UNUSUAL MRI FINDING IN A MALE WITH LYMPHOCYTIC HYPOPHYSITIS MIMICKING A PITUITARY TUMOUR

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Dear Editor,

Autoimmune hypophysitis (AH) is a chronic inflammatory disease, described for the first time by Goudie and Pinkerton (1), which affects the pituitary gland, and can induce neurological, ophthalmological and endocrine damage. We have recently observed an unusual MRI finding in a 38-yr-old man who complained of sudden diplopia with ophthalmoplegia, blurred vision, and episodic headaches in the complete absence of symptoms or signs of pituitary dysfunction or diabetes insipidus. Neuro-ophthalmological evaluation revealed left oculomotor nerve (N III) palsy. MRI scan showed an inhomogeneous pituitary mass mimicking a tumour extended into the left cavernous sinus, with a dural tail sign and a hypointense “omega”- shaped line demarcating the lower portion of the mass. The pituitary stalk was thickened, but not deviated. The typical bright appearance of the posterior pituitary was absent (Fig.1). Serum PRL levels were slightly elevated (45 µg/L, normal range <21 µg/L), but serum levels of LH, FSH, ACTH, GH, TSH and related signals were comprised within the normal reference ranges. Diplopia, left ophthalmoplegia and headaches resolved spontaneously within a few days. For this reason, treatment was limited to normalization of serum PRL levels by cabergoline administration (0.25 mg/week p. os). Four months later, the patient begun to suffer from weakness, nausea, headache, erectile dysfunction and severe left-sided retro-orbital pain associated with body weight gain and blood hypotension (90/50 mmHg). Routine blood test revealed elevated inflammation parameters (ESR: 24 mm/h, C-reactive protein: 7.5 mg/L, fibrinogen 447 mg/dl) and an increased titer of APA (1:16). A new pituitary MRI showed a mild increase of the left-side intracavernosal part of the mass, whereas other findings were unchanged. After the withdrawal of cabergoline treatment, endocrine evaluation demonstrated, for the first time, panhypopituitarism, and replacement therapy with L-thyroxine (75 µg/day p.o.), cortisone acetate (25 mg/day p.o.) and testosterone enantate (250 mg/ 28 days, i.m.) was started. Since pituitary mass grew, transsphenoidal
pituitary decompression was performed and histology revealed typical features of AH. Thereafter, the patient was treated with high dose methylprednisolone pulse therapy. Since corticosteroid therapy was followed by the re-growth of the mass, treatment was subsequently switched to azathioprine. Last MRI showed decrease in size of the pituitary mass, and thickening of the hypointense “omega”-shaped line. However, anterior pituitary function did not recover.

Typical clinical features of AH include female preponderance, presentation during pregnancy or in the post-partum period, and association with other endocrine autoimmune disorders (2,3). Patients complain of headaches, visual field impairment, and/or mechanical ophthalmoplegia. Destructive hypopituitarism is described along with central diabetes insipidus in a significant proportion of patients (2-4). Elevated inflammation indexes may be found in the blood and APA are detected in the serum of only 70% of patients with biopsy-proven AH (3). Usually, MRI demonstrates a symmetrical “tent-shaped”, homogeneous mass in the pituitary area, and thickening of the pituitary stalk. After Gd-DTPA administration, homogeneous enhancement of the pituitary mass and of the tissue along the dura mater (“dural tail”) may be found, although the latter is considered nonspecific (5).

In this case, enhancement (both before and after Gd-DTPA) was inhomogeneous and the typical “tent-shape” appearance was absent. In addition, the extrasellar expansion was asymmetrical with infiltration of the left cavernous sinus. Normal hyperintensity of the neurohypophysis on T1-weighted pre-contrastographic images was also absent, but the patient was not diagnosed with diabetes insipidus. The first MRI also showed a hypointense area with a morphology similar to the Greek ω (omega) on T2-weighted images in the lower portion of the anterior pituitary, close to the sellar floor. At follow-up, this hypointensity had expanded to the entire perimeter of the gland. In our opinion, this omega feature could have been produced by the progressive fibrosis of the pituitary gland, which represents the outcome of
AH. We propose that this unusual finding, never described in literature until now, must be sought in other proven cases of AH in order to provide a possible validation as a new MRI marker of AH.
REFERENCES


**FIGURE LEGEND**

**Figure 1.** Coronal FSE T2-weighted MRI of the head shows: (a) cancellation of normal high signal of the cavernous sinus (black arrow) and of the apex of Meckel’s cavum on the same side (arrow-head). The adenohypophysis is enlarged and hyperintense (asterisk). A focus of a few millimetres of diameter is visible inside the gland (arrow); (b) an omega-shaped line, markedly hypointense, traces the periphery of the gland (arrow). Sagittal FSE T1-weighted imaging after gadolinium-DTPA administration MRI of the head shows: (c) strong enhancement of the adenohypophysis (arrow) and of the dura, slightly thickened, at clivus and at sphenoidal plane (arrow-heads); (d) thickening of the infundibular stalk (arrow).