

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:

Management of Acute Kidney Problems Achim Jörres, Claudio Ronco, John A. Kellum, 2010-03-11 Acute kidney failure is an important clinical area in the intensive care unit setting. An estimated 5-20% of critically ill patients experience an episode of acute kidney failure during the course of their illness and about 5% of patients admitted to an ICU will eventually require renal replacement therapy. In these patients, in-hospital mortality is extremely high, exceeding 50%. Thus, the early detection and causal treatment of acute kidney problems is vitally important for a successful outcome. Written by internationally renowned experts, this clinical reference offers helpful advice with the most recent information on the definition, epidemiology, pathophysiology, and clinical causes of acute kidney failure as a fundamental prerequisite for prevention of this disorder. Moreover, it also covers differential diagnostic approaches for patients with acute renal failure and provides a detailed outline of important measures for their clinical management. Finally, separate chapters are dedicated to various key aspects related to the adequate delivery of acute renal replacement therapy. It is intended as a helpful guide for all clinicians involved in the care of patients at risk of developing acute kidney problems. *Miller's Anesthesia* Lars I. Eriksson, 2009-01-01 From fundamental principles to advanced subspecialty procedures, this text is the go-to reference on the technical, scientific, and clinical challenges professionals face. Features new chapters, new authors, meticulous updates, an increased international presence, and a new full-color design. **Advanced Practice in Critical Care** Sarah McGloin, Anne McLeod, 2010-02-09 Advanced Practice in Critical Care provides experienced critical care nurses with a clear and distinct evidence base for contemporary critical care practice. Central to the book is the application of research and evidence to practice, and therefore case studies and key critical care clinical situations are used throughout to guide the reader through the patient care trajectory. Each chapter introduces an initial patient scenario, and as the chapter progresses, the patient scenario develops with theoretical perspectives and application. In this way, it is evident how multi-organ dysfunction develops, impacting upon and influencing other body systems, demonstrating the multi-organ impact that is often experienced by the critically ill patient. In this way, consequences of critical illness such as acute renal failure, haemostatic failure, and liver dysfunction are explored. Throughout the text, key research findings and critical care treatment strategies are referred to, applied, and evaluated in the context of the given patient case study. Advanced assessment techniques are explained, and the underlying pathophysiology is discussed in depth. Advanced Practice in Critical Care is an essential resource for experienced practitioners within critical care who primarily care for patients requiring high dependency or intensive care. Suki and Massry's Therapy of Renal Diseases and Related Disorders Wadi N. Suki, Shaul G. Massry, 2013-06-29 The field of renal disease has witnessed a huge increase in new knowledge in the 1990s. Advances in our understanding of the pathogenesis and treatment of this complex group of disorders have been escalating rapidly. This is a third edition of this book, which is intended to provide the physician with a clear, comprehensive text on the management of the diverse array of disorders of

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