中文題目:以多汗為初期表現的亞臨床高皮質醇症-病例報告

英文題目: Hyperhidrosis as Initial Presentation of Subclinical Hypercortisolism - Case

Report

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INTRODUCTION:

Subclinical hypercortisolism, or subclinical Cushing syndrome, is excessive cortisol secretion with no specific sign or symptom of overt Cushing syndrome. Here we report a case of autonomous cortisol-secreting adrenal nodule who had hyperhidrosis as initial presentation.

CASE REPORT:

A 31-year-old woman who was previously healthy presented to our endocrinology clinic for several months of easy sweating during her sleep at night. The first episode of excessive sweating happened two years ago and relieved spontaneously. The sweating was so serious that she had to change her wet bed. Several months ago, the sweating happened again and persisted almost every night. The patient also had bodyweight gain about 10 kilograms over 2 months. There was no fatigue, change in appetite, menstrual abnormality, easy bruising, facial plethora, muscle weakness, or abdominal striae. She did not smoke or drink alcohol.

The patient visited a clinic for weight gain and was prescribed fludiazepam, fluoxetine, topiramate, fenoterol, caffeine, thiamine, pseudoephedrine and aminophylline. She took these medication intermittently. The excessive sweating started before she took weight loss pills.

On physical examination, her height was 160cm and bodyweight was 62kg (body mass index of 24kg/m^2). Her vital signs were normal. The thyroid was normal in size, without palpable nodule. Laboratory tests showed normal thyroid function, normal lipid profile, normal growth hormone and insulin-like growth factor-1 level. 24-hour urine vanillyle-mandelic-acid, dopamine, epinephrine, and norepinephrine were within normal range. Baseline morning cortisol was $5.87~\mu$ g/dL and adrenocorticotropic hormone (ACTH) was 20.1pg/mL. However, the overnight 1mg dexamethasone suppression test showed cortisol $4.4~\mu$ g/dL. One month later she was admitted to our ward for 2-mg 48-h dexamethasone suppression test. The baseline morning (at 08:00) serum cortisol level was $4.57~\mu$ g/dL and plasma ACTH was 2.21~pg/mL. The baseline night (at 23:00) cortisol was $4.08\text{g}/\mu$ dL and ACTH was 2.21~pg/mL. Baseline 24-hour urine free cortisol was $5.13~\mu$ g/day. The post 2mg 48-hour dexamethasone suppression cortisol was $5.34~\mu$ g/dL.

For non-suppressed cortisol and low ACTH, contrast-enhanced computed tomography (CT) of adrenal was arranged. The adrenal CT showed a 1.1cm x 0.8cm nodule at the right adrenal gland.

The pre-contrast attenuation was 20.35 Hounsfield units. The contrast enhancement washout was 45% at 15 minutes (greater than 60% is characteristic of adrenal adenoma).

After the functional test, the hyperhidrosis relieved again. Her bodyweight was stable without taking those weight loss pills. Adrenal image and hormone testing will be repeated 6 months later.

DISCUSSION

Subclinical hypercortisolism, or subclinical Cushing syndrome, is characterized by chronic subtle cortisol excess in patients with no specific signs and symptoms of Cushing syndrome such as moon face, buffalo hump, easy bruising, facial plethora, proximal muscle weakness and purple striae. Autonomous cortisol secretion is found in about 0-11% of patients with adrenal incidentalomas. Continuous low grade of cortisol exposure may be associated with hypertension, glucose intolerance or type 2 diabetes mellitus, dyslipidemia, obesity, osteoporosis and vertebral fracture. However, there is clear evidence that patients with autonomous cortisol secretion very rarely develop overt Cushing syndrome.

There is lack of consensus in diagnosing autonomous cortisol secretion. The most acceptable screening test is dexamethasone suppression test. Post-dexamethasone serum cortisol levels between 1.9-5.0mcg/dl should be considered as possible autonomous cortisol secretion and serum cortisol more than 5.0mcg/dl should be taken as autonomous cortisol secretion. Additional tests might be required to confirm the cortisol secretory autonomy and to assess the degree of cortisol secretion. Measurement of basal morning plasma ACTH level and repeated the dexamethasone test after 3-12 months was suggested in all patients with possible autonomous cortisol secretion; while in patients with autonomous cortisol secretion, the additional measurement of 24-hour urinary free cortisol was recommended.

Clinical reassessment should be done annually to evaluate the potentially cortisol excess-related comorbidities. The presence or worsening of any conditions should re-evaluate the hormonal status at any time during follow up. Symptomatic treatment and surgical removal of the adrenal mass should be taken into consideration.