

CLINICAL STUDY OF URIC ACID UROLITHIASIS

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Uric acid urolithiasis develops from various causes. To investigate the clinical and biochemical presentation of patients with uric acid urolithiasis, a retrospective study was designed. A total of 46 cases were enrolled between January 2004 and December 2005. The compositions of the stones were analyzed by infrared spectrophotometry. There were 39 males (84.8%) and seven females (15.2%), with a mean age of 61.5 ± 10.6 years and mean body mass index (BMI) of 26.7 ± 3.1 kg/m². The stone location was kidney in 10 (21.7%), ureter in 22 (41.8%), and bladder in 14 (30.5%). Multiple stones were diagnosed in 36 patients (78.3%). Pre-existing comorbidities included diabetes mellitus in 11 patients (23.9%), hypertension in 23 (50%), gout in 13 (28.2%), and benign prostatic hyperplasia in 14 (30.4%). Mean serum creatinine and uric acid was 1.6 ± 0.6 mg/dL and 7.6 ± 1.8 mg/dL, respectively. There were 27 patients (58%) with creatinine >1.4 mg/dL. The mean urinary pH was 5.42 ± 0.46 . Patients with uric acid urolithiasis were predominantly male, older, with higher BMI, multiple stone presentation, with lower urinary pH, and hyperuricemia. Exacerbation of the renal function should also be of concern because of the high proportion of patients with renal insufficiency diagnosed in this study.

Key Words: body mass index, uric acid, urolithiasis

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Urolithiasis is a common urologic disease that affects approximately 10% of the population worldwide [1]. Uric acid is a frequent component of urinary stones and affects calcium stone formation. The prevalence of uric acid stones is estimated to be 5–10% of all urinary stone diseases [2]. A correct diagnosis and understanding of the pathophysiology of uric acid nephrolithiasis have important therapeutic implications. Thus, we

reviewed the current clinical pictures of uric acid stone as well as the related literature.

MATERIALS AND METHODS

Between January 2004 and December 2005, a retrospective study was performed in three hospitals in subtropical southern Taiwan. A total of 46 patients with uric acid stones verified by stone analysis were identified. Clinical data, including age, sex, body mass index (BMI), stone location, presentation of single or multiple stones, pre-existing comorbidities (i.e. diabetes mellitus, hypertension, gout), serum creatinine and uric acid levels, and urinary pH were collected. Stone

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composition was analyzed by Fourier transformed infrared spectrophotometry.

RESULTS

There were 46 patients, 39 males (84.8%) and seven females (15.2%), with a mean age of 61.5 ± 10.6 years and mean BMI of 26.7 ± 3.1 kg/m². The stone location was kidney in 10 (21.7%), ureter in 22 (41.8%), and bladder in 14 (30.5%). Multiple stone presentation was diagnosed in 36 (78.3%). Pre-existing conditions included diabetes mellitus in 11 patients (23.9%), hypertension in 23 (50%), gout in 13 (28.2%), and benign prostatic hyperplasia in 14 (30.4%). Mean serum creatinine and uric acid levels were 1.6 ± 0.6 mg/dL and 7.6 ± 1.8 mg/dL, respectively. The mean urinary pH was 5.42 ± 0.46 .

DISCUSSION

The prevalence of uric acid urolithiasis varies in different geographical areas and countries, e.g. 5–10% in the US, 17–25% in Germany, 4% in Sweden, and up to 40% in Israel. The apparent geographic variations indicate that genetic, dietary, and environmental factors may have important roles in the formation of uric acid stones [2].

Gout is an ancient disease and its association with uric acid stones has long been recognized. Uric acid stones are formed from dehydration, excessive sweating, intestinal alkali loss, and purine overload or overproduction. Idiopathic uric acid nephrolithiasis may also develop despite the absence of the above causes. People with uric acid stones may have normo-uricemia in some situations, since it is believed that uric acid urolithiasis is due to defects in urinary acidification and excretion of urates [3–5].

An increasing percentage of the population is affected by obesity and the metabolic syndrome, a condition that is metabolically characterized by insulin resistance and clinically defined by abdominal obesity, dyslipidemia, elevated blood pressure, and elevated fasting glucose. It is reported that patients with recurrent uric acid stones manifest clinical and metabolic abnormalities consistent with the metabolic syndrome [4,6,7]. Lower urinary pH increases concentrations of the sparingly soluble undissociated uric acid, which

directly promotes the formation of uric acid stones. Because of the overlapping clinical features between gouty diathesis and the metabolic syndrome, the relationship of the defective biologic activity of insulin and urinary acidification has been studied. It is suggested that the renal manifestation of insulin resistance may be low urinary ammonium and pH. This defect can result in increased risks of uric acid stone formation despite normo-uricosuria [8,9].

Our data show that patients with uric acid urolithiasis were mostly older males with high BMI, multiple stone presentation, lower urinary pH, and higher serum uric acid level. Acidic urine is a prerequisite for uric acid stone formation and growth. Management with urinary alkalization for stone dissolution and prevention of recurrence should be effective. Low purine diet ingestion to decrease hyperuricemia and lifestyle modification to reduce the incidence of overweight or obesity can also be helpful.

Furthermore, an exacerbation of renal function should be of concern because a high proportion of patients with renal insufficiency were noted in our study. Some reports have investigated the significance of hyperuricemia on the risk of developing end-stage renal disease. Strategies for decreasing serum uric acid levels may be recommended to prevent the progression of renal failure [10,11].

In conclusion, the profile of patients with uric acid urolithiasis was male, older, and with higher BMI, multiple stone presentation, lower urinary pH, and higher serum uric acid. A high proportion of patients with renal insufficiency was also diagnosed in this study.

REFERENCES

1. Anderson RA. A complementary approach to urolithiasis prevention. *World J Urol* 2002;20:294–301.
2. Shekarriz B, Stoller ML. Uric acid nephrolithiasis: current concepts and controversies. *J Urol* 2002;168:1307–14.
3. Pak CYC, Sakhaee K, Peterson RD, et al. Biochemical profile of idiopathic uric acid nephrolithiasis. *Kidney Int* 2001;60:757–61.
4. Pak CYC, Sakhaee K, Moe O, et al. Biochemical profile of stone-forming patients with diabetes mellitus. *Urology* 2003;61:523–7.
5. Abate N, Chandalia M, Cabo-Chan AV, et al. The metabolic syndrome and uric acid nephrolithiasis: novel features of renal manifestation of insulin resistance. *Kidney Int* 2004;65:386–92.

6. Powell CR, Stoller ML, Schwartz BF, et al. Impact of body weight on urinary electrolytes in urinary stone formers. *Urology* 2000;55:825–30.
7. Ekeruo WO, Tan YH, Young MD, et al. Metabolic risk factors and the impact of medical therapy on the management of nephrolithiasis in obese patients. *J Urol* 2004;172:159–63.
8. Maalouf NM, Cameron MA, Moe OW, et al. Novel insights into the pathogenesis of uric acid nephrolithiasis. *Curr Opin Nephrol Hypertens* 2004;13:181–9.
9. Sakhaee K, Adams-Huet B, Moe OW, et al. Pathophysiologic basis for normouricosuric uric acid nephrolithiasis. *Kidney Int* 2002;62:971–9.
10. Iseki K, Ikemiya Y, Inoue T, et al. Significance of hyperuricemia as a risk factor for developing ESRD in a screen cohort. *Am J Kidney Dis* 2004;44:642–50.
11. Siu YP, Leung KT, Tong MKH, et al. Use of allopurinol in slowing the progression of renal disease through its ability to lower serum uric acid level. *Am J Kidney Dis* 2005;47:51–9.

尿酸尿路結石的臨床研究

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尿酸成份的尿路結石形成的原因複雜，為了探討尿酸結石的臨床及生化表現，本文收集自 2004 年 1 月至 2005 年 12 月共 46 病例做研究，結石成份經由傅立葉紅外線圖譜分析而知，46 尿酸結石病例中，男性有 39 位 (84.8%)，女性 7 位 (15.2%)，平均年齡 61.5 ± 10.6 歲，平均體質指數 (BMI) 為 $26.7 \pm 3.1 \text{ kg/m}^2$ 。結石部位在腎臟有 10 位 (21.7%)，輸尿管 22 位 (41.8%)，膀胱 14 位 (30.5%)。多發性結石有 36 位 (78.3%)，患者同時合併糖尿病有 11 位 (23.9%)，高血壓有 23 位 (50%)，痛風 13 位 (28.2%)，前列腺肥大 14 位 (30.4%)。患者肌酸酐平均值為 $1.6 \pm 0.6 \text{ mg/dL}$ ，尿酸平均值為 $7.6 \pm 1.8 \text{ mg/dL}$ ，尿液酸鹼度 (pH) 平均值為 5.42 ± 0.46 。其中肌酸酐大於 1.4 mg/dL 者有 27 位 (58%)。大體而言，尿酸結石的患者以男性較多，年紀較大，體質指數較高，結石部位較為多發性，尿液酸鹼度偏低，血中尿酸值偏高。其中超過一半的病例有腎功能不良的現象，是值得注意的。

關鍵詞：體質指數，尿酸結石，尿路結石

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