

TRANSIENT CENTRAL DIABETES INSIPIDUS FOLLOWED BY PITUITARY APOPLEXY TREATED IN A CONSERVATIVE WAY

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Pituitary apoplexy is an acute clinical syndrome resulting from hemorrhage and/or infarction of the pituitary gland or adenoma¹⁻³. The frequency of sub clinical apoplexy may be as high as 25%⁴. The clinical syndrome has typical features with abrupt onset of severe headache, visual impairment, ophthalmoplegia, vomiting and/or altered mental status^{2,3}. These findings are often accompanied by various degrees of hypopituitarism, even without surgical management of the apoplexy. Even though, posterior pituitary function is nearly always preserved, a few cases of diabetes insipidus have been described^{5,6}. Diabetes insipidus occurs transiently in about 4% of patients with apoplexy and persistently in only 2 % of these patients⁵⁻⁷.

We report a patient with a previously non diagnosed nonfunctioning pituitary tumor that developed central diabetes insipidus following pituitary apoplexy.

CASE

A 49-year-old man, previously diagnosed with systemic arterial hypertension, presented to emergency room with severe headache. On physical examination blood pressure was 160x100 mm Hg. After 10 hours, despite of blood pressure normalization, the headache persisted and was followed by ophthalmoplegia, meningeal irritation signs and consciousness impairment. A lumbar puncture was performed and meningitis was excluded. Brain computed tomography was done and only maxillary sinusitis was detected. Three days after, the patient developed hypotension. Magnetic resonance imaging demonstrated a macroadenoma of 20x15x15 mm with supra-sellar extension and intratumoral hemorrhage (Figure). At that moment, pituitary function tests were normal (thyrotropin: 1.46 mcUI/mL; RV: 0.3 a 5.0 mcUI/mL; free T4: 1.3 ng/dL; RV: 0.8-1.9 ng/dL; prolactin: 3.4 ng/dL; RV: 2,0 a 15,2 ng/mL) except for a low cortisol level (5.7

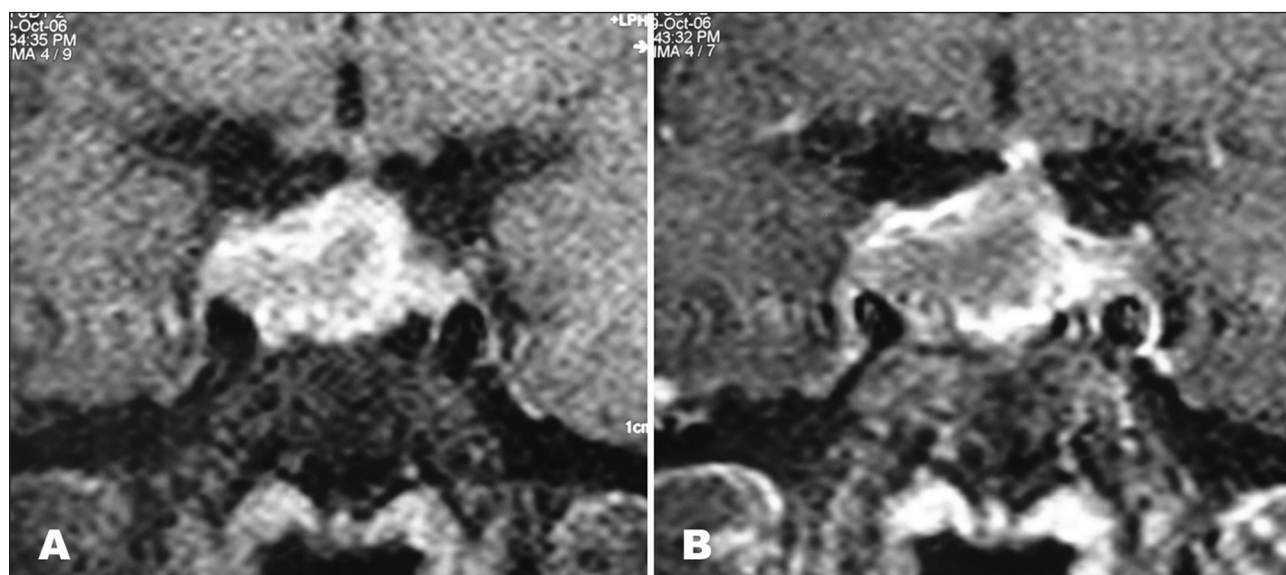


Figure. MRI (T1 weighed, coronal) showing a macroadenoma with supra-sellar extension and intratumoral hemorrhage, before gadolinium injection (A) and after gadolinium injection (B).

APOPLEXIA HIPOFISÁRIA COM TRATAMENTO CONSERVADOR SEGUIDO POR DIABETES INSIPIDUS CENTRAL TRANSITÓRIO

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mcg/dL). As there were no signs of progressive neuro-ophthalmologic deterioration or impairment in the level of consciousness, conservative management with high dose of glucocorticoids (dexametasone 16 mg/day) was chosen. Accordingly to the clinical improvement, already at the first week, the dose of dexametasone was tapered. Two weeks after apoplexy, the patient was receiving oral prednisone 7.5 mg/day and presented with polydipsia and polyuria. Urine volume was 10 L per 24h, urinary density was 1.005 and the serum sodium was 146 mEq/L (RV 136 a 146 mEq/L). Therefore, the diagnosis of diabetes insipidus was established and desmopressim (DDAVP) was started intranasally 30 mcg/day. In addition, evaluation of pituitary function showed hypogonadotropic hypogonadism (total testosterone level: 2393 pg/mL; RV: 2800-8000 pg/mL) and testosterone replacement was also started. After two months, DDAVP dose was gradually decreased and interrupted without changes in urine volume. The glucocorticoid replacement was also stopped and, after five months of apoplexy, the adrenocortical function was evaluated by a short 1 mcg corticropim (ACTH) test, demonstrating normal cortisol levels (21.5 mcg/dL).

DISCUSSION

We report a hypertensive patient with a previously undiagnosed nonfunctioning pituitary adenoma, who developed pituitary apoplexy, which was treated in a conservative way. After two weeks of the apoplectic episode, the patient developed a transient diabetes insipidus, an uncommon complication of pituitary apoplexy^{2,5-7}.

The classical clinical presentation of pituitary apoplexy with sudden onset of severe headache, visual impairment, diplopia, vomiting and altered mental status is rare^{2,3}. Subclinical tumor hemorrhage may be detected on routine imaging exams, and patients may experience only mild or no symptoms suggestive of an apoplectic event²⁻⁴. The patient reported here presented classical clinical signs of pituitary apoplexy.

Almost 50% of pituitary apoplexy occurs in patients who were not previously known to harbor a pituitary adenoma⁷. Due to this fact and also to the neurological signs and symptoms, pituitary apoplexy can mimic other intracranial disorders, making the diagnosis sometimes difficult to establish. The most important differential diagnosis to consider are aneurismal subarachnoid hemorrhage and bacterial meningitis. Midbrain infarction and cavernous sinus thrombosis, although much less common, also must be excluded^{2,8}. Our patient had no signs of intracranial hemorrhage at the computed tomography, and a normal lumbar puncture excluded the diagnosis of meningitis.

Systemic arterial hypertension could have been a predisposing factor in this case. Apoplexy is spontaneous in approximately 50% of cases, but it has been associated

with numerous precipitating factors, such as head trauma, systemic arterial hypertension, diabetes mellitus, radiotherapy, dynamic tests of the pituitary function, surgery (mainly cardiac), coagulation disorders and several medications including aspirins, estrogens, heparin, bromocriptine and cabergoline⁹⁻¹⁴.

Pituitary surgery was not performed because there were no signs of progressive deterioration in neuro-ophthalmologic system and/or in the level of consciousness in the mentioned patient. Also, the resolution of his symptoms occurred within two days after the apoplectic episode. However, early decompression of the pituitary has been proposed as the treatment of choice of pituitary apoplexy by some authors. The major arguments are the possibility of better visual and endocrine outcomes¹⁵. Several studies have favored conservative management of pituitary apoplexy when no progressive neuro-ophthalmologic signs or consciousness impairment are present, with undetectable deleterious effect on visual or endocrine outcome¹⁵⁻¹⁷.

Diabetes insipidus after pituitary apoplexy treated clinically has been rarely reported in the literature^{2,5-7}. Gebel¹⁸ described the first case of pituitary apoplexy associated with diabetes insipidus in 1962. He reported a 33-year-old man who developed polyuria and polydipsia 30 days after pituitary apoplexy clinically treated and the evaluation of pituitary function showed hypogonadotropic hypogonadism and adrenal insufficiency. This case is similar to that one described by Gebel¹⁸. Yue et al.¹⁹ described two cases of diabetes insipidus at the presentation of pituitary apoplexy. Our patient developed central diabetes insipidus two weeks after the pituitary apoplexy and it was transitory, lasting for two months.

Various degrees of hypopituitarism often occur after pituitary apoplexy. A prompt diagnosis of pituitary apoplexy is important, as a majority of these patients have deficiency of one or more pituitary hormones at presentation, and timely replacement of deficient hormones, mainly glucocorticoids, can reduce morbidity and mortality^{2,7}. Pituitary apoplexy is more often limited to the anterior lobe of the pituitary, however in some instances, it might involve the posterior lobe. The anterior lobe is supplied via the superior hypophyseal arteries, which are derivated from internal carotid artery and traverse the diaphragma sellae along the pituitary stalk. The posterior lobe is supplied via the inferior hypophyseal arteries, which also arise from internal carotid artery, nevertheless go down to the pituitary gland rather than within the stalk²⁰. The cause of hypopituitarism after apoplectic event may be due to compression or destruction of the pituitary gland, interruption of the blood supply to the gland or to the pituitary stalk. Verrees et al.⁷ postulated

that diabetes insipidus occurred as result of impingement on the intracavernous portion of the inferior hypophysial artery, which caused diminished perfusion to the posterior lobe. Alternatively, kinking or pressure on the infundibulum by edematous, hemorrhagic material, which impeded transit of antidiuretic hormone from the preoptic and paraventricular nuclei of the hypothalamus to the posterior lobe, might prove to be the originating cause of diabetes insipidus²¹. The possible pathophysiology involved in this case is the pressure on the infundibulum by edematous or hemorrhagic material, which makes the diabetes insipidus transitory more probable.

In conclusion, it is important to be aware that apoplexy can be followed by central diabetes insipidus, in order to avoid hydroelectrolytical alterations that can deteriorate the evolution of those patients.

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