CUSHING'S DISEASE, HASHIMOTO'S THYROIDITIS AND SEVERE HYPERANDROGENEMIA

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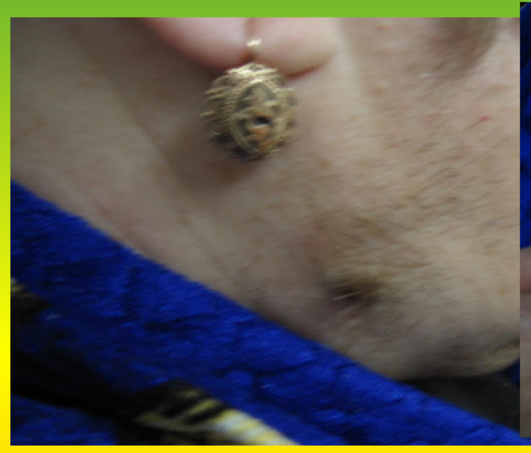
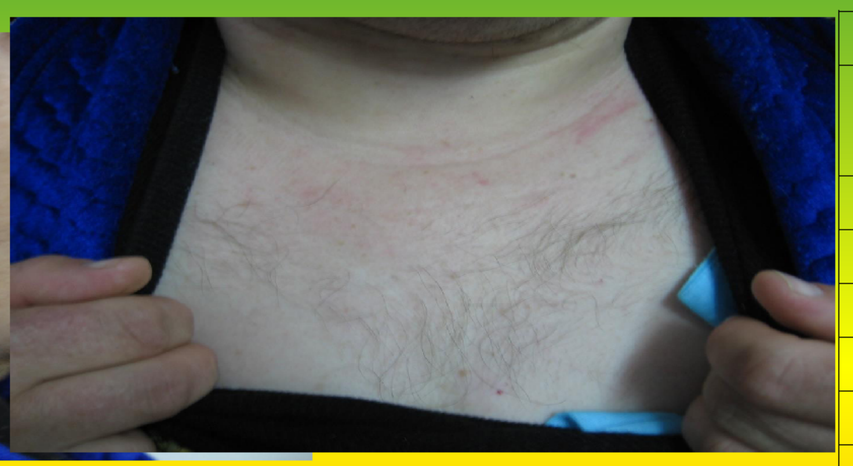
Introduction

Cushing's disease (CD) is caused by high adenocorticotropic hormone (ACTH), usually by a pituitary microadenoma.

Material & Methods

This is a case report investigated in several Romanian centers by performing: morning plasma cortisol, circadian rhythm of plasma cortisol, plasma ACTH, dexamethasone (DXM) suppression tests.
The endocrine tests results are presented.





Variable	Result	Normal range
Urinary free cortisol/24h	200.7 µg/24h	50-190 µg/24h
ACTH	24.56 pg/ml	7.2-63.3 pg/ml
TSH	1.24 µUI/ml	0.4-4 µUI/ml
FT4	1.06 ng/dl	0.89-1.76 ng/dl
25-OHD	21.3 ng/ml	30-100 ng/ml
Beta-crosslaps	0.36 ng/ml	≤ 0.584 ng/mL
Osteocalcin	20.9 ng/mL	14-42 ng/mL

Case data

A 57- year female, known with hypertension and osteopenia, was admitted for high blood pressure, fatigue, anxiety, sweating and hirsutism. Clinical examination revealed: classic features of Cushing's disease: moon face, plethora, hirsutism (on upper lip, lower jaw, sideburn, lower abdomen and thigh; Ferriman-Gallwey score of 18, centripetal obesity; buffalo hump, enlarged supra-clavicle fat pads and cervical. Endocrine evaluation indicated normal thyroid function with positive anti-thyreoglobulin antibodies (283.3UI/ml, normal <50UI/ml), high levels of morning plasma cortisol (249ng/ml, normal 70-225ng/ml) with loss of circadian rhythm, ACTH level within the upper reference range (41.1pg/ml, normal <46pg/ml) and a very high testosterone level (6.1ng/ml, normal 0.2-0.75ng/ml). Biochemical parameters indicated hypertriglyceridemia, low potassium and neutrophilia. Non-suppression at DXM 1 mg overnight test and 50% suppression at high dose of DXM leaded to a diagnosis of CD. Imagery did not identify any tumor. No ovarian, neither adrenal tumor was found in correlation to high testosterone levels so CD was considered the only cause.
Therefore the patient was treated with steroid synthesis inhibitor-Ketoconazole (800 mg/day), with clinical improvement: decrease of hirsutism and normalization of testosterone level to 0.2ng/ml within months. Follow-up and a definitive therapy is needed.

Conclusion

Severe hyperandrogenism as seen here in CD is rare, most probably is correlated with adrenal androgen production and thyroid autoimmunity is incidental; therapeutically control of CD represents a clue of the etiological diagnosis of hyperandrogenemia.