The coexistence of pituitary adenoma and cerebral aneurysm is rare, although its prevalence is higher than would be expected in general population\textsuperscript{1-5}. 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 $\mu$g/mL and mean IGF-1 level was 703 $\mu$g/mL (Reference: 78–258 $\mu$g/mL by immunoradiometric assay). Results obtained during pre- and postoperative glucose tolerance tests (OGTT) and octreotide response test (100 $\mu$g administered subcutaneously every six hours) can be seen in Table 1 and 2, respectively. Prolactin baseline level was 28.6 $\mu$g/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 transsphephoidal 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 $\mu$g/mL, respectively. Four days later, IGF-1 was 543 $\mu$g/mL (78–258 $\mu$g/mL). One month after adenoma removal, GH and IGF-1 levels were 5.0 $\mu$g/mL and 527 $\mu$g/mL, respectively. After 3 months of follow-up, patient’s GH and IGF-1 levels were 4.1 $\mu$g/mL and 413 $\mu$g/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 $\mu$g/mL (Refer-
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 achieve 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 apparatus16-18.

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.

REFERENCES