THE EVALUATION OF RENIN-ANGIOTENSIN-ALDOSTERONE SYSTEM IN PATIENTS WITH ASYMPTOMATIC PRIMARY HYPERPARATHYROIDISM

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INTRODUCTION

There is some evidence suggesting an interaction between renin-angiotensin-aldosterone system and parathyroid hormone (PTH). Decreased aldosterone levels after parathyroidectomy have been reported in patients with primary hyperparathyroidism (PHPT). Recently, most of the diagnosed patients with PHPT are mild or asymptomatic. Our aim was to investigate plasma aldosterone and renin concentrations in patients with asymptomatic PHPT.

METHODS

Thirty-two patients with asymptomatic PHPT and 22 healthy control subjects were recruited for this study. The levels of renin, aldosterone, PTH, calcium, phosphorus and 25-OH vitamin D were investigated.

RESULTS

Median PTH and calcium levels were significantly higher in the patients with asymptomatic PHPT than the control group [155.0 (77.60) pg/ml vs 54.0 (13.20) pg/ml and 10.80 (0.45) vs 9.48 (0.56) mg/dl,$p<0.05$, respectively)]. Median creatinine clearance levels were similar [100 ml/min (22.80) vs 102 ml/min (19.73), $p>0.05$, respectively]. There was no statistically significant difference between plasma renin levels and plasma aldosterone concentrations [5.98 (7.35) vs 5.02 (12.87) and 95.14 (88.77) vs 105.92 (55.17), $p>0.05$, respectively]. There were no correlations between calcium and PTH levels and renin or aldosterone levels ($p>0.05$).

Table 1. Demographic and biochemical characteristics of the groups

	Asymptomatic PHPT (n=32)	Controls (n=22)	P value
Age (years)	53 (37-69)	58 (37-67)	p>0.05
BMI	28.0 (18.8-36.6)	26.9 (23.1-35.7)	p>0.05
Gender (F/M)	28/4	18/4	p>0.05
Creatinine (mg/dL)	0.7 (0.5-1.0)	0.7 (0.5-0.9)	p>0.05
Creatinine clearance (mL/min)	100.0 (64.0-163.0)	102.0 (71.0-123.0)	p>0.05
Calcium (mg/dL)	10,8 (10,1-11,3)	9.4 (8.8-9.9)	**p<0.05**
Phosphorus (mg/dL)	2.7 (1.9-3.6)	3.5 (3.0-4.1)	**p<0.05**
Parathormone (ng/L)	155.0 (64.5-736.0)	54.0 (33.4-70.3)	**p<0.05**
25 (OH) vitamin D (ng/mL)	12.7 (3.8-62.5)	20.7 (5.9-33.1)	p>0.05
Albumin (g/dL)	4.2 (3.9-5.0)	4.3 (4.0-4.8)	p>0.05
Aldosterone (ng/dL)	95.0 (41.0-192.0)	105.9 (37.3-265.0)	p>0.05
Renin (mU/L)	5.9 (0.5-34.8)	5.0 (0.8-35.1)	p>0.05

DISCUSSION

Some recent evidence suggests that there may be a complex interaction between PTH and RAAS [1] . Regarding the possible mechanisms related to this association between PTH and RAAS, it has been proposed that the acute modulation of PTH by the RAAS seems to be mediated by Angiotensin II, whereas the long-term influence of the RAAS on PTH may involve aldosteron [2]. In our study, plasma aldosterone concentrations were found to be similar between patients with asymptomatic PHPT and control subjects. This result may be related to mild hypercalcemia which is not enough to expose some alterations in RAAS in our patients with asymptomatic PHPT.

In the present study, we also found that plasma renin levels were similar between two groups. In consistent with our finding, Valvo et al. [3] reported no change in plasma renin activity after parathyroidectomy in patients with PHPT. Regarding the association between renin, PTH and calcium, Atchison et al. showed that acute hypercalcemia suppresses plasma renin activity with the effect of high levels of PTH in renal cortical interstitial area through juxtraglomerular calcium-sensing receptor cells (CaSR) [4]. In contrast, it has been suggested that plasma renin activity may increase due to polyuria mediated by chronic hypercalcemia [22]. Therefore, the possible effects of high PTH on renin levels are not well known. One can hypothesize that renal blood flow may be a reason for the alteration in renin levels in PHPT. In our study, creatinine clearance and creatinine levels between two groups were similar. This may suggest that in asymptomatic PHPT group, renal blood flow was not affected considerably – which, in turn, caused similar renin levels.

One of the limitations in our study is our measurement of plasma renin level instead of renin activity. It would have been more significant if we measured renin activity and calculated aldosterone/renin ratio. However, we think that the evaluation of renin levels instead of renin activity is also suitable because we found no change in aldosterone levels.

In conclusion, our results suggest that, in patients with asymptomatic PHPT who do not need parathyroid surgery, no significant change occurs in RAAS as reflected by circulating concentrations of renin and aldosterone. For patients with PHPT, further research is necessary to reveal whether substantial changes occur in RAAS, and if so, what is the importance of this activation.

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