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Letter to the Editor

Combined modalities of surgery, radiotherapy, radiosurgery and chemotherapy for invasive pituitary carcinoma

Management multimodal par chirurgie, radiothérapie, radiochirurgie et chimiothérapie pour un carcinome pituitaire invasif

A 24-year-old male presented with bitemporal hemianopia and visual acuity decrease; MRI showed a lesion located at the sellar level, with suprasellar extension; biological tests in this contexts showed a prolactin (PRL) value of 41 μg/L; the diagnostic of a prolactinoma or a silent prolactin (PRL) tumor, was evoked. He received dopaminergic agonists (PRL passing to 22–27 and 12 μg/L, respectively) with good response and approximately 30% tumor size reduction. Eleven years after discovery, the patient developed again evidence of bitemporal hemianopia. MRI displayed pituitary tumor growth with compression of the optic apparatus. To decompress the former, due to obvious resistance to medical treatment, transsphenoidal macroscopically incomplete resection was performed (Fig. 1.1,a). Histopathological exam revealed pituitary adenoma (PA), without immunohistochemichal expression of hormone and signs of atipicity (no polymorphism, no mitosis, number of cells in proliferation marked by MIB-I < 1%). The vision partially recovered, with persisting right supero-extern quadranopia (PRL of 18 μg/L). Fractionated radiotherapy was performed on residual volume of 32.83 cm^3 (28 fractions, 50.4 Gy at the 85% isodose). Initial clinical course was unremarkable. Two weeks later the patient suddenly developed left visual acuity decrease (0.4), with bitemporal hemianopia and partial left IIIrd nerve paralysis. MRI revealed further cystic transformation on the left side, with major compression of the optic apparatus. Radiotherapy (RT) was stopped for urgent pterional craniotomy (left suprasellar extension (Fig. 1.1,b)) of a residual tumor left untouched within the cavernous sinus and histopathology showing gliosis brain tissue (cyst). Postoperatively, RT was continued with no adverse effect. The PRL value remained stable for the next seven years and MRI showed progressive tumor size decrease. Eighteen years after discovery, left visual acuity decreased from 0.4 to complete blindness. MRI revealed major increase of tumor volume, with important compression of the optic apparatus, PRL increasing from 50 to 386 μg/L (Fig. 2). Left fronto-temporal craniotomy was performed (Fig. 1.1,c). The histopathological result experienced major change, showing a PA, with strong and diffuse PRL expression in immunohistochemistry. A postoperative MRI confirmed presence of residual tumor and, concerning the patient only one right functional eye, due to the very closeness of the remnant to the right optic nerve. He was therefore addressed for Gamma Knife surgery (GKS, Fig. 1.2, a and b). The marginal dose was 18 Gy at the 50% isodose, not higher so as to preserve the only functional eye of the patient (8 Gy maximal dose for the optics). The target volume (TV) was 10.3 cm^3 and prescription isodose volume (PIV) 13.3 cm^3. Six months latter, PRL dramatically decreased (52.6 μg/L) and MRI revealed shrinkage of the tumor. Twenty-one years after discovery, MRI showed out-of-filed recurrence, outside the first GKS TV, and therefore a second GKS was performed (Fig. 1.3), with 18 Gy at 50% isodose (8.5 Gy maximal dose for optic apparatus). The TV was 1.71 cm^3 and PIV 1.8 cm^3. The patient additionally underwent microsurgical excision of left frontal lesion (Fig. 1.4). The histopathological examination revealed MIB-1 5–10%, with multiple nuclei (> 100 nuclei/10 fields) expressing P53 (PC in accordance with Trouillas et al. [1], strong and diffuse PRL expression). A second RT was performed (by tomotherapy, 60 Gy in 30 fractions). A new postoperative MRI revealed a second metastasis, within right orbital gyrus, close to the right optic nerve, the only functional eye of our patient. A third GKS was performed (18 Gy at the 50% isodose, Fig. 1.5, maximal dose received by the optic apparatus 12 Gy), with TV of 564 mm^3 and PIV 861 mm^3. Spinal MRI revealed lesions at the level of S1 and S2 (Fig. 1.6, a). Lumbar puncture failed to find malignant cells within the CSF. A proposal for radio- and chemotherapy (temozolomide) was decided. The PRL value was 70.6 at this time and dropped off to 22.2 μg/L. Clinically he remained stable and MRI revealed decrease in size of all lesions treated by GKS, with no postoperative recurrence for the operated left frontal. Spinal MRI showed a decrease in size of all nodules (Fig. 1.6, b). Up-to-date, the patient remains clinically and radiologically stable, 14 months after the initiation of temozolomide and 19 months after the appearance of first cranial metastasis.

Pituitary tumors are rather usual intracranial neoplasms, accounting for approximately 10% of the former [2]. Pituitary carcinomas are extremely rare entities, with incidence of less than 0.5% of the symptomatic pituitary tumors and only 132 cases described over a period of 48 years (1961–2009 [3]). They are defined as pituitary tumors with subarachnoid, brain or systemic metastasis [4]. Natural history of such lesions is

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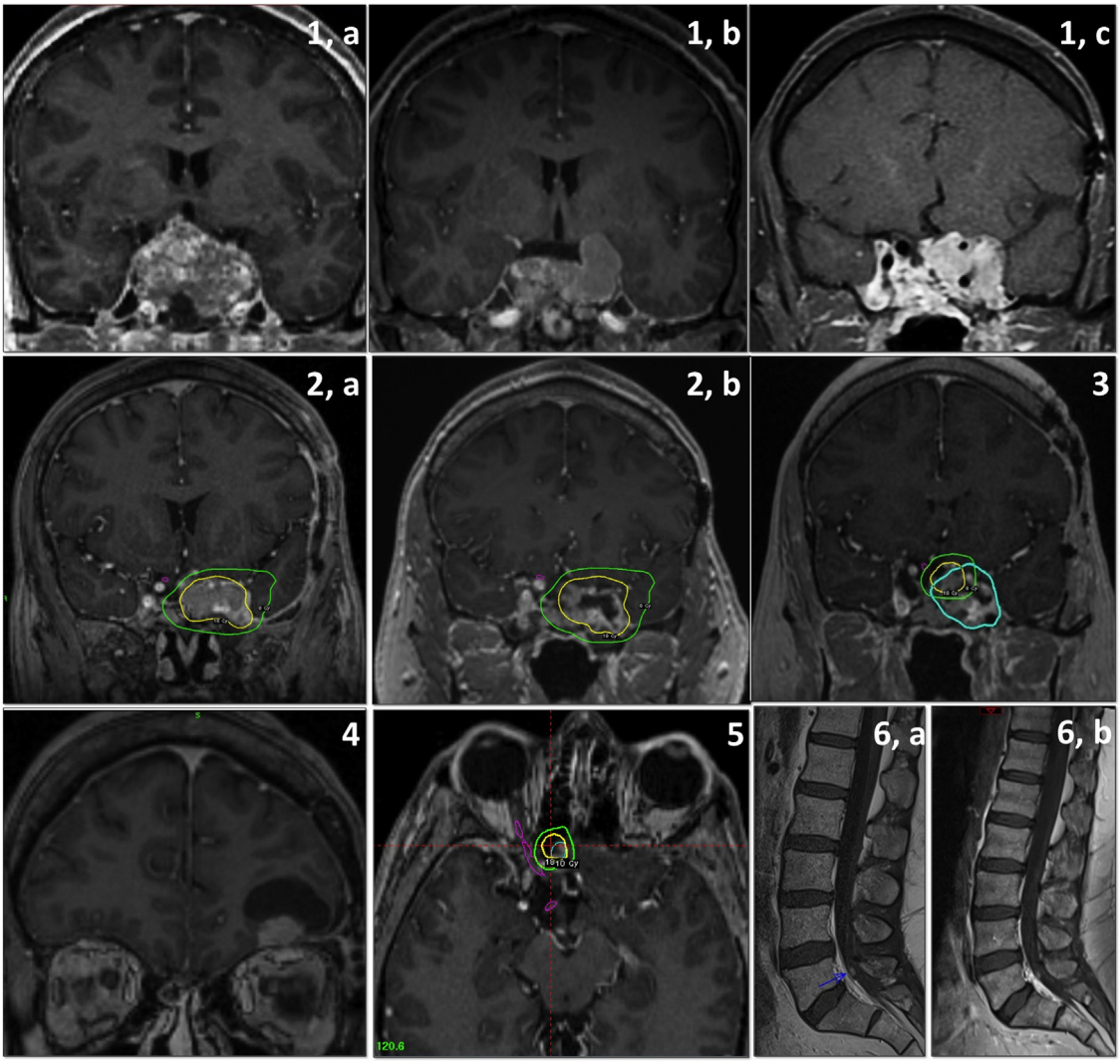

Fig. 1. 1, a: T1 injected gadolinium MRI (coronal plane), displaying the pituitary adenoma before the first surgical excision (done by transsphenoidal approach); one can see the major compression of the optic apparatus, in a symptomatic patient; 1, b: T1 injected Gadolinium MRI (coronal plane), acquired during the radiotherapy course, following the first surgery, showing a cystic transformation on the left side, with major compression of the optic apparatus and left suprasellar extension, requiring a further surgical approach (pterional, due to the anatomical extension), with residual tumor left untouched within the cavernous sinus; 1, c: T1 injected gadolinium MRI (coronal plane), 18 years after discovery, revealing major increase of the tumor volume, with important compression of the optic apparatus; a left fronto-temporal craniotomy was further performed; 2, a: T1 injected gadolinium MRI (coronal plane), showing the first GKS treatment, having as target the residual tumor located at the level of the left cavernous sinus, with the 50% isodose line in yellow, the 8 Gy green line with regard to the safety dose that could be received by the optic apparatus, and the right optic nerve coloured in magenta (the only functional eye of the patient); 2, b: T1 injected gadolinium MRI (coronal plane), revealing shrinkage, 6 months after GKS; 3: T1 injected gadolinium MRI (coronal plane), 21 years after discovery and 26 months after the first GKS; the MRI shows an out-of-field recurrence, outside the first GKS target volume, in the presence of an increased PRL; a second GKS was decided and performed; the blue line represents the first GKS treatment, co-registered with the new MRI, acquired during the second GKS; the yellow line represents the 50% isodose line, the green line represents the 8 Gy absolute dose, with regard to the safety dose that can be delivered to the optic apparatus; the right optic nerve (the only functional eye of the patient) is coloured in magenta; 4: T1 injected gadolinium MRI (coronal plane), showing a right frontal metastasis, located at the level of the right orbital gyrus, which has been further operated with complete excision; 5: T1 injected gadolinium MRI (axial plane), displaying a new right fronto-basal lesion, close to the right optic nerve, the only functional eye of the patient; a third GKS was performed; the yellow line represents the 50% isodose line, the green line represents the 8 Gy absolute dose, with regard to the safety dose that can be delivered to the optic apparatus; the right optic nerve (the only functional eye of the patient) is coloured in magenta; 6, a and b: spinal sagittal MRI revealing lesions at the level of S1 and S2, before (a) and after (b) radio-chemotherapy.

marked either by an initial benign course, with gradual tumor genetic alterations, or by an early malignant behavior, with important initial invasiveness, multiple recurrences and early development of metastasis, the former being less common [2]. The criteria needing to be fulfilled for PC are: identification of pituitary tumor by histology, exclusion of an alternative primary tumor, discontinuous spread of subarachnoid metastatic deposits, systemic metastatic deposits and the structural features or marker expressions of the metastases are corresponding or similar to those of a pituitary tumor [2]. Most cases of PC are resulting from the malignant transformation of tumors restrained to pituitary initially considered to be benign macroadenomas

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