

Recurrent Nephrolithiasis in Renal Tubular Acidosis. Metabolic Profiles, Therapy and Course

W. Schneeberger¹, A. Hesse², and W. Vahlensieck²

¹ Rehabilitations-Krankenhaus, W-5303 Bornheim-Merten, ² Experimentelle Urologie in der Urologischen Universitäts-Klinik, W-5300 Bonn, FRG

Abstract. 13 patients with recurrent urolithiasis and distal renal tubular acidosis (RTA I) were investigated for lithogenic metabolic disorders. Treatment was given and the patients observed for periods of up to 10 years.

Key words: Urolithiasis, Renal Tubular Acidosis

In the case of patients with recurrent urolithiasis, RTA can be diagnosed by checking their urine acidification and blood gas analysis. This will also permit a distinction to be made between complete and incomplete forms. RTA is particularly suspect as the causal metabolic disorder in cases of exclusively alkaline urine reaction where no infection is present, and in cases of nephrocalcinosis. In the literature, the frequency of RTA in urolithiasis is given as approximately 10%, which corresponds to the frequency of primary hyperparathyroidism in nephrocalcinosis. Of the 1,029 patients we have examined since 1976, 20 had RTA. We were able to observe and re-examine 13 of these over an extended period of time. A course of treatment, dependent on their individual metabolic profiles, was initiated for each patient. This was subsequently modified as required, and the efficiency of the various measures taken duly recorded.

Material and Methods

During a 10-day basic check-up as in-patients on a standard diet, RTA diagnosis was made using the ammonium chloride loading test and blood gas analysis. We found 9 patients with incomplete, and

4 with complete RTA. Apart from RTA diagnosis, calcium, phosphate, and oxalate levels were investigated, and oral calcium loading tests and stone analyses conducted. We then began the treatment and observed patients for up to 10 years. Where required, the therapy was either modified or changed.

Results

The distribution of metabolic disorders is shown in Table 1. In addition to calcium oxalate and calcium phosphate stones, one female patient also had low cystinuria and cystine stones.

In order to normalize the acid-base metabolism, patients with complete RTA were treated with sodium bicarbonate or citrate complexes (Uralyt-U) or ethacrynic acid (Hydromedin). Patients with incomplete RTA received urine-acidifying drugs, mainly ammonium chloride or methionine. In cases of severe hypercalciuria, fibre (Farnolith = mixed brans) or thiazides were administered.

Patients with hyperuricaemia or hyperuricosuria received Allopurinol. Hypopotassemia was substituted and urinary infection was treated.

Table 2: (Therapy)

In 2 out of 4 cases with complete RTA the urinary stone disease was not progressive in terms of X-ray status or stone episodes. 1 patient passed further stones, but no new ones were formed. In 1 patient, stone formation and renal insufficiency were progressive.

Table 3: (Stone Episodes)

Table 1: Metabolic disorders in our patients (n = 13) with

	Complete RTA (n = 4)	Incomplete RTA (n = 9)
Hypercalciuria	2	9
Hyperphosphaturia	0	1
Hyperoxaluria	0	0
Hyperuricosuria	0	2
Hypocitraturia	3	5
Hypokalemia	3	6
Ca-Phosphate Stones	1	5
Ca-Phosphate/Ca-Oxalate Stones	2	3
Ca-Oxalate Stones	0	1
Ca-Phosphate/Ca-Oxalate/ Cystine Stones	1	0
Nephrocalcinosis	3	7
Renal insufficiency	1	0

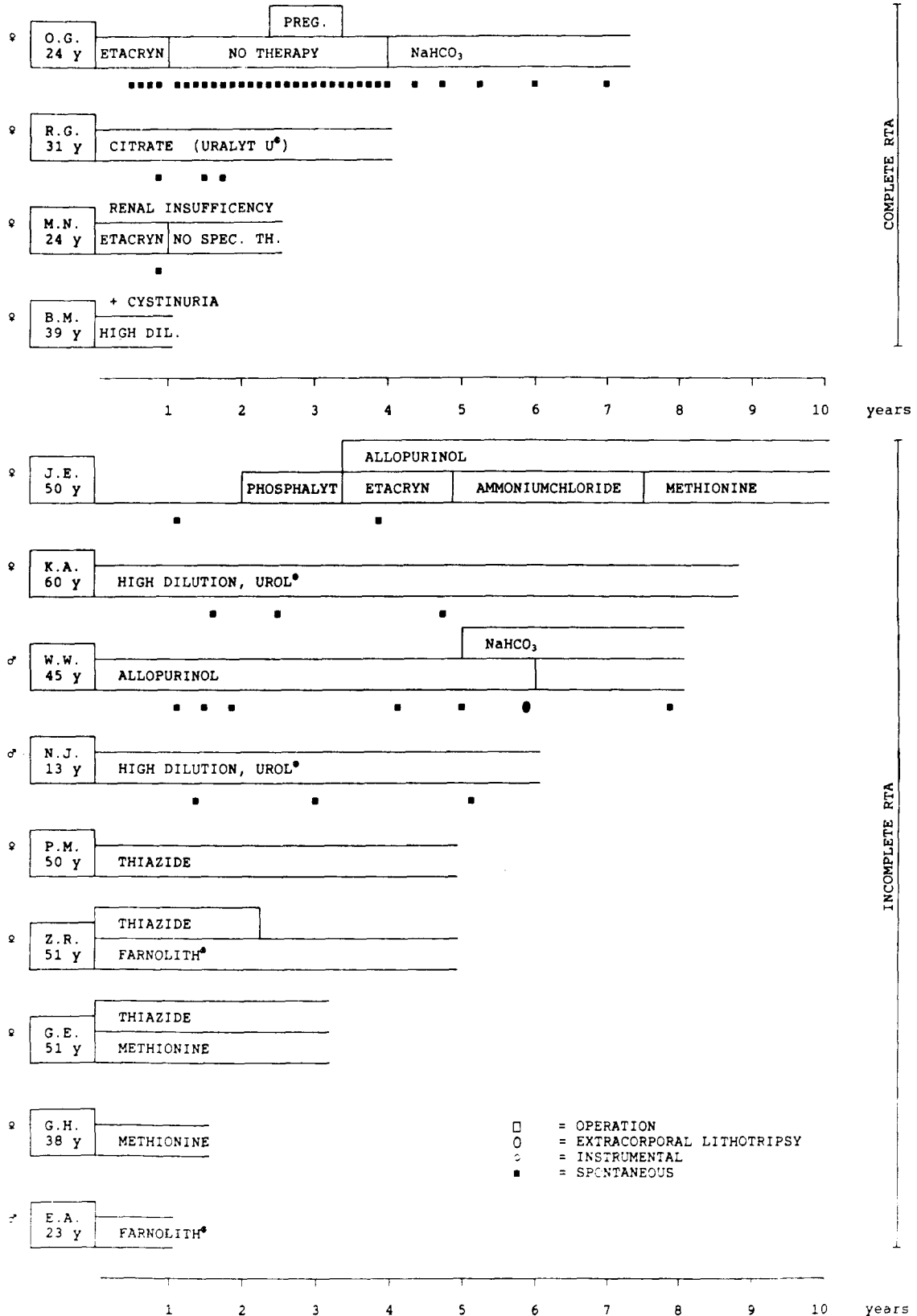
Conclusions

The treatment of RTA in terms of its main aspect - nephrocalcinosis - is differential and is only successful to a limited degree. The therapeutic approach has to be based on precise metabolic investigations, and must be monitored by follow-up examinations. In the case of our patients, we were able to achieve an improvement in 9 out of 13 cases.

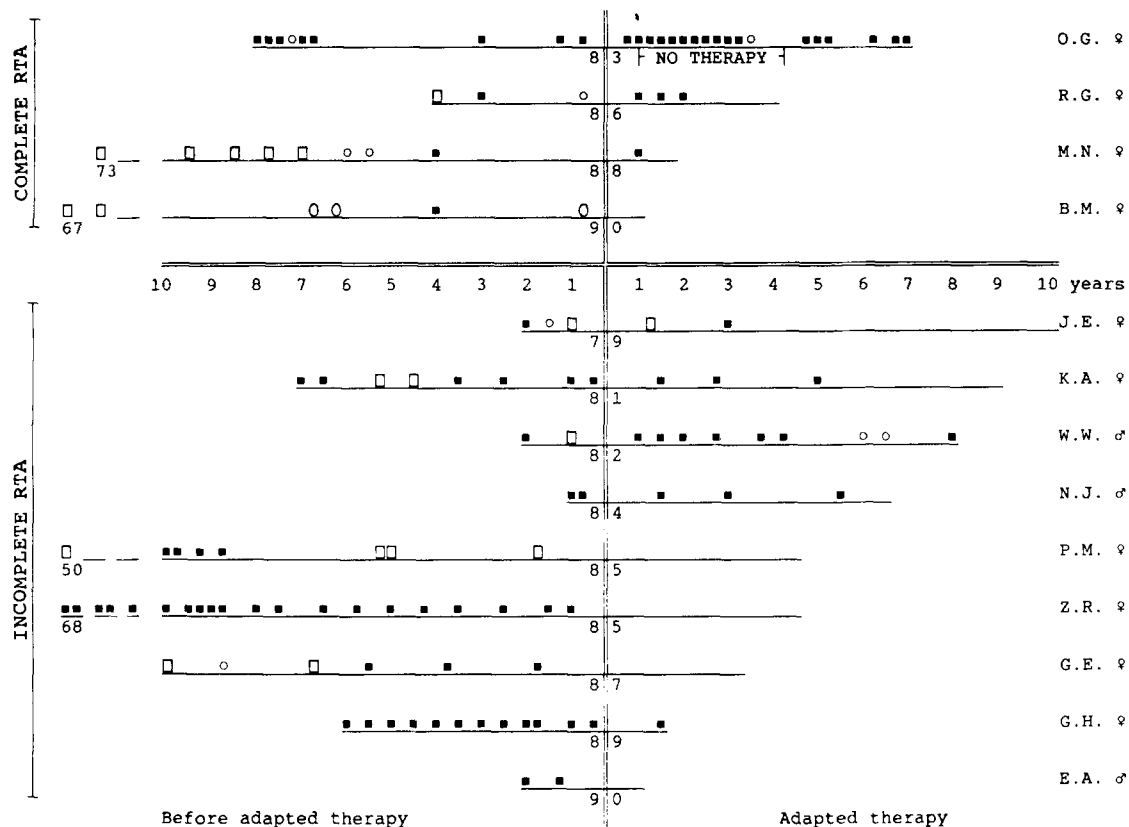
References

1. Bachmann V et al. (1981) The clinical importance of renal tubular acidosis in recurrent renal stone formers. In: Smith HL, Robertson WG, Finlayson F. Urolithiasis. Plenum Press, New York - London, pp 67 - 69
2. Battle D, Kurrman NA (1982) Distal renal tubular acidosis: Pathogenesis and Classification.

THERAPY



STONE EPISODES



- Am J Kidney Dis 1:328-344
3. Genova R, Guerra A et al. (1979) Distal tubular acidosis. Minerva Med 70:3015-3027
 4. Hesse A (1980) Laboruntersuchungen. In: Vahlensieck W. Urolithiasis 1. Springer, Berlin-Heidelberg-New York, p 52
 5. Huber A (1985) Renale tubuläre Acidose und Urolithiasis. Diss. Bonn
 6. Lindenberg K, Schmucki O (1980) Die renale tubuläre Acidose: Ursache oder Folge einer Nephrolithiasis. Ther Umsch 37:368-372
 7. Morris RC jr (1981) Renal tubular acidosis. N Engl J Med 304:418-420
 8. Quintanilla AP (1980) Renal tubular acidosis: mechanism and management. Postgrad Med 67:60-73
 9. Sommerkamp H (1977) Diagnostik der renalen tubulären Acidose. Fortschr Urol Nephrol 9:165-167
 10. Sommerkamp H (1980) Steinbildung bei der renalen tubulären Acidose. In: Vahlensieck W, Urolithiasis 4. Springer, Berlin-Heidelberg-New York pp 59-64
 11. Vahlensieck W, Hesse A, Bach D (1981) Inzidenz, Prävalenz und Mortalität des Harnsteinleidens in der Bundesrepublik Deutschland. Fortschr Urol 17:1-5
 12. Wrong OM (1981) Renal tubular acidosis. N Engl J Med 304:1548-1549
- Dr. W. Schneeberger
Rehabilitations-Krankenhaus
Klosterstr. 2
W-5303 Bornheim-Merten, FRG