PublisherInfo		
PublisherName	:	BioMed Central
PublisherLocation	:	London
PublisherImprintName	:	BioMed Central

Acidosis in diabetics with renal failure

ArticleInfo		
ArticleID	:	4115
ArticleDOI	:	10.1186/ccf-1999-342
ArticleCitationID	:	342
ArticleSequenceNumber	:	52
ArticleCategory	:	Paper Report
ArticleFirstPage	:	1
ArticleLastPage	:	4
ArticleHistory	:	RegistrationDate : 1999–5–24 OnlineDate : 1999–5–24
ArticleCopyright	:	Current Science Ltd1999
ArticleGrants	:	
ArticleContext	:	130541111

Heidi Robertshaw

Keywords

Chronic renal failure, diabetes, metabolic acidosis

Comments

This paper highlights the differences in the acid-base status of diabetics, many of whom are admitted to intensive care units (ICUs). It may be clinically important in those diabetic patients with advanced renal failure, admitted to ICU, to consider such differences in their physiology when using the results of biochemical studies in determining their management. Certainly the importance of ketoacids should be remembered in these patients.

Introduction

Patients with chronic renal failure commonly develop metabolic acidosis. The failure to excrete acid depletes extracellular buffers leading to the biochemical features of reduced serum bicarbonate levels, elevated anion gap with or without hyperchloraemia.Observations have been made that a small proportion of patients with end-stage renal failure can have normal serum bicarbonate with no overt therapeutic intervention. Most of these patients have diabetes mellitus. The mechanism by which these patients maintain normal bicarbonate levels is unclear.

Aims

This study aimed to evaluate whether there is a difference in the prevalence and severity of metabolic acidosis in patients with advanced renal failure with and without diabetes.

Methods

A total of 113 patients were recruited to the study. They were successive referrals to the renal service that met the criteria for entry. The criteria were a creatinine clearance corrected for body surface area of less than 30 ml/min, no alkali intake during the previous 30 days and the absence of pulmonary disease. Forty-eight patients had diabetes mellitus.Samples were taken from these patients in the morning after fasting for 8 h. The samples were analysed for urea, creatinine, sodium, potassium, chloride, calcium, phosphorus, alkaline phosphate, magnesium, albumin, uric acid, glucose, hematocrit, hemoglobin and venous bicarbonate. Urine was collected over 24 h and analysed for urea, creatinine and protein excretion. Anion gap was calculated from (Na + K) ? (Cl + HCO_3) and the result was corrected for plasma albumin concentration.

Results

Mean serum bicarbonate levels were significantly higher and the mean anion gap was significantly less in patients with diabetes. Eight patients had hyperchloremia and were excluded from the study as the authors felt that they may have had a superimposed metabolic alkalosis.Patients were classified according to their bicarbonate, chloride and anion gap serum values. Pure elevated anion gap followed by mixed (elevated anion gap and hyperchloremia) were the most common in both the diabetic and nondiabetic populations. Nine of the 11 patients with normal bicarbonate had diabetes. Nondiabetic patients had more mixed metabolic acidosis.The authors searched for confounding factors that might explain the difference in serum bicarbonates between the groups. Patients with diabetes were more frequently on angiotensin converting enzyme (ACE) inhibitors and they had a slightly better mean renal function.

Discussion

The authors conclude from their results that:

(1) Metabolic acidosis was a very common feature in these patients with advanced renal failure, with a severity related to the severity of their renal failure;

(2) Patients with diabetes, and mainly those with diabetic nephropathy or type I diabetes, had a lower prevalence or a less severe degree of metabolic acidosis than the rest of the patients;

(3) This finding cannot be explained by the use of drugs, reduced protein catabolic rate, or overt gastrointestinal hydrogen ion loss.

The authors conclude that their findings suggest that there is an efficient extrarenal buffer generation in patients with diabetes. Further studies of ketoacid anions, as a potential source of buffer capacity in patients with diabetes, should be undertaken.

References

1. Caravaca F, Arrobas M, Pizarro JL, Esparrago JF: Metabolic acidosis in advanced renal failure: differences between diabetic and nondiabetic patients. Am J Kidney Dis. 1999, 33: 892-898.