

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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Renal Tubular Dysfunction Discussions In Patient Management

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Renal Tubular Dysfunction Discussions In Patient Management:

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Table of Contents Renal Tubular Dysfunction Discussions In Patient Management

1. Understanding the eBook Renal Tubular Dysfunction Discussions In Patient Management
 - The Rise of Digital Reading Renal Tubular Dysfunction Discussions In Patient Management
 - Advantages of eBooks Over Traditional Books
2. Identifying Renal Tubular Dysfunction Discussions In Patient Management
 - Exploring Different Genres
 - Considering Fiction vs. Non-Fiction
 - Determining Your Reading Goals
3. Choosing the Right eBook Platform
 - Popular eBook Platforms
 - Features to Look for in an Renal Tubular Dysfunction Discussions In Patient Management
 - User-Friendly Interface
4. Exploring eBook Recommendations from Renal Tubular Dysfunction Discussions In Patient Management
 - Personalized Recommendations
 - Renal Tubular Dysfunction Discussions In Patient Management User Reviews and Ratings
 - Renal Tubular Dysfunction Discussions In Patient Management and Bestseller Lists

5. Accessing Renal Tubular Dysfunction Discussions In Patient Management Free and Paid eBooks
 - Renal Tubular Dysfunction Discussions In Patient Management Public Domain eBooks
 - Renal Tubular Dysfunction Discussions In Patient Management eBook Subscription Services
 - Renal Tubular Dysfunction Discussions In Patient Management Budget-Friendly Options
6. Navigating Renal Tubular Dysfunction Discussions In Patient Management eBook Formats
 - ePub, PDF, MOBI, and More
 - Renal Tubular Dysfunction Discussions In Patient Management Compatibility with Devices
 - Renal Tubular Dysfunction Discussions In Patient Management Enhanced eBook Features
7. Enhancing Your Reading Experience
 - Adjustable Fonts and Text Sizes of Renal Tubular Dysfunction Discussions In Patient Management
 - Highlighting and Note-Taking Renal Tubular Dysfunction Discussions In Patient Management
 - Interactive Elements Renal Tubular Dysfunction Discussions In Patient Management
8. Staying Engaged with Renal Tubular Dysfunction Discussions In Patient Management
 - Joining Online Reading Communities
 - Participating in Virtual Book Clubs
 - Following Authors and Publishers Renal Tubular Dysfunction Discussions In Patient Management
9. Balancing eBooks and Physical Books Renal Tubular Dysfunction Discussions In Patient Management
 - Benefits of a Digital Library
 - Creating a Diverse Reading Collection Renal Tubular Dysfunction Discussions In Patient Management
10. Overcoming Reading Challenges
 - Dealing with Digital Eye Strain
 - Minimizing Distractions
 - Managing Screen Time
11. Cultivating a Reading Routine Renal Tubular Dysfunction Discussions In Patient Management
 - Setting Reading Goals Renal Tubular Dysfunction Discussions In Patient Management
 - Carving Out Dedicated Reading Time
12. Sourcing Reliable Information of Renal Tubular Dysfunction Discussions In Patient Management
 - Fact-Checking eBook Content of Renal Tubular Dysfunction Discussions In Patient Management
 - Distinguishing Credible Sources
13. Promoting Lifelong Learning

- Utilizing eBooks for Skill Development
 - Exploring Educational eBooks
14. Embracing eBook Trends
- Integration of Multimedia Elements
 - Interactive and Gamified eBooks

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