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Hormonal Mechanisms

5.3 Secondary Hyperparathyroidism to Hyperaldosteronism

F. Fortina, S. Agliata, S. Cusinato, E. Ragazzoni, P. Carpani, D. Motta, A. Cavgnino

Ospedale di Borgomanero, Borgomanero, Italy

Introduction: Hyperparathyroidism has been associated to essential hypertension. Parathormone (PTH) has direct effect on renin secretion. Volumes extensions, DOC-salt model and primitive hyperaldosteroidism in rats, decrease plasmatic Ca++ and cause secondary hyperparathyroidism. In these rats urinary Ca decreases with tiazidic and/or antialdosteronic, while the furosemide gives opposite effect. One case of parathyroide and surrenalic adenoma, and breast cancer, variation of MEN 1, has been described in men. Liquorice, as well, causes an increase of PTH and Cau.

Methods: In order to estimate these information 13 patients have been studied: 2 of them with primitive hyperaldosteronism, 4 with adenomas and 9 with bilateral surrenalic hyperplasia. Only 6 of them with renal colics. The protocol stated seric and urinary PTH, K, Ca, P dosage, before and after the beginning of antialdosteronic therapy.

Results: All had an increased PTH that decreased after the therapy (*t-test< 0,05) [see table].

	Ca mg/dl	P mg/dl	Cau mg/24 h*	Pu mg/24 h*	PTH pg/ml*	K mEq/l*
PRE	9,1	2,9	302	1099	95	3,6
POST	9,5	3,	163	566	72	4,6

Conclusions: It is supposed that hyperaldosteronism can provoke a contemporary urinary K, Ca and P loss as well as stimulate the secretion of parathormone. Antialdosteronic has the function to decrease urinary K, Ca and P and to increase the calcaemia inhibiting the PTH. In nephrolithiasis natural history the presence of ipocalaemia, renal calculosis and hypertension is often observed. Hyperaldosteroidism delayed diagnosis increases PTH as well as nefrolitiasi risk. As for the essential hypertensives simil Lidde and simil AME (our data) and in liquorice abuse, urinary K, P and Ca loss seems to be the primum movens of nephrolithiasis also for these patients. Though in the first two cases there are genetic tubular anomalies, in the second two aldosterone and liquorice should be the causes. Our data seem to confirm the hypothesis of hyperparathyroidism from hyperaldosteroidism. Antihypertensive therapy based on antialdosterone drugs normalise almost all parameters considered in the present sudy. Therefore there are interrelations between hormones, although apparently different, and between these and the renal tubule that, if not prematurely diagnosed, also favour litogenesis.