EXPERIENCE WITH THE SHORT AMMONIUM CHLORIDE TEST

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INTRODUCTION

Since renal tubular acidosis is often complicated by stone formation, it seemed justified to investigate a stone forming population in order to study the incidence of renal acidification defects. For this purpose it was necessary to develop a clinically useful method and to standardize the ammonium chloride test.

MATERIALS AND METHODS

Ammonium chloride was given in an oral dose of 17.5 g to all patients except to children, where the dose was reduced in relation to body weight. The ammonium chloride was given as whole tablets, crushed tablets, capsules or in solution. Capsules were found to be absorbed very slowly. Ammonium chloride solution has an awful taste and was found impossible to use. Crushed tablets were found to be the most convenient way to administer the ammonium chloride, because rapid absorption was achieved without too many side effects, especially when given together with a small breakfast.

The degree of metabolic acidosis in each subject was measured in arterialized capillary blood samples using a blood gas analyser (Radiometer, Copenhagen, Denmark). Hourly urine samples were collected during 3-5 hours and analyzed for pH, titratable acid (TA), ammonium ions$^1$ and phosphate. Urinary pH was measured immediately after voiding. The urinary parameters were related to the degree of metabolic acidosis achieved by the ammonium chloride loading.
PATIENTS AND CONTROLS

From the investigation of 47 apparently healthy persons (age 20 to 45 years) recruited from the medical staff, the medical students as well as from a health survey reference values were obtained.

Five hundred eighty-six consecutive stone formers at the stone clinic were investigated using the standardized ammonium chloride test.

BICARBONATE LOADING TEST

To evaluate tubular reabsorption of bicarbonate some patients were investigated by a bicarbonate-loading test. Bicarbonate was given intravenously in a continuous infusion following an induction of acidosis with ammonium chloride the previous day. The glomerular filtration rate was measured by inulin. Arterialized capillary samples were taken for analysis of acid-base status. Urine samples for measurements of pH and \( \text{PCO}_2 \) were taken under anaerobic conditions. The urinary bicarbonate was calculated from the Henderson-Hasselbach equation.

Four patients with normal acidification of the urine as well as five patients with a proximal renal tubular acidosis diagnosed by an ammonium chloride test were investigated.

RESULTS

From the investigation of the apparently healthy persons reference values were calculated for the urinary parameters in relation to the degree of metabolic acidosis obtained as illustrated in Figure 1. The critical level of acidosis appeared to lie around a base excess of \(-6\) mmol/l, below which all subjects could acidify the urine to a pH below 5.0.

The frame of reference was used in the investigation of the stone formers. The distal type of RTA could be clearly distinguished by the inability of acidifying the urine to a value of 5.0 or below, as illustrated in Figure 1. The vast majority of the distal defects belonged to the incomplete type of the renal tubular acidosis (depicted as 2 in Figure 1).

The proximal type of RTA characterized by an increased urinary bicarbonate due to a defective bicarbonate reabsorption in the proximal tubules could also be distinguished by the test. The proximal RTA picture was characterized by an acidification of the urine to a pH below 5.0 but not until a low serum bicarbonate was present as illustrated in Figure 2. Most patients with the proximal defect had the incomplete type of the syndrome (depicted as 4 in Figure 2).