

# INTRASELLAR INTERNAL CAROTID ANEURYSM COEXISTING WITH GH-SECRETING PITUITARY ADENOMA IN AN ACROMEGALIC PATIENT

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The coexistence of pituitary adenoma and cerebral aneurysm is rare, although its prevalence is higher than would be expected in general population<sup>1-5</sup>. We report on an extremely rare condition where growth hormone (GH)-secreting adenoma coexisted with an intrasellar internal carotid artery (ICA) aneurysm.

## CASE

A 58 years old female presented with headache and acromegalic phenotype. She had hypertension for 10 years and galactorrhea for 3 years. Mean plasma GH was 8.1  $\eta\text{g}/\text{mL}$  and mean IGF-1 level was 703  $\eta\text{g}/\text{mL}$  (Reference: 78–258  $\eta\text{g}/\text{mL}$  by immunoradiometric assay). Results obtained during pre- and postoperative glucose tolerance tests (OGTT) and octreotide response test (100  $\mu\text{g}$  administered subcutaneously every six hours) can be seen in Table 1 and 2, respectively. Prolactin baseline level was 28.6  $\eta\text{g}/\text{mL}$  and pituitary function was otherwise intact. Magnetic resonance imaging (MRI) of the sellar region disclosed a 1.2 cm ICA aneurysm occupying the left hemisella and a 0.7 cm pituitary adenoma just below it (Fig 1A). Angiography disclosed left ICA aneurysm posterior to the emergency of the ophthalmic artery (Fig 1B). The patient tolerated well a balloon occlusion test and was submitted to aneurysm exclusion through ICA occlusion in the neck and transcranial clipping of the supraclinoid ICA immediately below the ophthalmic artery's exit. One month afterwards, she was submitted to complete adenoma removal

through a transsphenoidal approach (Fig 2). The excluded aneurysm was easily seen occupying the entire left hemisella. Immunohistochemical examination confirmed the presence of GH-secreting adenoma. Immediate postoperative GH and prolactin levels were 3.7 and 5.6  $\eta\text{g}/\text{mL}$ , respectively. Four days later, IGF-1 was 543  $\eta\text{g}/\text{mL}$  (78–258  $\eta\text{g}/\text{mL}$ ). One month after adenoma removal, GH and IGF1 levels were 5.0  $\eta\text{g}/\text{mL}$  and 527  $\eta\text{g}/\text{mL}$ , respectively. After 3 months of follow-up, patient's GH and IGF-1 levels were 4.1  $\eta\text{g}/\text{mL}$  and 413  $\eta\text{g}/\text{mL}$ , respectively. It was not possible to perform a follow-up MRI since the clip used in surgery was not MRI-compatible.

She was then started on Sandostatin® LAR 30 mg / month. Four months later, IGF-1 and GH levels were 107  $\eta\text{g}/\text{mL}$  (Refer-



Fig 1. (A) A gadolinium-enhanced MRI showing a round flow-void mass in the left hemisella with 1.2 cm (arrow-head) associated to a subjacent pituitary microadenoma (small arrow). (B) A lateral-scan angiography showing a left paraclinoid ICA aneurysm directed down into the sella.

Table 1. Pre-/ postoperative oral glucose tolerance test.

Time (minutes)	0	30	60	90	120
Glucose level (mg/dL)	93 / 92	193 / 169	181 / 204	150 / 174	108 / 111
GH ( $\eta\text{g}/\text{mL}$ )	11.4 / 2.7	6.6 / 1.1	6.8 / 2.7	6.8 / 2.5	7.1 / 2.7

Table 2. Octreotide acute response test (100  $\mu\text{g}$  sc 6/6h).

Time (hours)	0	2	4	6	8	24	26
GH ( $\eta\text{g}/\text{mL}$ )	5.5	0.44	0.52	0.63	0.71	0.37	0.22

## ANEURISMA INTRASELAR DA CARÓTIDA INTERNA COEXISTINDO COM ADENOMA SECRETOR DE GH EM PACIENTE ACROMEGÁLICO

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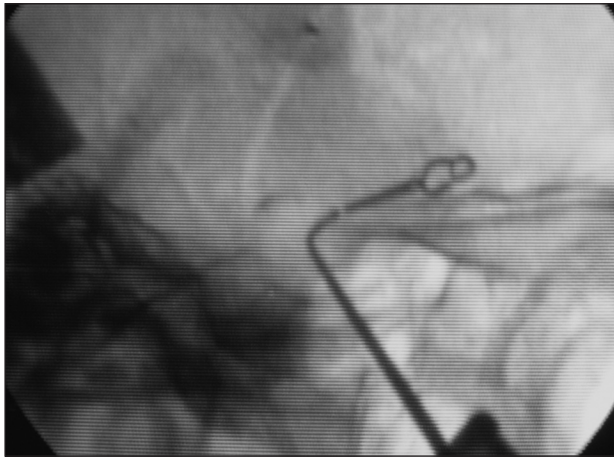


Fig 2. An intraoperative plain film showing a lateral view of the sellar region depicting the relationship between the previously clipped ICA and the curette during the transsphenoidal removal of adenoma.

ence: 81–225 ng/mL) and 0.9 ng/mL, respectively. By this time, patient underwent radiosurgery. After two years of follow-up, the patient remained in remission and receiving Sandostatin LAR.

This case report was agreed by the local ethic committee.

## DISCUSSION

The prevalence of sellar-region's aneurysm among others is 1-2%.<sup>2</sup> The prevalence of the coexistence of pituitary adenoma and cerebral aneurysm is higher than that with other benign brain tumors in the general population<sup>5</sup>. Approximately 50% of these patients have acromegaly<sup>1,3,5-8</sup>, suggesting that high GH and IGF-1 levels or their biological effects might be implicated in the aneurysm's genesis. High IGF-1 induces artery dilation<sup>8</sup>, atherosclerotic and degenerative changes of the artery wall<sup>8,9</sup>, tumor invasion and tumor-directed neovessels<sup>9</sup>. Hypertension and diabetes are very likely to be involved in the process<sup>5</sup>. Patients with intrasellar (not intracranial) aneurysms and pituitary adenomas are extremely rare.

Mostly, the diagnosis of such aneurysms is incidental, and occurs when performing the preoperative investigation for adenomas<sup>7</sup>. However, different clinical presentations may occur, such as fatal epistaxis<sup>10</sup> or pituitary apoplexy<sup>11</sup>, as a result of aneurysmal bleeding into the adenoma. Misdiagnosis of this condition may have hazardous hemorrhagic consequences.

These two conditions must be treated. Although simultaneous microsurgical treatment of the aneurysm and the adenoma through a pterional or a supraorbital keyhole approach has been advocated in different reports<sup>6,12,13</sup>, approaching the vascular lesion first is usually the best choice. In our case, we performed an ICA entrapment by endovascular occlusion in the neck and transcranial clipping of ICA just bellow the ophthalmic

artery's emergence. This treatment was possible because the patient tolerated well a previous balloon occlusion test. One month later, the adenoma was completely removed through a transsphenoidal route. Nevertheless, the patient did not achieved endocrinological remission and needed adjuvant therapy with Octreotide and radiosurgery. Clinical and endocrinological control were then obtained.

Although the results obtained while treating this patient were good, a minimally invasive therapeutic option would also be available for such patients: the vascular lesion could be treated by endovascular trapping of the internal carotid artery and the GH-secreting tumor could have been treated primarily with somatostatin analogs or transsphenoidal surgery. Primary clinical treatment with somatostatin should be considered especially in patients with known cavernous sinus invasion by the tumor and no mass effect directed to the optic apparatus<sup>14-15</sup>.

We believe that the treatment of this dual-pathology should be carried out in two steps: vascular pathology should be treated first to avoid potential future catastrophic hemorrhage and the pituitary pathology afterwards.

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