

A case report of a GH and TSH secreting pituitary adenoma

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Abstract- We present the case of a 32 years old females with both GH and TSH secreting pituitary adenoma. The case had particular difficulty in management since few years after the first surgery the tumor had an important suprasellar extension and medical treatment had to be preferred to surgery. Ocreotide treatment was choiced having a very good follow up

Index Terms- pituitary adenomas, GH, TSH, ocreotide

I. INTRODUCTION

Central hyperthyroidism due to a thyrotropin (TSH)-secreting pituitary adenoma (TSH-oma) is a rare cause of hyperthyroidism and account for fewer than 1% of all pituitary adenomas. The mildness of the signs signs and symptoms compared to primary hyperthyroidism, postpone the tumor diagnosis (1). Even though often underdiagnosed in GH-producing pituitary adenomas, 13% have been shown to demonstrate immunopositivity to TSH (2). Plurihormonal pituitary tumors are either morphologically monomorphous (single cells producing different hormones) or plurimorphous (different cells producing different hormones). Plurihormonal pituitary tumors seem to predict a higher risk of tumor recurrence, in comparison to tumors that secrete only one hormone; therefore, careful follow-up of this population is essential (3). TSH omas are characterized by high levels of circulating free thyroid hormones (FT4 and FT3) in the presence of nonsuppressed serum TSH concentrations. We present a case of TSH/GH secreting pituitary adenoma.

II. CASE REPORT

A 32 years old female patient presented in our hospital with typical acromegalic signs and symptoms (headache, abnormal growth of the hands and feet, lower jaw protrusion, bitemporalhemianopsia, irregular menstrual cycle. She also reported being treated previously with methimazole for hyperthyroidism. Hormonal examinations are reported on Table 1.

Hormone	Value	Normal range
FT4	15pg/ml	(7-18 pg/ml)
Cortisol 16:00	129 mg/dl	(55-230 mg/dl)
IGF-1	3029 ng/ml	(107-310 ng/ml)
Anti-TPO Ab	0.1	(<70)
Ab anti TG	31.9	(<35)
Prolactin	1.2	(<25)
Ab anti TSH receptor	0.1	(<1)
ACTH 16:00	50.6	(28-140)

As shown a high IGF-1 level were detected and diagnosis of acromegaly was set. pituitary – gonadal axis was normal. Thyroid ultrasound revealed a hypoechoic thyroid goiter with bilateral increased vascularization. Thyroid scintigraphy revealed increased uptake of Tc 99 at both lobes, full up-take 13.3%.

The patient underwent transphenoidal pituitary surgery. After surgery clinical and hormonal levels were analyzed. Glucocorticoid insufficiency was set and hydrocortisone treatment was necessary. Under estro-progestinic treatment regular menstrual cycle was achieved. Hormonal examination one month after surgery are shown in table 2. IGF-1 level three months later dropped to 448ng/ml under treatment with dopamine agonist, methimazole, estroprogestative. The methimazole treatment wasn't controlling hyperthyroidism.

Hormone	Value	Normalrange
TSH	4.45 mUI/l	0.4-4 mUI/l
FT4	40 pg/ml	(7-18 pg/ml)
FT3	6.3 pg/ml	2-4.25 pg/ml
GH	3.1 ng/ml	<10 ng/ml
IGF-1	1552ng/ml	(107-310 ng/ml)
Prolactin	11.5	(<25)

Post surgery RMI revealed residual tissue that increased over time. The patientLast RMI (after 4 years) showed a sellar and suprasellar formation that infiltrate the cavernous sinus. Bilateral frontal cerebral malacia. No lesions in fossa crani posterior. Theadenoma growth has significantly decreased the visual field. Last hormonal values while the patient was taking dopamine agonist and suspended methimazole. Table 3.GH suppression test GH min 0 = 7.6; min 30 = 3.4; min 60 = 2.6.

Table 1 Hormonal results before surgery

Table 3 Hormal values

Hormone	Value	Normalrange
TSH	5.48 mUI/l	0.4-4 mUI/l
FT4	38.8 pg/ml	(7-18 pg/ml)
FT3	7.7 pg/ml	2-4.25 pg/ml
IGF-1	277 ng/ml	(107-310 ng/ml)
Prolactin	18.9	(<25)

A TRH stimulation test

was performed showing discrepancy (table 4).

Table 4 TRH stimulation test

min	TSH (mIU/l) Normalrange 0.17-4.04	FT4 (ug/dl) N. r 7-18 ug/dl
0	5.47	38.2
20	5.32	37.5
60	5.55	34.5
90	5.7	36.0

Due to the tumor extension the surgery was not indicated. According to literature 90% of the TSH-omas have a positive response to somatostatin treatment. Octreotid treatment was started, depot injection every 4 weeks. After 6months a normalization the thyroid hormones were observed. TSH 3.24mIU/l, FT4 16.2ug/dl, FT3 3,4ug/dl.

DISCUSSION

We presented a case of GH and TSH secreting pituitary adenoma. TSH-producing adenoma is a rare disorder, accounting for about 0.5% to 2% of all pituitary adenomas (4). Even though TSH secreting adenoma are usually benign tumors they often are large and invasive making them difficult to have a first line surgery approach. Medical therapy was chosen for our patient. The patient was treated according to the suggestions of European Thyroid Association guidelines for the diagnosis and treatment of thyrotropin-secreting pituitary tumors (1). This study confirms the efficacy of primary medical treatment with somatostatin analogs in terms of hormonal control in patients with TSH-secreting pituitary adenomas, and its good safety profile. It also emphasizes the importance of considering multi hormonal secretion in case pituitary adenoma.

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