

PITUITARY ADENOMA OF AGGRESSIVE BEHAVIOUR

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INTRODUCCION

Pituitary adrenocorticotrophic hormone (ACTH)-secreting tumor presents with variety of clinical features: silent corticotroph adenomas (normal cortisol secretion and ACTH-immunopositive staining) and subclinical Cushing's disease (ACTH-induced mild hypercortisolism without typical features of Cushing's disease).

CLINICAL CASE

We present a 53-year old male with history of hypertension.

First visit (2005): he had visual loss, bitemporal hemianopsia in campimetry and in MRI pituitary adenoma (2.5cm) invading cavernous sinus and suprasellar cister. Hormonal study supported panhypopituitarism.

After transsphenoidal surgery, there was a tumour rest (9mm) with left cavernous sinus invasion. Pathology: atypical pituitary adenoma, ACTH-immunopositive staining. There were normal campimetry and persistence of hormonal deficit. In 2006, he received photon radiotherapy (50 Gy) with radiologic stability.



Fig. 1. Left eyelid ptosis

After six years, left eyelid ptosis (Fig. 1) (cranial nerves III-VI palsy) appeared. In replacement therapy with hydrocortisone, levothyroxine and testosterone, presented TSH<0.05 mcgUI/ml, FT4 1.1ng/dl, FSH<0.3mIU/ml, LH<0.07mIU/ml, Prolactin 3.2ng/ml, Testosterone 3.7ng/ml, basal cortisol 10 mcg/dl, ACTH 53pg/ml, IGF-1 24ng/ml. MRI: pituitary solid lesion (1.7cm) extending to the left cavernous sinus.

Transsphenoidal surgery was performed. Ophthalmoplegia persisted. Pathology: pituitary adenoma ACTH-producer, Ki<1%, P53 75%. (Fig. 2)

After surgery: TSH<0.005mcgUI/ml, FT4 1.2ng/dl, cortisol 10mcg/dl, ACTH 64pg/ml. Hydrocortisone was suspended and an ACTH-test (250 mcg) was performed: peak cortisol 14.4mcg/dl, so corticosteroid therapy was maintained.

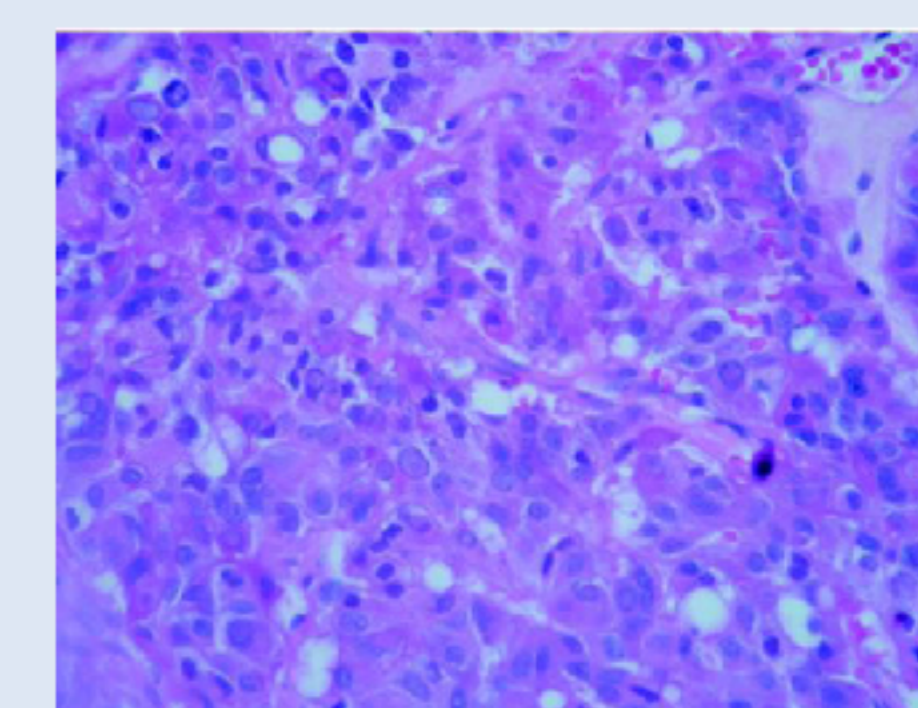


Fig. 2. Pituitary adenoma ACTH-producer

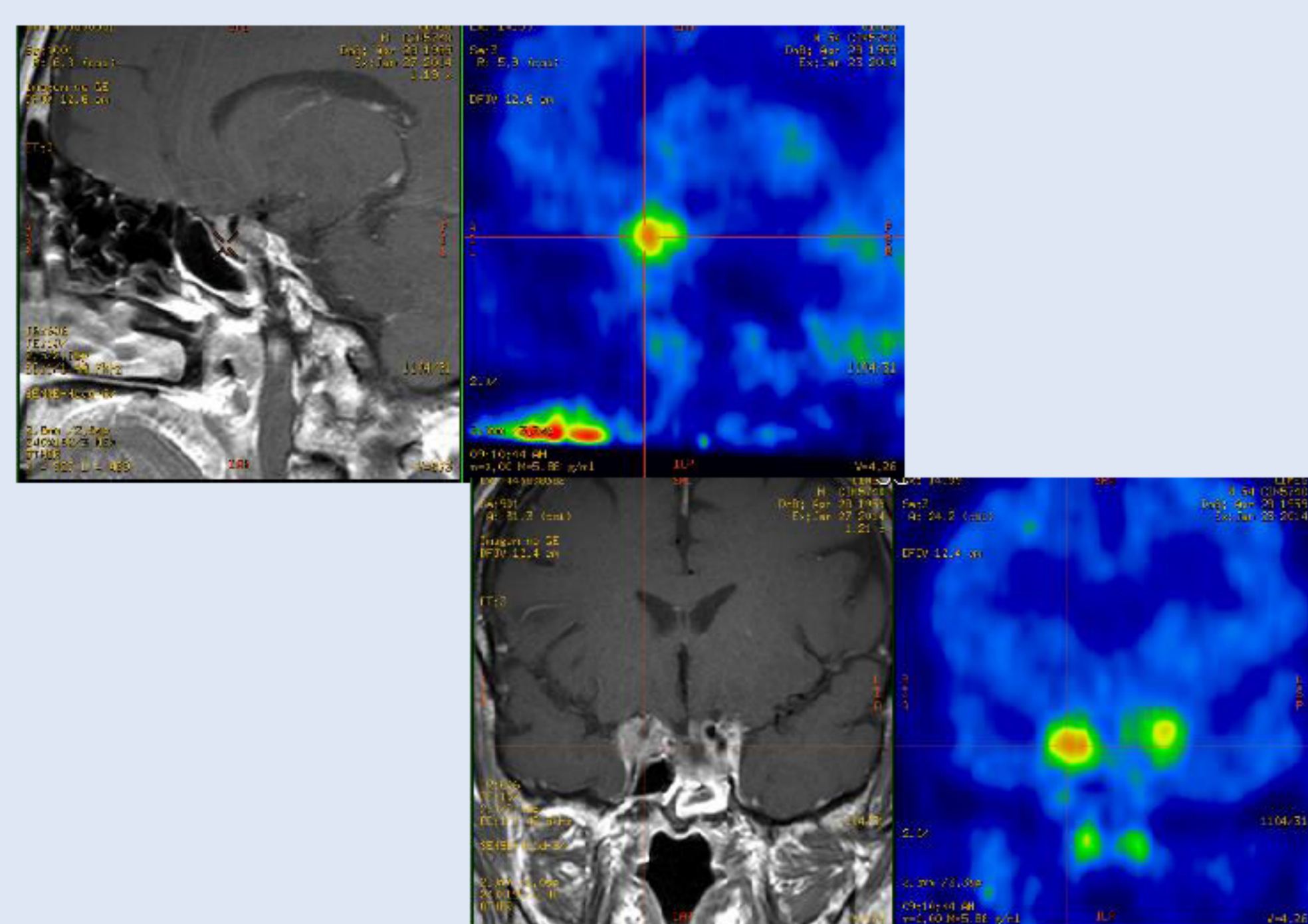


Fig. 3. PET-TC-fluodesoxiglucose-metionine and MRI pituitary

6 months after surgery: MRI with tumour rest (11mm) in the left cavernous sinus and right cavernous sinus (16 mm). PET-TC-fluodesoxiglucose-metionine: metabolically active injury in both cavernous sinuses (Fig. 3). Hormone Study: Cortisol 17mcg/dl, ACTH 97pg/ml, so we suspended hydrocortisone. Without treatment cortisol was 16mcg/dl. Nugent test: cortisol was 18mcg/dl.

Fractionated stereotactic radiotherapy (50 Gy) was administered. After 2 months: cortisol 16mcg/dl, ACTH 127pg/ml, UFC 320mcg/24h. We began cabergoline 1mg/week.

CONCLUSION

The development of Cushing syndrome in patients with silent corticotropinomas determines a factor of aggressiveness in these tumours.

Intensification with cabergoline was performed, and temozolomide was planned in case of progression.

