are expected to be less than 0.6 mmol/L.

We conclude that the B-OH-B assay does not adequately cross-react with GHB and cannot be used as a rapid serum assay. It remains to be seen whether the higher concentrations of GHB that are seen in the urine of intoxicated patients may yet cross-react with the B-OH-B assay.

This is one case among many that are part of a study approved by our Investigational Review Board

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47/8/126371
doi:10.1067/mem.2002.126371

Recurrent Life-Threatening Acidosis Induced by Acetazolamide in a Patient With Diabetic Type IV Renal Tubular Acidosis

To the Editor:

Carbonic anhydrase inhibitors are widely used for the treatment of wide-angle glaucoma and are usually well tolerated. Severe metabolic acidosis, rarely reported, occurs predominantly among elderly people, in patients with advanced renal failure, in patients on chronic dialysis, and during concomitant use of other nephrotoxic drugs.[1-5]

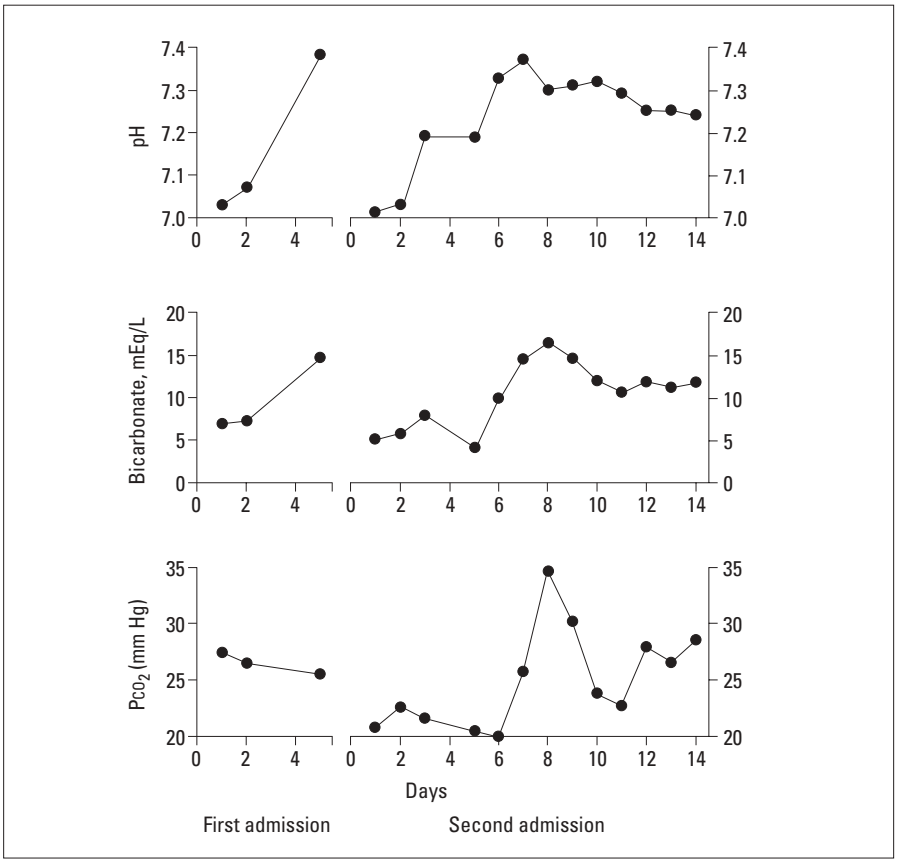
An 80-year-old male patient was admitted with progressive tachypnea and declining mental status. Past medical history included non–insulin-dependent diabetes, moderate chronic renal failure (creatinine 140 to 160 μmol/L), and open-angle glaucoma treated with 250 mg of acetazolamide 3 times a day.

Physical examination was remarkable for severe tachypnea. Laboratory evaluation revealed severe hyperchloremic metabolic acidosis (pH 7.03, bicarbonate 6.9 mEq/L, chloride 123 mEq/L, potassium 5.1 mEq/L, calculated anion gap 16) and deteriorated kidney function (creatinine 250 μmol/L, urea 20 mmol/L). Other laboratory tests, including CBC count, biochemistry, toxic screen, urinalysis, and chest radiograph were unremarkable. Blood, urine, and sputum cultures were sterile.

Aggressive fluid and bicarbonate infusion and discontinuation of acetazolamide resulted in rapid convalescence, with a gradual, though partial normalization of serum pH and bicarbonate (Figure). Their values on discharge were 7.37 and 14 mEq/L, respectively. Plasma creatinine fell gradually to baseline level.

A year later the patient was erroneously prescribed with acetazolamide and was hospitalized again with general deterioration associated with severe metabolic acidosis (pH 7.01, HCO_3 5 mEq/L, Figure). Again, aggressive fluid and bicarbonate infusion and discontinuation of acetazolamide resulted in full recovery, with a substantial, but only partial, normalization of serum pH and bicarbonate levels (7.3 and 12 mEq/L, respectively). On continued evaluation, serum pH was 7.32, bicarbonate was 12 to 14 mEq/L, chloride was 120 to 125 mEq/L, and serum

Figure (Elinav).
*Changes in acid base determinants, pH, bicarbonate (mEq/L), and P_{CO_2} (mm Hg) in the first **(left)** and second **(right)** hospitalization courses.*



potassium was 5 to 5.8 mEq/L, with low urinary potassium excretion and a urine pH of 5. Abdominal ultrasonography findings were normal. These findings are most probably consistent with type IV distal renal tubular acidosis, related to long-standing diabetes mellitus. The patient was discharged with strict precautions of avoiding acetazolamide and has been doing well since that time.

Mild hyperchloremic metabolic acidosis is often encountered among patients treated with acetazolamide. However, severe acidosis seldom develops because bicarbonaturia diminishes during chronic inhibition of carbonic anhydrase and usually disappears at bicarbonate plasma levels below 18 mEq/L.

Rare case reports of severe life-threatening acidosis occur almost exclusively during concomitant use of nephrotoxic medications, in the presence of advanced chronic renal failure, among elderly patients with diminished kidney function, or in patients on chronic dialysis.[2-5]

Acidosis complicating acetazolamide treatment among patients with preexisting renal tubular acidosis has not been well documented. This case stresses this association and emphasizes the potential of the inhibition of proximal tubular bicarbonate regeneration to critically amplify acidosis associated with defective hydrogen ions secretion (as ammonium) in the distal tubule.

Thus, distal renal tubular acidosis should be regarded as a risk factor and has to be excluded in patients at risk (ie, patients with diabetes or those with obstructive uropathy) before the initiation of carbonic anhydrase inhibition.

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47/8/126373
doi:10.1067/mem.2002.126373

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2003 Sports Medicine Subspecialty Examination

The American Board of Emergency Medicine (ABEM), the American Board of Family Practice (ABFP), the American Board of Internal Medicine (ABIM), and the American Board of Pediatrics (ABP) will administer the certifying examination in Sports Medicine on Friday, April 11, 2003.

The eligibility criteria are available from each of the four board offices.

Physicians must submit applications to the board through which they hold their primary specialty certificate. Physicians certified by more than one of the sponsoring boards may select the board through which they apply. On successful completion of the examination, certification is awarded by the board through which the physician submitted the application.

Application materials will be available from the ABEM office on September 15, 2002, and will be accepted with postmark dates through November 15, 2002.

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