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Pituitary apoplexy due to macroadenoma bleeding

Apoplejía hipofisaria por sangrado de macroadenoma

A 54-year-old male attended the emergency room for sudden bifrontal headache, nausea, and vomiting. Nine years previously, he had experienced a similar condition associated with diplopia and had been diagnosed a non-secretory pituitary macroadenoma (Fig. 1 A-B) with secondary panhypopituitarism (TSH 0.026 mcU/mL, T4 0.54 ng/dL, basal prolactin 1.88 ng/mL, FSH 1.01 mlU/mL, LH < 0.5 mlU/mL, ACTH < 10 pg/mL, cortisol < 1 μ g/dL, GH < 0.3 ng/mL). The patient had refused any therapeutic intervention other than hormone replacement therapy.

Physical examination revealed partial paresis of the right third cranial nerve with no campimetric or visual acuity deficits. Both CT and MRI of the head showed a 2.6 x 2.3 cm pituitary adenoma which had increased in size since the previous examination, extending to the chiasm without compressing it and had recent intratumoral bleeding (Fig. 2 A-B). Based on a diagnosis of pituitary apoplexy (PA) due to macroadenoma bleeding, transsphenoidal hypophysectomy with evacuation of intratumoral hematoma was performed with no complications. The postoperative course was uneventful, with complete neurological recovery.

PA is an uncommon complication in the course of pituitary tumors. It is caused by a sudden expansion of the pituitary gland secondary to an ischemic or hemorrhagic infarction which almost invariably occurs in the presence of a pituitary adenoma. The true incidence of PA is difficult to establish because pituitary tumor bleeding is often asymptomatic. The reported data suggest that up to 25.7% of pituitary tumors exhibit some degree of surgically documented bleeding¹. However, PA occurs as a syndrome in 0.6%-21% of the cases²⁻⁵. In addition, a vast majority of them occur in non-functioning adenomas (77% in the Semple et al series⁶). The rapid growth of sellar contents compresses both adjacent structures and pituitary vascularization and causes sudden headache, vomiting, disorders, oculomotor nerve palsy, meningism, impaired consciousness, and hypopituitarism. PA spontaneously occurs in 60%-80% of previously asymptomatic patients⁷. However, it has been reported as being associated with a number of triggering factors such as head trauma, arterial hypotension or hypertension, a history of irradiation, heart

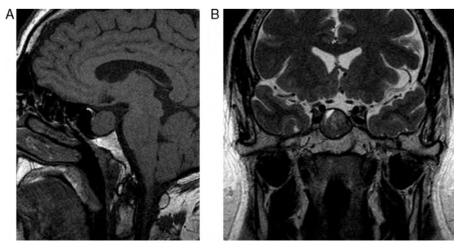


Figure 1

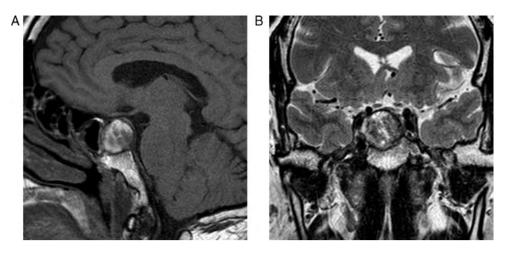


Figure 2

surgery, anticoagulation, bromocriptine administration, and pituitary dynamic tests⁷. Diagnosis is mainly based on clinical signs, and pituitary MRI is the imaging test of choice⁴.

The optimal treatment for this condition is controversial. There is general agreement that initial management should consist of the monitoring of water and electrolyte balance, strict clinical control, and the correction of hormone deficiencies, as well as the administration of glucocorticoids at supraphysiological doses⁷⁻⁹. Once clinical status is stabilized, surgical decompression of sellar contents through a transcranial or transsphenoidal approach could be indicated, but the need or urgency of this procedure has not been fully established. If severe neuroophthalmological damage (chiasm compression, impaired consciousness, or focal neurological signs) or progressive clinical impairment occur despite conservative treatment, surgical decompression is indicated^{8,10}. Conservative treatment should be adequate for patients with no headache and ophthalmoplegia. Controversy also exists as to the optimal timing of surgery. The most representative studies reported in the literature suggest that the best visual prognosis and recovery of pituitary function are achieved with early (within 7-8 days of the start of symptoms), but not necessarily urgent, surgical decompression of sellar contents^{3,4,9}. An exception should be made for patients with sudden, severe visual impairment, for whom urgent surgical decompression is recommended³. Most patients who experience PA have transient or permanent pituitary insufficiency, and up to 80% of patients require hormone replacement therapy at least once⁷. Prognosis will depend on the degree of initial involvement and delay in treatment. In the past PA had a high mortality rate, but it is currently associated with a significant improvement in survival rates as a consequence of a greater understanding of the condition at both the clinical and the therapeutic level and also due to the development of new imaging techniques.

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