A Case of Combined L-Lactate and Renal Tubular Acidosis

To the Editor:

L-Lactate acidosis, a rare acquired metabolic disorder, occurs in patients who have had most of their small intestine resected. It is thought to be due to overgrowth of L-lactate-producing bacteria in the remaining intestine (1). The L-lactic acid produced by these organisms is absorbed from the bowel and enters the blood stream, resulting in a high-anion-gap acidosis. Unrecognized by native mammalian L-lactic acid dehydrogenase, this L-lactic acid is excreted unchanged in the urine.

This condition is well known in veterinary medicine, being observed in ruminants that overfeed on grain (2,3). It has only recently been described in human beings; a review of the recent literature disclosed description of only eight cases (1,4-8).

The patient described here had this condition, which was further complicated by a disorder resembling acquired distal renal tubular acidosis (DRTA).

Over a two-year period a 32-year-old woman presented on nearly 20 occasions with severe metabolic acidosis. She previously had extensive small bowel resection for mesenteric thrombosis, which left her with only the proximal and distal 10 cm of small intestine.

Her plasma analyte values during a later admission, which were typical of those on all admissions, showed (reference intervals in parentheses, all values in mmol/L unless otherwise stated): sodium 138 (132-144), potassium 2.6 (3.2-4.8), chloride 114 (98-108), bicarbonate 8 (23-35), urea 3.3 (3.0-8.0), creatinine 0.052 (0.06-0.12), anion gap 16-17 (8-20), ketones (ketonuric), and lactate 1.0 (0-2). The blood pH was 7.14 (7.35-7.45), the pCO2 17 mmHg (35-45), and the calculated bicarbonate 6 mmol/L (24-32).

Her urinary pH was 5.69 and at no period during any admission did this value drop below 5.60, even though she was severely acidic (blood pH 7.15 to 7.25, plasma bicarbonate 6 to 10 mmol/L).

Further investigations revealed: blood L-lactate 13.2 mmol/L, urinary L-lactate 43.0 mmol/L, urinary titratable acidity (during severe systemic acidemia) 3-9 mmol/min (reference interval: >25 mmol/min), urinary ammonia excretion (during acidosis) 25-56 mmol/min (reference interval: >33 mmol/min). Further investigation of the DRTA was refused by the patient.

After treatment with vancomycin, 250 mg daily, and oral bicarbonate, 42 mmol/day, was begun, the patient's symptoms were relieved, her plasma-biochemistry results returned to normal, and no further episodes of metabolic acidosis occurred for 18 months.

The interesting feature of this patient, other than the D-lactic acidosis, is the DRTA, which was manifested by (a) inability of the kidney to lower the urinary pH below 5.5 during severe systemic acidemia, (b) low urinary titratable acidity, and (c) the mixed hyperchloremic and high-anion-gap acidosis (9).

There are three possible causes of the DRTA in this patient.

Firstly, the patient could have an idiopathic or secondarily acquired DRTA. However, there was no evidence of any of the disease processes (e.g., nephrocalcinosis, autoimmune-type disorders) usually associated with acquired DRTA.

Secondly, the inability of the distal nephron to acidify the urine could be related to the very low pCO2 found in this patient during episodes of metabolic acidosis. There is some evidence, from experiments on dogs, that severe hypocapnia decreases distal renal tubular urinary acidification (10), and this will occur even though the animals have a severe metabolic acidosis (11). However, in these experiments, although there was good evidence for decreased renal excretion of hydrogen ion, no data were given about the urine-cell H+ gradient—i.e., the lowest urinary pH reached.

The third possibility is that L-lactate itself could suppress renal H+ secretion and excretion. However, there is no experimental evidence to support this view.

The latter two possibilities are more likely than is the co-incident occurrence of acquired DRTA and L-lactic acidosis, as three of four of the published cases in which the plasma chloride concentration was reported had hyperchloremia as well as a high-anion-gap metabolic acidosis (1,5,7, i.e., a mixed hyperchloremic and high-anion-gap acidosis. This suggests a concurrent, perhaps transient, DRTA causally related to the L-lactic acidosis.

References:

A. McNeil R. N. Walmsley
Dept. of Clin. Biochem.
Flinders Medical Centre
Bedford Park
South Australia 5042

Clinical Import of Small Increases in Serum Aluminum

To the Editor:

During multi-element screening of blood samples for abnormalities of trace and toxic elements, some groups of patients were found to have above-"normal" values for serum aluminum.

Much of the interest in aluminum in serum has centered on its importance as one cause of degenerative disease of the central nervous system in renal dialysis patients. For many applications relatively simple methods of analysis are appropriate, by which values below 50 µg/L are considered normal. Reported "normal" values vary considerably (1); contamination probably accounts for some of the higher values. Results from the highly sensitive methods now available make it seem probable that the true normal lies below 10 µg/L (2-5).

In reviewing the serum concentrations of aluminum measured in our above-mentioned study, we found that a high proportion of the values between 20 and 50 µg/L seemed to be from patients in four categories:

1. Patients with a history of nonindustrial exposure to aluminum, usually in the form of antacid drugs.