Thyrotoxicosis presenting as orthostatic hypotension

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Summary

The patient described, whose symptoms were masked initially by the lack of steroids, illustrates an unusual case of orthostatic hypotension due to secondary adrenal insufficiency, caused by thyrotoxicosis.

Introduction

Orthostatic hypotension is a relatively common symptom caused by a variety of aetiologies. The most common of these are: neuropathies due to systemic diseases such as diabetes, amyloidosis and porphyria; vaso-vagal attacks; hypovolaemia; Shy-Drager syndrome (idiopathic orthostatic hypotension); adverse effects of anti-adrenergic drugs, L-dopa and dopaminergic drugs; phaeochromocytoma with prevalent secretion of epinephrine; and Addison's disease (Weissler and Warren, 1978).

A patient is reported who presented with orthostatic hypotension and who was subsequently found to suffer from hypoadrenocorticism secondary to thyrotoxicosis.

Case report

A 42-year-old woman, who had previously been in good health, was admitted to hospital with severe weakness, palpitations and syncope while standing or walking. Orthostatic hypotension was diagnosed before her admission and was based on marked decrease of her systolic and diastolic BP (to 40/0 mmHg), palpitations and paleness. Her disease had started 3 weeks before admission with headache, fever and orthostatic hypotension, the former 2 symptoms subsiding spontaneously within a few days. Examination showed her to be moderately obese, pale, with a tachycardia of 120/min. Her BP was 110/80 mmHg while supine and 40/0 mmHg while standing. She had mild hyperpigmentation of the eyelids and palm creases, mild exophthalmos (OD = 22 mm, OS = 24 mm) and negative von Graefe's sign. The thyroid was not enlarged and no tremor was noted.

Investigations revealed normal blood picture, ESR, serum electrolytes, urea, SMA 12 metabolic screen and 24-hr urinary catecholamine excretion. ECG confirmed sinus tachycardia of 120/min. Examinations of her neurological status and eye fundi were normal. Skull films and tomography of the sella turcica revealed nothing.

Endocrine studies revealed low plasma ACTH (20 ng/l) in the presence of low plasma cortisol (110 μmol/l) in the morning, 66.2 μmol/l in the evening) and low 24-hr urinary excretion of free cortisol (59 μg). The 24-hr urinary excretion of the 17-KS (ketosteroids) was normal (6.1 mg) and that of the 17-OHCS (hydroxycorticosteroids) was slightly elevated (17.7 mg). The plasma cortisol responses to stimulation by corticotrophin, vasopressin and insulin hypoglycaemia were normal. Plasma aldosterone levels (18.6 μg/l) while recumbent and 27.9 μg/l after 4 hr walking) as well as 24-hr urinary aldosterone excretion (22.8 μg) were slightly elevated in the presence of normal PRA (plasma renin activity) (1.5 ng/ml/hr, basal and 6.6 ng/ml/hr, stimulated). The serum thyroxine (236 nmol/l and T3 5.4 μg/l) concentrations were elevated in the presence of a normal serum TSH (4.9 μU/ml) which did not rise in response to TRH stimulation.

Because of the low levels of plasma cortisol and the clinical picture that was compatible with hypoadrenocorticism treatment with prednisone 20 mg daily was started. During the following 2 days the patient’s clinical picture changed: weakness, apathy and orthostatic hypotension disappeared and typical symptoms of hyperthyroidism, such as excitation, tremor, increased appetite, excessive sweating, heat intolerance and ocular signs (bright-eyed appearance, positive von Graefe’s sign) were observed. At this time 600 mg of propylthiouracil and 160 mg/day of DL-propranolol were administered, combined with gradual tapering of prednisone dosage until the complete cessation of therapy with steroids within a one-week period.
The patient showed significant clinical improvement with the above-mentioned regime and could be discharged. One month later she was re-admitted for re-evaluation. The endocrine studies performed at this time showed normalization of her serum T₄ (102 nmol/l), T₃, 2.2 μg/l, as well as of her plasma cortisol concentrations (229.1 μmol/l morning and 132.5 μmol/l, evening) and 24-hr urinary steroid excretion.

Discussion

Despite the absence of presenting clinical symptoms there can be little doubt that this patient suffered from thyrotoxicosis (Labhart, 1974). Arterial hypotension in general and orthostatic hypotension in particular are not presenting signs of thyrotoxicosis (Ingbar and Woebber, 1974) but are frequent symptoms of hypoadrenocorticism (Labhart, 1974).

The absence of thyrotoxic symptoms in this patient during the initial phase of her illness and their appearance after the initiation of steroid therapy are explained by the loss of permissive action of cortisol on catecholamine-mediated effects of thyroid hormones, thereby masking the thyrotoxic symptoms (Martino and Braverman, 1965; Landsberg, 1977).

The same mechanism may be invoked in the explanation of the pathogenesis of orthostatic hypotension in the present patient). Her basal BP was maintained normal by the intact functioning of the renin-aldosterone system, which is a distinctive feature of secondary adreno-cortical insufficiency (Labhart, 1974). The absence of the cortisol-permissive action for her α-adrenergic receptor-acting agonists brought about attacks of orthostatic hypotension when she was subjected to higher steroid demands.

The low basal plasma cortisol levels, as well as basal urinary excretion of free cortisol in the presence of low normal daily urinary excretion of 17-ketosteroids and slightly increased daily urinary excretion of 17-ketogenic steroids are compatible with the diagnosis of thyrotoxicosis (Hellman, Bradlow and Zumoff, 1970). The dynamic tests performed showed that the hypothalamic-pituitary-adrenal axis was normal. The diagnosis was confirmed by high T₃ and T₄ levels and by the flat thyrotrophin (TSH) curve after TRH stimulation.

Hyperthyroidism accelerates the catabolism of cortisol (Beale, Croft and Powell, 1973), but its plasma levels usually remain within normal limits (Linquette et al., 1975), so that in most cases thyrotoxic patients remain eucorticoid. However, thyrotoxicosis does cause limitation of ACTH reserve in some cases (Ambrosi et al., 1970). This could not be proved in this patient by the insulin tolerance and vasopressin tests although it is conceivable that stress of another kind (febrile illness in this case) could be sufficient to turn the unstable adrenocortical condition into clinically apparent insufficiency.

References


