Brief Note

Autoantibodies to ACE (Angiotensin Converting Enzyme) in a Patient with Pseudo-Bartter's Syndrome Due to Laxative Abuse

Shoji SUMITOMO, Michihiro MATSUKI, Tamaki SASAKI* and Gengo OSAWA*

*Division of Endocrinology, *Division of Nephrology, Department of Medicine, Kawasaki Medical School, Kurashiki 701-01, Japan*

Accepted for publication on July 12, 1994

Key words: pseudo-Bartter's syndrome—laxative abuse—autoantibodies to ACE

Bartter's syndrome is characterized by hypokalemia, normotensive hyperreninemia, hyperaldosteronism and vascular insensitivity to stimulation with angiotensin II.[1,2] It is often difficult to distinguish Bartter's syndrome from pseudo-Bartter's syndrome,[2] a condition which may be caused by diuretic or laxative abuse. Recently, Nishida et al found that in patients with Bartter's syndrome or related disease, increased plasma renin activity (PRA) may be associated with a change in the angiotensin I / II ratio due to inhibition of angiotensin converting enzyme (ACE).[3] Furthermore, they found the circulating autoantibodies to ACE to be a possible cause of the ACE inhibition in patients with Bartter's syndrome.[4,5]

In the present study, we examined the anti-ACE autoantibodies in the serum from a 50-yr-old female patient with pseudo-Bartter's syndrome due to laxative abuse. The patient had been talking 10 laxative tablets (Sennoside Ca) once at night for 20 years. Her height was 155 cm and her body weight was 43 kg. Her blood pressure was 114/70 mmHg. Laboratory findings revealed: serum K, 2.5 mEq/L; Na, 137 mEq/L; Cl, 97 mEq/L; PRA, 21.0ng/mL/h (normal range, 0.3-2.9); plasma angiotensin (ANG) I, 1300 pg/mL (normal range, below 250); ANG II, 110 pg/mL (normal range, below 25); ACE activity, 11.4IU/L/37C (normal range, 8.3-21.4); bradykinin, 63.0 pg/mL (normal range, 9.6-21.0); and serum aldosterone, 310pg/mL (normal range, 35-240). Histopathologic examination of the kidneys ealed no juxtaglomerular hyperplasia.

Results of immunological studies. As shown in Fig 1, though IgM antibody was not detected, IgG antibody to ACE was found by the method previously reported.[4,5] The association constant (Ka) and the capacity of the antibody were $1.8\times10^{8}M^{-1}$ and $0.5\times10^{-11}M$, respectively.

It is very interesting that circulating autoantibody to ACE was detected in a patient with pseudo-Bartter's syndrome due to laxative abuse. Nishida et al[5] reported that both IgG antibody and IgM antibody to ACE were found in adult patients with Bartter's syndrome. In the patient in the present study, only IgG antibody was found to be positive. Nishida et al[5] also reported that IgG

住友正治，松木道裕，佐々木環，大澤源吾

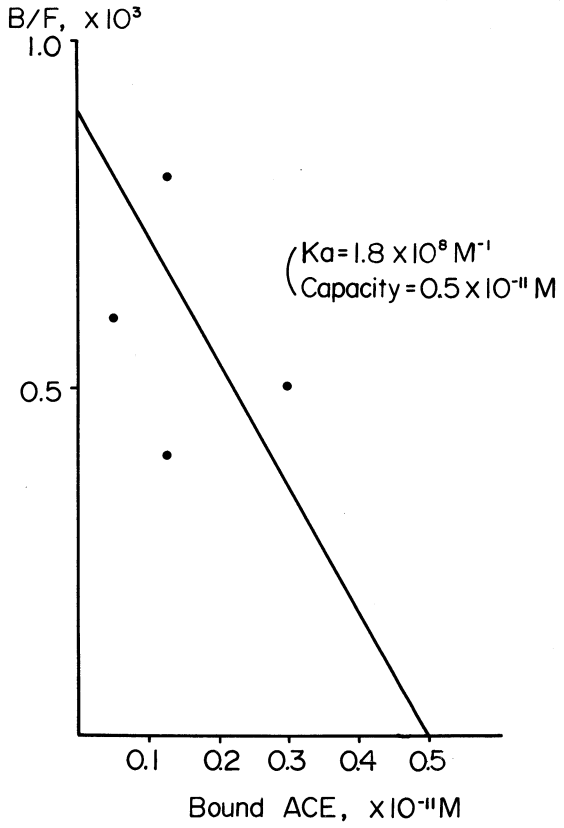

Fig 1. A scatchard analysis of the anti ACE-IgG antibody of the patient determined by the method previously reported (cf. text).

autoantibodies to ACE, though not for IgM, were found in five out of the six patients with chronic edema who were taking furosemide in large amounts for long periods, and that these five patients revealed no signs or symptoms of Bartter's syndrome. Thus, it is unclear if these circulating autoantibodies to ACE are causative of Bartter's syndrome.

REFERENCES

1) Bartter FC, Pronove P, Gill JR Jr, MacCardle RC : Hyperplasia of the juxtaglomerular complex with hyperaldosteronism and hypokalemic alkalosis. Am J Med **33** : 811-828, 1962
2) Gill JR Jr : Bartter's syndrome. Ann Rev Med **33** : 405-419, 1980
3) Nishida S, Matsuki M, Tsushima K, Kawai Y, Yoneda M, Oyama H, Horino M, Andoh C : An increase in the ratio of ANG I to ANG II due to ACE inhibition in patients with Bartter's syndrome and related disease. Folia Endocrinol **66** : 436, 1990 (in Japanese)
4) Nishida S, Matsuki M, Horino M, Nohno T, Fukushima T, Ohmori H, Yamaguchi K : Autoantibodies to ACE in patients with Bartter's syndrome. Folia Endocrinol **69** : 347, 1993 (in Japanese)
5) Nishida S, Matsuki M, Nohno T, Morita T, Ohsawa G, Ohmori H, Ono J and Yamaguchi K : Autoantibody (IgM) to ACE in patients with Bartter's syndrome. Folia Endocrinol **70** : 178, 1994 (in Japanese)