Gastric Hypoacidity in Distal Renal Tubular Acidosis

V.		Sitprija
S.		Eiam-Ong
R.		Suvanapha
P.		Kullavanijaya
S.		Chinayon

Departments of Medicine and Laboratory Medicine, Faculty of Medicine, Chulalongkorn University, Bangkok, Thailand

Prof. Dr. Visith Sitprija, Department of Medicine, Chulalongkorn University Hospital, Bangkok 10500 (Thailand)

Dear Sir,

Failure to acidify urine in the presence of acidosis is the hallmark in the pathophysiology of distal renal tubular acidosis. Whether or not there is a defect of hydrogen ion secretion in the other systems of the body has not been explored. The interrelationship between hydrogen ion transport in the collecting tubules and gastric parietal cells thus deserves investigation. In this report we assorted to study the gastric acidity in 7 patients with primary distal renal tubular acidosis of secretory type. The patients were all female and their ages ranged from 10 to 49 years. They presented with proximal muscular weakness. The laboratory data on admission revealed serum bicarbonate ranging from 6 to 20 mmol/l, blood pH from 7.1 to 7.38, serum potassium from 1.4 to 2.6 mmol/l, serum creatinine from 44.2 to 97.3 μ mol/l, and urine pH from 6.0 to 7.0. Phosphate infusion (1 mmol/l total body water) elicited the difference between urine and blood pCO₂ below 25 mm Hg being consistent with hydrogen ion secretion deficit [1]. Gastric analysis was performed by a standard method [2] a few weeks following alkaline treatment when hypo-kalemia and acidosis had been corrected with clinical recovery. Alkaline treatment was discontinued for 24 h before the test. A baseline gastric acidity was obtained by gastric fluid collection during 4 periods of 15 min each. This was followed by intramuscular injection of penta-gastrin (6 μ g/kg) with continued gastric fluid collection for 1 h at the same interval. The maximal and peak acid secretions were calculated. The results showed low basal acid secretion when compared with the control group of 13 normal subjects. However, the difference was not statistically significant. The maximal and peak acid se-

cretions were significantly lower than the control values ($p < 0.05$; table I). The data thus show the decreased basal gastric acidity and decreased response to pentagastrin stimulation. These patients did not have iron deficiency anemia, pernicious anemia or clinical evidence of gastric diseases to account for hypoacidity. The findings are of interest for two reasons. First, it indicates that in renal tubular acidosis the defect in hydrogen ion transport is not confined only to the renal tubules. Second, there may be some similarity between hydrogen transport in the collecting tubules and gastric parietal cells, or a common mechanism may be shared by both systems. It has been shown that hydrogen ion secretion by the gastric parietal cells requires H⁺/K⁺-ATPase

[3]. The enzyme is also found in the colonic mucosa [4]. In rabbits and rats H^+/K^+ -ATPase activity has been detected in the distal nephrons in proportion to the density of intercalated cells

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with the highest activity in the connecting tubules [5]. The enzyme, originating in intercalated cells, is involved in potassium reabsorption and hydrogen ion secretion, and is vanadate- and omeprazole-sensitive [5]. The enzyme activity is enhanced in potassium depletion. Our findings in man are therefore of physiological importance and will have to be confirmed in a larger number of patients. Further study is warranted.

References

Kurtzman, N.A.: Acquired distal renal tubular acidosis. *Kidney int.* 24:807–819(1983).

Feldman, M.: Gastric secretion; in Sleisenger, Fordtran, *Gastrointestinal disease: pathophysiology, diagnosis, management*, pp. 541–558 (Saunders, Philadelphia 1983).

Saccomani, G.; Helander, H.F.; Crago, S.; Chang, H.H.; Dai-ley, D.W.; Sach, G.: Characterization of gastric mucosal membranes. *Immunological studies of gastric ($H^+ + K^+$) ATPase*. *J. Cell Biol.* 55:271–283(1979).

Gustin, M.C.; Goodman, D.B.P.: Isolation of brush-border membrane from the rabbit descending colon epithelium. Partial characterization of a unique K^+ -activated ATPase. *J. biol. Chem.* 256:10651–10656 (1981).