GLUCOCORTICOID AXIS IN PATIENTS WITH PRIMARY ALDOSTERONISM

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OBJECTIVES
To assess glucocorticoid axis in patients with primary aldosteronism as compared to patients with ACTH-independent Cushing syndrome and control hypertensive patients without gluco or mineralocorticoid excess.

BACKGROUND
Primary aldosteronism is associated with increased prevalence of cardiometabolic complications1. The mechanisms are not fully elucidated, but an association with autonomous cortisol secretion could increase vascular and metabolic risk2.

METHODS
- 22 patients with primary aldosteronism - PA (10M/12F, aged 43.9±11.2 years) (14 adrenal tumors, 8 uni/bilateral adrenal hyperplasia) – group 1
- 13 patients with ACTH-independent Cushing syndrome (2M/11F, aged 43.8±13.3 years) – group 2
- 42 control hypertensive patients Ctrl (16M/26F, aged 35.8±14.5 years) – group 0 - were retrospectively reviewed.

Plasma aldosterone and plasma direct renin: chemiluminiscence (method’s sensitivity 2.2 ng/dl for aldosterone and 0.27 ng/dl for renin, respectively);

Serum cortisol and ACTH: electrochemiluminiscence.

RESULTS
BMI, maximum systolic blood pressure, fasting glycaemia and total cholesterol were similar in the three groups.

Serum kalemia in patients with PA (2.6±0.5 mmol/l) was significantly lower than in patients with ACTH independent Cushing syndrome (4.3±0.9 mmol/l, p<0.0001) and in control patients (4.4±0.4 mmol/l, p<0.0001).

ACTH levels were similar in PA patients and in control group.

One operated patient with PA developed postoperative adrenal insufficiency and required glucocorticoid replacement therapy.

*DXM- Dexamethasone

CONCLUSIONS
Mild cortisol excess may co-exist in primary aldosteronism patients, reflected by higher cortisol levels after overnight low-dose dexamethasone test.

REFERENCES