New onset Graves’ disease as a cause of an adrenal crisis in an unrecognized empty sella syndrome

H. Marmouch.
F. Boubaker, S. Arfa, T. Slim, H. Sayadi, M. Jmai, I. khochtali
Endocrinology Unit - Internal Medicine-Endocrinology Department
Fattouma Bourguiba University Hospital-Medicine Faculty- MONASTIR UNIVERSITY

MONASTIR- TUNISIA

INTRODUCTION

A 66 year old woman was admitted as an emergency with vomiting, hypotension and serum cortisol of 0.92 microg/dl indicative of adrenal failure. She was found to be hyperthyroid (free T4= 72 pmol/l).

CASE REPORT

She had hypotension (blood pressure 80/40 mmHg). She was fit and well till the age of 65.

Endocrine assessment revealed deficiency in ACTH-cortisol, growth hormone, and gonadotropin, as well as low-normal free T4.

On the day of his emergency admission he looked ill and dehydrated, though was fully conscious and cooperative.

Heart rate was 110 beats/min (sinus rhythm), blood pressure 85/60 mm Hg.

There were no obvious features of infection, but there was marked tremor and thyroid bruit. She received treatment with intravenous fluids and hydrocortisone.

Administration of large dose of methimazole (40 mg/day) resulted in gradual decrease in free T4. Pituitary MRI showed empty sella [Fig 1].

The patient was found to have increased titre of antithyroidperoxidase (anti-TPO) and anti-TSH receptor (anti-TSHR) antibodies [2100 IU/l (ref. range <40) and 3.7 IU/l (ref. range <1.0), respectively].

She was referred for radioactive iodine treatment.

Iodine uptake scan performed prior to radioiodine administration confirmed uniformly increased iodine uptake consistent with Graves’ disease (GD) [Fig 2].

DISCUSSION

• GD with concomitant hypopituitarism is rare but has been described previously, there are few reports of GD occurring with ESS [1].

• We encountered a patient with unrecognized adrenocortical disease du to empty sella, in whom development of Graves’ hyperthyroidism caused an adrenal crisis.

• Empty sella syndrome (ESS) in this case can be related to autoimmune hypophysitis.

• Concomitant pituitary deficiency and GD is seldom reported. TSH levels are typically low with pituitary deficiency; however, serum TSH measurements alone are insufficient for accurate diagnosis and free hormone levels should also be considered.

CONCLUSION

• Our case illustrates coexistence of hypopituitarism and clinically significant autoimmune thyroid disease. The presence of hypopituitarism does not preclude the development of autoimmune thyrotoxicosis. Our diagnosis was GD co-existing with empty sella syndrome. Hyperthyroidism unleashed acute adrenocorticotrophic failure.

• We recommande to monitor symptoms of adrenal crisis in patients with hyperthyroidism and hypopituitarism.

References