KAWASAKI MED. J. VOL. 14 : NO. 4, 207-209 (1988)

Brief Note

Bartter's Syndrome and Pseudo-Bartter's Syndrome

Accepted for publication on January 6, 1989

Key words : Bartter's syndrome — pseudo-Bartter's syndrome — furosemide abuse — anorexia nervosa

Bartter's syndrome is characterized by hypokalemia, normotensive hyperreninemia, hyperaldosteronism and vascular insensitivity to stimulation with angiotensin II.[1,2] And 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 or may be accompanied with anorexia nervosa. In this paper, we describe four patients who presented with hypokalemia. In one of these four patients, observations led to the diagnosis of Bartter's syndrome, and remaining three patients were distinguished from Bartter's syndrome as having pseudo-Bartter's syndrome, since two of the three patients admitted to taking of furosemide (diuretic), and one of them showed anorexia nervosa.

As shown in Table 1, the patients with pseudo-Bartter's syndrome as consequence of diuretic abuse or one such patient accompanied with anorexia nervosa were not clearly distinguished from the patient with Bartter's syndrome by any examination, though urinary chloride determinations[3] or urinary potassium determinations in response to spironolacton[4] may be of considerable value for the purpose. Diuretic abuse generally has been dismissed on the basis of the patient's denial of this ingestion even if being mentioned of search for diuretics in the urine. Therefore, it is recommended to perform an assay for diuretics in the urine in patients who have obscure hypokalemia to exclude self-intoxication with diuretics to attain a slim figure. Although the cause of Bartter's syndrome is still controversial, excess renal prostaglandin production[5] and an intrinsic prostaglandin-independent impairment of chloride reabsorption by the loop of Henle[6] have been suggested as etiological factors. Recently, very interesting studies are reported concerning the action of furosemide that furosemide releases renal prostaglandin[7] or it acts at the thick ascending limb of Henle's loop resulting in an impairment of chloride reabsorption.[8] This suggests that Bartter's syndrome and pseudo-Bartter's syndrome (due to diuretic abuse) may develop on the same pathognomonic basis.

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TABLE 1. Clinical and laboratory data of the patients

	Case 1	Case 2	Case 3	Case 4	Normal range
Age, sex	29, M	26, F	46, F	20, F	
Cause, complication		furosemide	furosemide	anorexia nervosa	
Na (mEq/l)	136	131	135	132	134 - 144
K (mEq/l)	1.7	1.9	1.9	1.7	3.2 - 4.7
Cl (mEq/l)	91	82	87	66	98 - 107
Plasma renin activity (PRA, ng/ml/hr)	18.90	36.42	38.20	14.84	0.5 - 2.0
Angiotensin I (pg/ml)	844	5500	4600	1400	below 250
Angiotensin II (pg/ml)	18.5	200	150	59	below 25
Aldosterone (pg/ml)	140	275	379	472	below 180
Angiotensin converting enzyme (ACE, IU/l/37 C)	12.4		8.7		8.3 - 21.4
Prostaglandin E (pg/ml) ;					
blood		182	165		57 - 1144
urine	2790	156			
Blood gas analysis ;					
pH	7.495	7.512	7.466	7.544	7.36 - 7.46
HCO_3^-(mEq/l)	28.5	39.5	30.3	47.7	24.0 - 32.0
base excess (mEq/l)	6.1	15.3	6.5	22.6	−2.5 - 2.5
Kallikrein (urine, ng/ml)		45			11 - 980
Vascular reaction to angiotensin II	poor	poor			
Pathology		no findings		hyperplasia of JG apparatus, positive granules of Bowie's stain	

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