

Distal renal tubular dysfunction: a common feature in calcium stone formers

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Abstract. Distal renal tubular acidosis has been reported as an uncommon cause of urinary calcium stone disease. However, this defect appears to be more frequent when appropriate tests are performed systematically. Twenty-nine patients with recurrent calcium stones have been separated into three groups: normocalcaemic (group A), renal hypercalcaemic (group B) and absorptive hypercalcaemic (group C). Distal tubular functions were investigated by the (urine-blood) $p\text{CO}_2$ gradient and by an ammonium chloride test. (Urine-blood) $p\text{CO}_2$ gradient was (mean \pm SEM), 3.33 ± 0.59 in group A, 2.95 ± 0.34 in group B and 3.31 ± 0.58 kPa in group C. All these values differ significantly from those observed in controls (4.11 ± 0.28 kPa; $P < 0.05$). After 3 days of ammonium chloride loading, ammonium excretion averaged 54.7 ± 4.2 in group A, 54.4 ± 4.3 in group B and 64.3 ± 5.5 $\mu\text{mol min}^{-1}$ in group C. Values obtained in the first two groups were significantly lower than that achieved by control subjects (76.4 ± 14.9 $\mu\text{mol min}^{-1}$). It is concluded that tubular dysfunctions defined as impairments in hydrogen ion secretion and ammonium excretion after an acid challenge are a common feature of the urinary calcium stone disease and play a contributory role in its pathogenesis.

Key words. Renal stones, renal tubular defects.

Introduction

Complete distal renal tubular acidosis has been reported on few occasions as a cause of recurrent renal stones. Coe reviewed the literature in 1980 and recorded fourteen such cases to which he added six personal observations [1]. More recently, five cases of incomplete distal renal tubular acidosis associated with urolithiasis have been published by Konnak *et al.* [2]. The scarcity of these reports contrasts with the finding that elevated urinary pH is the third risk factor after hyperoxaluria and hypercalcaemia in calcium stone disease [3]. Furthermore, when distal renal tubular acidosis is present in hypercalcaemic subjects, it has been thought that the defect in urinary acidification was secondary to hypercalcaemia rather than a

primary dysfunction [4]. However, Backman *et al.*, studying by the short ammonium chloride loading test 310 consecutive recurrent renal stone formers, have found a much higher incidence of impaired acidification capacity than expected from reports of isolated case. Nineteen per cent of their patients with a female/male ratio of 2.9 had such a defect [5].

Therefore, the present study was designed: (a) to evaluate the prevalence of impaired acidification of the urine in patients with renal stones using the (urine-blood) $p\text{CO}_2$ gradient, which allows the recognition of even discrete defect in tubular hydrogen ion secretion; and (b) to determine if this defect was a primary or a secondary phenomenon in calcium stone disease by comparing calcium stone formers with and without hypercalcaemia.

Patients and Methods

Twenty-nine male patients with calcium recurrent urolithiasis were divided into three groups on the basis of a calcium loading test: normocalcaemic, group A ($n=9$) (mean age 43.5 ± 4.6 years), renal hypercalcaemic, group B ($n=12$) (mean age 38.5 ± 9.5 years) or absorptive hypercalcaemic, group C ($n=8$) (mean age 37.5 ± 3.5 years). All the patients were free from urinary tract infection at the time of the investigation. None of them presented an obstructive nephropathy during the month preceding the functional evaluation. This investigation was performed in ambulatory patients on their usual diet. After an overnight fast, the patients were asked to empty their bladder. The urine was discarded and thereafter a 2-h urine collection period was made between 0700 and 0900 h. At the end of this period, a standard breakfast was served along with a tablet containing 1 g of calcium (Calcium Sandoz® 1000) and urine collected again over a 4-h period. Calcium/creatinine ratio was determined in each period and citrate/creatinine ratio only in the first one. Results were interpreted according to a slight modification of the proposal of Pak *et al.* [6], described by Megevand & Chollet [7]. Normocalcaemic patients are those who had a normal calcium/creatinine ratio before and after calcium load; renal hypercalcaemic patients are defined as patients presenting an elevated calcium/creatinine ratio both before and after calcium

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