Quiz of the Month

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Questions

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A 51-year-old White woman with a 12-year history of hypertension had been treated with a variety of medications, the most recent being guanfacine HCl 3 mg per day. She complained of easy fatigability, nocturia 3 times per night, headaches, and occasional weakness, muscle cramps, and orthostatic dizziness. She denied vomiting, diarrhea, or use of illicit drugs, estrogens, or diuretics.

Physical examination revealed a normal-appearing middle-aged woman in no distress. She was afebrile, weighed 61 kg, supine BP was 184/108 mm Hg, pulse 86 bpm; standing BP 146/96 mm Hg, pulse 92 bpm; respirations 12. Retinal fundi showed grade 11/VIK-W retinopathy; neck, heart, and lungs were normal; there were no abdominal bruits or masses; pelvic/rectal examination was normal; there was no edema; neurologic examination was unremarkable except for mild, generalized weakness.

Initial laboratory screening revealed plasma Na⁺ 148mmol/l, K⁺ 2.8mmol/l, Or 110mmol/l, tCO₂ 32mmol/l, BUN 14mg/dl, creatinine 1.0mg/dl, glucose 14mg/dl, calcium 9.2mg/dl, phosphate 3.4mg/dl, uric acid 6.3 mg/dl, urine specific gravity 1.005, pH 6.8, no proteinuria, and benign sediment. The EKG was compatible with left ventricular hypertrophy, and chest x-ray was within normal limits.

24-hour urinary Na⁺ 103mmol, K⁺ 47 mmol, Cl⁻ 92 mmol, creatinine 957 mg. Plasma aldosterone ([aldo]) 24.8 ng/dl and plasma venous renin activity (PVRA) 0.9 ng/ml/h after 2 h in an upright posture; [aldo]p 6.4 ng/dl after infusing 2 liters normal saline in the supine position. Computerized tomographic scan (CTS) of the abdomen with 4-mm cuts through the adrenals was within normal limits.

Questions

1 What is the diagnosis?

A Renovascular hypertension (fibromuscular dysplasia)

B Liddle's syndrome;

C Idiopathic hyperaldosteronism (IHA, bilateral adrenal hyperplasia);

D Primary hyperaldosteronism (PHA, aldosterone-producing adenoma);

E Pseudohyperaldosteronism.

2 What further diagnostic study would be least helpful? A Plasma aldosterone after dexamethasone suppression;

B ^{99m}Tc-DTPA renal flow scan 1 h after 25 mg captopril per os;

C Plasma 18-hydroxycortisol after saline suppression;

- D Selective bilateral adrenal venous plasma aldosterone ([aldo]p);
 E [¹³¹I]iodocholesterol adrenal scintiscan with dexamethasone.
 3 What treatment would you initially prescribe?
 A Amiloride;
 B Nifedipine;
 C Lisinopril;
 D Hydrochlorothiazide with dietary Na⁺ restriction;
 E Spironolactone;
 F Surgical exploration.

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- 4 Which are the primary aldosterone secretagogues?
 A Angiotensin II (A-II), hyperkalemia, aldosterone-stimulating factor (ASF), parathyroid hormone (PTH), catecholamines;
 B A-II, hyponatremia, adrenocorticotrophic hormone (ACTH), 8-arginine vasopressin (AVP), serotonin;
 C A-II, hyperkalemia, α-melanocyte-stimulating factor (αMSF), (3MSF, (3-endorphins, insulin;
 D A-II, hyperkalemia, hyponatremia, ACTH.
 True or false: aldosterone stored in zona glomerulosa secretory granules is released by aldosterone secretagogues.
 True or false: all acute aldosterone regulatory factors are known to modify the late pathway of aldosterone synthesis (18-hydroxylase conversion of corticosterone to aldosterone).
 True or false: low-Na⁺ and high-K⁺ diets primarily affect the early pathway (20,22-desmolase conversion of cholesterol to pregnenolone).

True or false: the mechanisms by which diet modifies aldosterone secretion are unknown.
 True or false: the steroid hormone binding affinities of the six types of steroid receptor [mineralocorticoid (MC), glucocorticoid (GC), androgen, progesterone, estrogen, and vitamin D] are each highly specific for their particular class of hormone, with the exception of progesterone and estrogen receptors.
 True or false: the specificity of MC target tissues (e.g., toad bladder, sweat gland, kidney, parotid gland, and distal colon) for aldosterone is receptor, not enzyme mediated.
 True or false: aldosterone increases synthesis of Na⁺, K⁺-ATPase, citrate synthetase, and flavokinase in its target tissues.
 True or false: aldosterone activity in the cortical collecting duct is mainly due to increase conductance through principal cell apical Na⁺ ion channels, whereas its activity in the outer medullary collecting duct is mainly due to increase proton conductance through α-intercalated cell apical H⁺ ATPase.