

Prevention of Recurrent Stone Formation: Long Time Results under Treatment Based on an Extended Metabolic Investigation

K. Klocke¹, A. Hesse¹, W. Vahlensieck¹, W. Schneeberger²

¹ Department of Urology, University of Bonn, FRG, ² Rehabilitationskrankenhaus, Bornheim-Merten, FRG

Introduction

Nationwide surveys of the population of the Federal Republic of Germany have shown that the prevalence of urinary stone disease is 4%. The incidence of new stone formation in the years 1979 and 1984 was 0.5% and 0.4%; 60% were recurrent stones (1). The high incidence of recurrent stone formation shows the need for an effective prophylactic treatment. Using an inpatient protocol previously described (2) since 1975 more than 1000 recurrent stone formers who did not respond to unspecific therapeutic measures were evaluated in the department of Urology of the University of Bonn. By means of this protocol up to 90% of the recurrent stone formers revealed pathogenetic causes for their stone formation. In detail the results of these investigations of the first 800 consecutive patients are reported elsewhere (3). Based on this information certain treatment was specifically chosen for each stone former according to the detected metabolic disturbances. The validity of this selective approach to diagnosis and therapy of recurrent stone formation requires the demonstration that new stone formation can be successfully inhibited. This communication concerns with the clinical response to selective treatment in 307 of the first 800 evaluated recurrent stone formers, who could be followed more than 6 months.

Patients and Methods

Patients

Of 307 recurrent stone formers who could be followed more than 6 months 233 formed calcium oxalate stones, 34 formed calcium phosphate, 12 struvite, 11 uric acid and 17 cystine stones. The mean follow up

was 19.5 months (range: 0.5 - 11.5 years). Hypercalciuria was present in 61.8% of the patients, hyperoxaluria in 45.1%, we detected hyperuricosuria in 58.4% and urinary acidification defects (according to the acid load test of Wrong and Davies) or relative urinary hyperacidity during dietary reduction of acid load in 42.3%. Hypocitraturia was found in 51.7%. The frequency of risk factors in the different stone groups is shown in Figure 1.

Treatment

Hypercalciuria was controlled with alkali-citrate and/or thiazide. Hyperuricosuria was treated with allopurinol. Acidifying agents were recommended to calcium phosphate and struvite stone formers. Relative urinary hyperacidity in uric acid and calcium oxalate stone formers was treated with alkali-citrate. Cystinuria was treated with ascorbate and/or alphanerceptopropionylglycine. Hypocitraturia was controlled with alkali-citrate.

Assessment of stone activity

Pretreatment stones were calculated as the sum of all new stones formed. To calculate recurrence rate (stones per year) the first stone formed by each patient was omitted. Then the number of recurrent stones was divided by the total interval from the first stone to the onset of treatment. New stones were defined by the passage, removal, or radiographic or sonographic visualization of a stone that was not seen on previous radiographs or sonographs. At least an abdominal flat plate and ultrasound of the kidneys were obtained just before starting treatment to document pre-existing stones. Each patient was contacted two to four times a year for interview and control of stone forming activity by radiography or ultrasound. All patients were newly reviewed for this report.

Results

In calcium oxalate stone formers the pretreatment stone frequency of 1.57 stones per year was lowered under therapy to 0.3 stones per year. 80.7% of the patients remained stone-free. The rate of new stone production fell in calcium phosphate stone formers from 1.81 to 0.12 stones per year, 91.2% did not form new stones. In struvite stone formers the recurrence rate was reduced from 0.70 to 0.17 stones per year, just 1 patient had a recurrency. The highest pretreatment stone production rate had uric acid stone formers with 4.12 stones per year that could be lowered to 0.12 stones per year. 90.9% showed no sign of further stone forming activity. In cystine

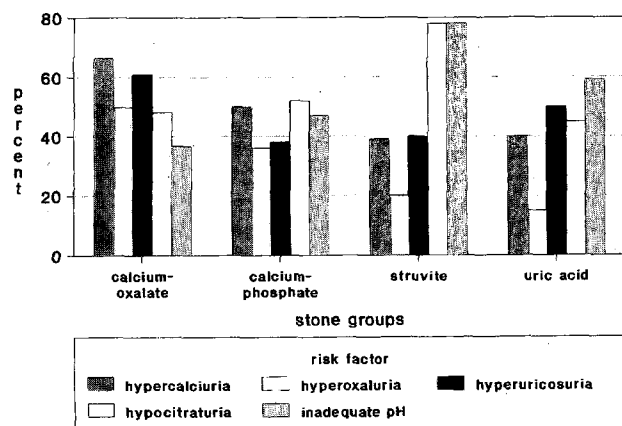


Fig. 1: Risk factors in recurrent stone formers (n = 307)

stone formers the recurrence rate decreased from 1.43 stones per year to 0.58 stones per year, only 67.7% remained stone-free (Figure 2).

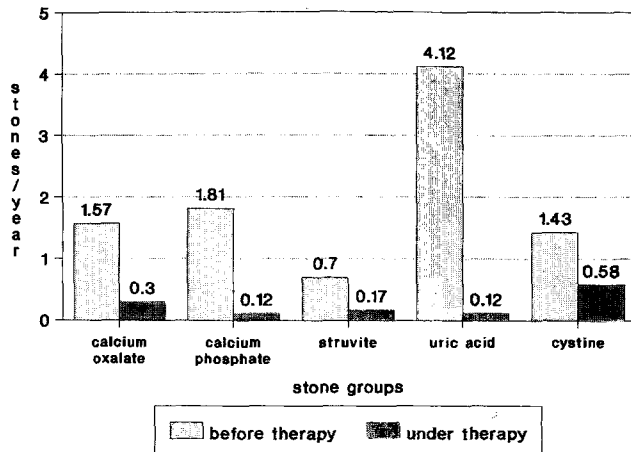


Fig. 2: Recurrence rate before and under therapy

Comments

The frequency of new stone formation could be drastically reduced in 307 recurrent stone formers by a treatment that was based on the results of an extended and standardized metabolic investigation on an in-patient basis. The best results could be achieved in uric acid stone formers whereas cystine stone formers showed the lowest decrease in recurrence rate. These observations support the validity of our concept of subtle diagnostic and specifically selected therapeutic measures in preventing recurrent urinary stone formation. They cannot prove it,

because we did not include randomized placebo groups. The positive "stone clinic effect" is well known. But it should be pointed out that nearly all of the patients who were selected for our inpatient protocol had not responded to unspecific treatment measures before. This strongly supports the assumption, that at least in some recurrent stone formers a subtle diagnosis of physiochemical and physiological disturbances and a specifically chosen treatment is necessary to achieve satisfactory prevention of stone recurrence.

References

1. Vahlensieck W (1987) Epidemiologie und Kausalfaktoren. In: Vahlensieck W (Hrsg) Das Harnsteinleiden; Ursachen - Diagnose - Therapie. Springer-Verlag, Berlin, Heidelberg, New York London Paris Tokyo, pp 1-46
2. Bach D, Hesse A, Vahlensieck W (1981) Labor-diagnostisches Stufenprogramm beim Harnsteinleiden. *Therapiewoche* 31:1260-1266
3. Vahlensieck W, Hesse A, Klocke K, Schneeberger W (1989) Extended investigations in 800 recurrent stone formers: Methods and results. In: Walker VR, Sutton RAI, Cameron ECB, Pak CYC, Robertson G, *Urolithiasis*, Plenum Press, New York London, pp 691- 693

Dr. K. Klocke
Department of Urology
University of Bonn
Sigmund-Freud-Str. 25
W-5300 Bonn 1, FRG