CLINICAL IMAGE

Remarkable remission of an invasive giant prolactinoma under high-dose bromocriptine monotherapy

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A therapy of choice in prolactinomas is pharmacological treatment with dopamine antagonists, while cabergoline is a preferred agent.[1] However, in the case of large and sight-threatening lesions, the recommendations are not precise and often neurosurgical intervention or radiotherapy (or both) is required.[2-5] Giant prolactinoma (GP) is a pituitary adenoma of the largest diameter of 4 cm or higher, massive extrasellar extension, a prolactin concentration exceeding 20 000 μlU/ml, and no growth hormone or adrenocorticotropic hormone secretion. GPs constitute from 2% to 3% of prolactinomas and usually affect middle-aged men.[1] In GPs, therapy exclusively with dopamine antagonists had been very rarely performed and it has led to normalization of serum prolactin just in a few patients.[1,2]

A 39-year-old obese male patient with hypertension and mild hypercholesterolemia had been suffering from headaches and progressive visual loss in the right eye for about 1 year. He also reported decreased libido and erectile dysfunction, mild gynecomastia, and slight reduction in facial hair. Magnetic resonance imaging (MRI) revealed a pituitary tumor (4.2 × 2.7 × 3.1 cm) invading asymmetrically both cavernous sinuses, with compression on the right optic nerve but intact optic chiasm (FIGURE 1A). The baseline serum prolactin level was 163 922 μlU/ml (reference range, 85–390 μlU/ml), with 75% being an active form following precipitation with polyethylene glycol. He presented hypogonadotropic hypogonadism: free testosterone, 3.1 pg/ml (reference range, 9–42 pg/ml); luteinizing hormone, 0.6 mlU/ml (reference range, 1.7–8.6 mlU/ml); and follicle-stimulating hormone, 1.8 mlU/ml (reference range, 1.5–12.5 mlU/ml), but normal pituitary function in other axes. Bromocriptine therapy was initiated at a dose of 2.5 mg/d, and was gradually increased to 30 mg/d over 3 months. During the therapy, the patient reported mild headaches, weakness, and benign orthostatic hypotension. After 3 months of treatment, the prolactin level dropped dramatically to 150 μlU/ml, and 2 months later, MRI revealed a reduction in the tumor size to 3 × 3 × 2.4 cm. Prolactin levels remained low, while the dose of bromocriptine was gradually tapered to 2.5 mg/d over 4 years. The prolactin value of 82.6 μlU/ml was achieved at that time, and almost complete radiological remission as well as full clinical and biochemical resolution of symptoms were observed (FIGURE 1B).

A literature review revealed that in patients with a prolactin level between 25 000 and 250 000 μlU/ml (comparable to that in our patient), pharmacotherapy even with addition of neurosurgery or radiation therapy (or both) resulted in prolactin normalization only in 37% of the patients.[5] Mascarell and Sarne[5] described also 2 patients with GP after decompressive surgery on a high dose of bromocriptine (30 mg/d and 40 mg/d, respectively), but the first patient was lost to follow-up and the other was switched to cabergoline due to poor response. Cases of successful bromocriptine monotherapy have already been reported; however, compared with our patient, those patients presented lower baseline serum prolactin levels and were treated with lower dose of bromocriptine (22.5 mg/d).[4]

Our report indicates that even in the case of invading and symptomatic GPs, high-dose bromocriptine monotherapy might be a sufficient, safe, and well-tolerated treatment. It seems advisable that solely pharmacological treatment alone should be attempted even in GPs, unless there is high risk of sight loss, leakage of cerebrospinal fluid, or tumor resistance to the therapy.

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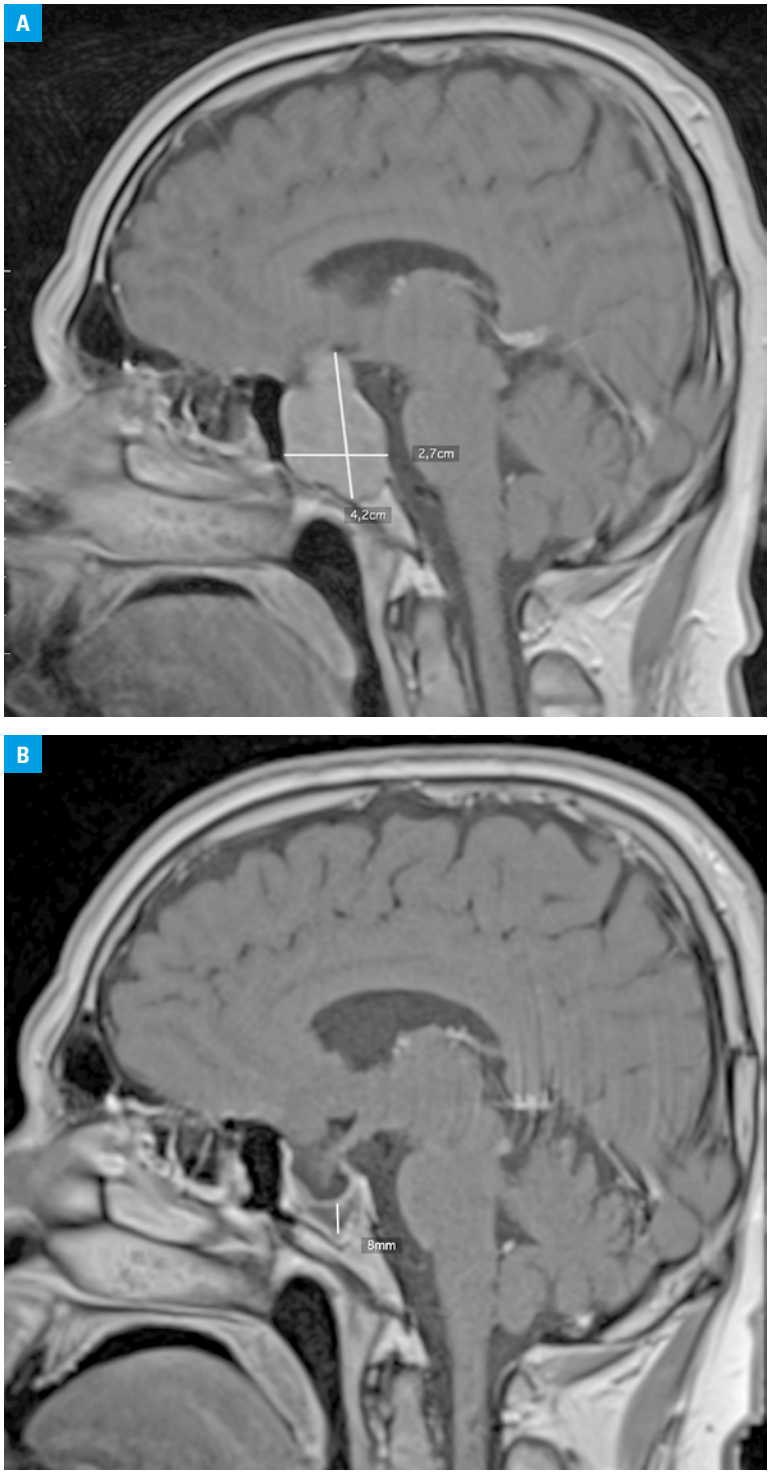

FIGURE 1 Magnetic resonance imaging of a giant prolactinoma before therapy (**A**) and following bromocriptine monotherapy (**B**). In remission, only residual scar tissue and minor liquid areas in the pituitary fossa of a total size of 8 mm, free bilateral cavernous sinuses, and intact optic chiasm were observed.

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