**SHORT COMMUNICATION**

**Prolactin in primary aldosteronism**

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**Summary**

1. This study was done to examine the possibility that escape from the sodium retention produced by aldosterone may be associated with an inhibition of prolactin secretion. Plasma prolactin concentrations were determined in seven patients with adrenal adenoma and aldosteronism, before and after unilateral adrenalectomy, at a time when they were in balance on a sodium intake of 109 mmol/day.

2. After operation, plasma aldosterone was normal [before operation, 52.7 ± 12.1 (supine), 64.6 ± 9.1 ng/dl (upright); after operation, 6.8 ± 1.5 (supine), 11.1 ± 3.0 ng/dl (upright)], while plasma prolactin remained unchanged [before operation, 19.6 ± 2.8 (supine), 15.5 ± 3.3 ng/ml (upright)]; after operation, 23.8 ± 2.3 (supine), 11.1 ± 3.0 ng/ml (upright)].

3. Our results do not support a role for prolactin in the renal response to aldosterone in man.

Key words: aldosterone, prolactin, renal sodium excretion.

**Introduction**

Several investigators have suggested a role for prolactin in the regulation of water and of sodium chloride excretion by the kidney (Burstyn, Horrobin & Manku, 1972; Horrobin, Lloyd, Lipton, Burstyn, Durkin & Minuri, 1971; Horrobin, 1973; Richardson, 1973), and some investigators have even proposed prolactin as a mediator of the renal effects of aldosterone (Burstyn et al., 1972; Horrobin, 1973). The studies by Berl, Brautbar, Ben-David, Czaczkes & Kleeman (1976) and Carey, Johanson & Seif (1977) have excluded a significant role for prolactin in the regulation of water excretion by the kidney, at least in man. Other studies indicate that the hypothesis that an inhibition of prolactin release leads to escape of the kidney from the sodium retention produced by sodium-retaining steroids is also very unlikely (Ogihara, Matsumura, Onishi, Miyai, Uozumi & Kumahara, 1977; Olgaard, Hagen, Madsen & Hummer, 1977). The findings that stimulation of prolactin release by thyrotrophin-releasing hormone was unaltered during escape from sodium-retaining effects of 9α-fluorocortisol in normal man and that the increase in prolactin had no effect on sodium excretion have cast doubt on any inter-relationship between aldosterone and prolactin in the regulation of renal function (Baumann & Loriaux, 1976). Also, plasma prolactin concentration in patients with primary aldosteronism was not different from that in normal control subjects (Holland, Gomez-Sanchez, Kem, Weinberger, Kramer & Higgins, 1977). To pursue the question of whether aldosterone has any effect on plasma prolactin concentration, we have studied the relationship of plasma aldosterone and prolactin concentrations in seven patients with primary aldosteronism before and after unilateral adrenalectomy.
Methods and results

The studies were performed before and at least 2 weeks after operation in seven patients with primary aldosteronism, in whom an adrenal adenoma was confirmed by postoperative histological examination. All medications were discontinued 4 weeks before admission to the metabolic
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unit for study. The patients were given a diet which contained 109 mmol of sodium/day and studies were performed when the patients were in balance. Blood for measurement of plasma aldosterone and prolactin was drawn at 08.00 hours, with patients supine, and at 11.00 hours, after the patients had been walking or standing for 3 h. Plasma aldosterone (Ito, Woo, Hanning & Horton, 1972) and prolactin (Aubert, Becker, Saxena & Raiti, 1974) were determined by radioimmunoassay and serum sodium and potassium concentrations were measured by flame photometry. Statistical analysis of data was performed with a Student's paired t-test.

The results are shown in Fig. 1. Operation, which corrected elevated plasma aldosterone and hypokalaemia (4.9 ± 0.1 mmol/l after operation; 2.9 ± 0.2 mmol/l before operation) in all patients, had no detectable effect of prolactin.

Conclusions

Our results indicate that an increased production of aldosterone, with escape from its sodium-retaining action, does not affect prolactin concentration or its responsiveness. These findings, taken together with those cited above, give no support to the view that prolactin participates in any way in the renal response to aldosterone in man.

References


