be warranted based on this analysis.

There are few descriptions of the pathophysiological changes in renal tubular function immediately after relief of ureteral obstruction in man. Better et al. have studied a patient with complete unilateral ureteral obstruction of 3 months' duration and have showed that the creatinine clearance in the obstructed kidney increased from 2.6 to 10.2 ml/min within 1 week after relief of obstruction and remained unchanged in the following 4 weeks [2]. However, in this study the urine collection was started 3 days after PCN. Jones et al. investigated the effects of chronic obstructive uropathy on kidney function during the obstructive phase and after relief of obstruction and concluded that the renal function recovered in two phases following relief of obstruction: an early tubular phase with

marked changes in tubular handling of water in the first 2 weeks and a later glomerular phase occurred between 2 weeks and 3 months [7]. In the present study, we observed that the Ccr and the absolute excretions of Na, K, Ca, Mg, and Pi of PCN kidney were higher in the first 2 h after relief of obstruction than the other periods. This phenomenon could be due to the high levels of creatinine and electrolytes retained in the dilated renal pelvis or as a result of renal function changes. Nevertheless, Ccr and the renal excretory function gradually reached a steady-state 24 h after relief of obstruction, as shown in Table 4. Interestingly, we noted one patient with low Ccr but lower FE_{Na} and FE_K in the PCN kidney than the contralateral kidney. On the other hand, both the absolute and fractional excretions of phosphate were lower in the obstructed kidney. These results are consistent with the previous study and are probably due to excessive tubular reabsorption of phosphate by the kidney with prolonged obstruction [2]. Because all of our patients had surgical intervention later for correction of the underlying causes of the obstruction, no further study was performed via PCN to monitor renal tubular function thereafter.

In summary, our study indicates that the individual renal calcium excretion of urolithiasis patients is correlated with Ccr. If the total Ccr is within the normal range, hypercalciuria may be detected adequately, but if the total Ccr is far below normal, the total amount of urinary calcium excretion may be unreliable as a screening procedure. On the other hand, immediately after relief of unilateral ureteral obstruction, there were indeed some changes in Ccr and renal tubular handling of Na, K, Ca, Mg, and Pi that may reach a steady-state within 24 h. Last but not least, the calcium-containing stone is the most common in stone disease. The characteristic relationship between various ions and obstructive uropathy and the tubular mechanisms merits further study.

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