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Magnetic resonance imaging finding of coexistence of bilateral paraclinoid aneurysms in a patient with a nonfunctioning macroadenoma, simultaneous resection, and clipping: illustrative case

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BACKGROUND Unruptured incidental intracranial aneurysm can coexist with pituitary adenoma, however, the occurrence is extremely rare. Timely diagnosis of asymptomatic intracranial aneurysms with pituitary adenoma may lead to planning a tailored surgical strategy to deal with both pathologies simultaneously. A case of a patient who underwent transcranial resection of a pituitary adenoma with clipping of two mirror aneurysms is reported.

OBSERVATIONS A 55-year-old female presented with deterioration of visual acuity that progressed over 1 year, as well as presence of right eyelid ptosis. Magnetic resonance imaging of the head showed the presence of an intrasellar pituitary macroadenoma. Bilateral paraclinoid aneurysms were documented to be in contact with the pituitary tumor. The patient underwent surgery with simultaneous aneurysm clipping and tumor resection through a standard pterional approach with intradural clinoidectomy. The aneurysms were successfully clipped after the tumoral debulking. After clipping, the pseudocapsule was fully resected.

LESSONS Various treatment options are available. Although endovascular securing of the aneurysms prior to the tumor resection would be ideal, in cases in which this resource is not readily available at all times, the surgeon must be prepared to solve pathologies with an elevated level of complexity.

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KEYWORDS bilateral paraclinoid aneurysms; pituitary adenoma; microsurgery; coexisting adenoma and aneurysm

Unruptured incidental intracranial aneurysm can coexist with pituitary adenoma, however, its occurrence is extremely rare. Recent increased usage of magnetic angiography and cerebral angiography for the screening of intracranial aneurysms has led to increased detection of the coexistence of intracranial aneurysms and brain tumors.[1]

Synchronous asymptomatic intracranial aneurysms and pituitary adenoma can prevent accidental rupture of aneurysm during surgical resection of the pituitary adenoma, which may enable the planning of a tailored surgical strategy to deal with both pathologies simultaneously.[1]

Giant pituitary adenomas are tumors of 4 cm or greater in maximum diameter, account for 5%–14% of adenomas in most series.[2] These tumors represent a very important therapeutic challenge. Due to their large size, irregular form, and proximity to critical neurovascular structures, their gross total resection is infrequent.

In our institution, the endoscopic endonasal transsphenoidal approach is the favored management for giant pituitary adenomas, the benefits of which include short hospital stay, important visual improvement, as well as better esthetic results. Unfortunately, this method is not ideal to effectively resect neoplasms that have an extensive lateral extension beyond the lateral wall of the cavernous sinus because of the anatomical limits marked by critical neurovascular structures and a limited range of vision. To extend our treatment's reach, we perform either open or endoscopic endonasal approaches, and on some occasions, both simultaneously.

We present here the case of a patient with coexisting bilateral internal carotid artery aneurysms encased within a giant

ABBREVIATIONS GH = growth hormone; ICA = internal carotid artery; MRI = magnetic resonance imaging; UIA = unruptured intracranial aneurysm.

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nonfunctioning pituitary adenoma, her management, and postoperative results.

Illustrative Case

A 55-year-old female presented to our institution due to progressive deterioration of visual acuity over 1 year, as well as presence of right eyelid ptosis. Past medical history included longstanding primary hypothyroidism treated with levothyroxine.

The physical examination demonstrated bilateral vision loss, with 20/200 in both eyes, with a bilateral temporal hemianopia documented in the kinetic campimetry test. No alterations of the eye movements were observed. Right ptosis was documented. The rest of the neurological examination was normal. The patient's hormone profile is described in Table 1.

Magnetic resonance imaging (MRI) of the brain, performed with a 1.5 Tesla unit revealed the presence of a homogeneously enhancing intrasellar pituitary macroadenoma, with extension to the suprasellar region that encompassed the visual pathways, as well as extension into the third ventricle. No invasion of the cavernous sinus was observed. Incidental bilateral nonruptured paraclinoid aneurysms were documented to be in contact with the pituitary tumor, seen as a flow void image on T2-weighted MRI (Fig. 1A) that was confirmed on the contrast T1-weighted image (Fig. 1B).

An angiography was performed, which confirmed the presence of the aforementioned nonruptured aneurysms, the first one a bilobed saccular paraclinoid aneurysm that emerged at the dorsal aspect of the right internal carotid artery (ICA), distal to the ophthalmic artery,[3] with a neck measuring 6.7 mm and a neck to dome distance of 5.7 mm (Fig. 1C). From the left ICA, a paraclinoid saccular aneurysm emerged that originated form the dorsal aspect of the artery,[3] in proximity to the ophthalmic artery with a neck measuring 4 mm, neck to dome distance of 2.1 mm, and a diameter of 3.5 mm with caudolateral orientation (Fig. 1D).

TABLE 1. Hormonal profile of the patient

Hormone	Baseline	Normal Value
T4, nmol/L	127	71.2–141
fT4, pmol/L	21.3	10–28.2
TSH, μIU/mL	0.015	0.47–4.68
T3, nmol/L	1.75	1.49–2.6
fT3, pmol/L	5.13	4.26–8.1
Prolactin, ng/mL	55	
Diluted prolactin, ng/mL	1:100 260	
Cortisol, μg/dL	15.4	
Estradiol, pg/mL	21.961	
LH, mUI/mL	2.6	
FSH, nmol/l	21.2	
ACTH, pg/mL	25.42	4.7–48.8
IGF-1, ng/mL	56.91	
Growth hormone, ng/mL	0.050	

T3 = triiodothyronine; T4 = thyroxine; fT3 = free triiodothyronine; fT4 = free thyroxine; TSH = thyroid-stimulating hormone; LH = luteinizing hormone; FSH = follicle-stimulating hormone; ACTH = adrenocorticotropic hormone; IGF-1 = insulin-like growth factor 1.

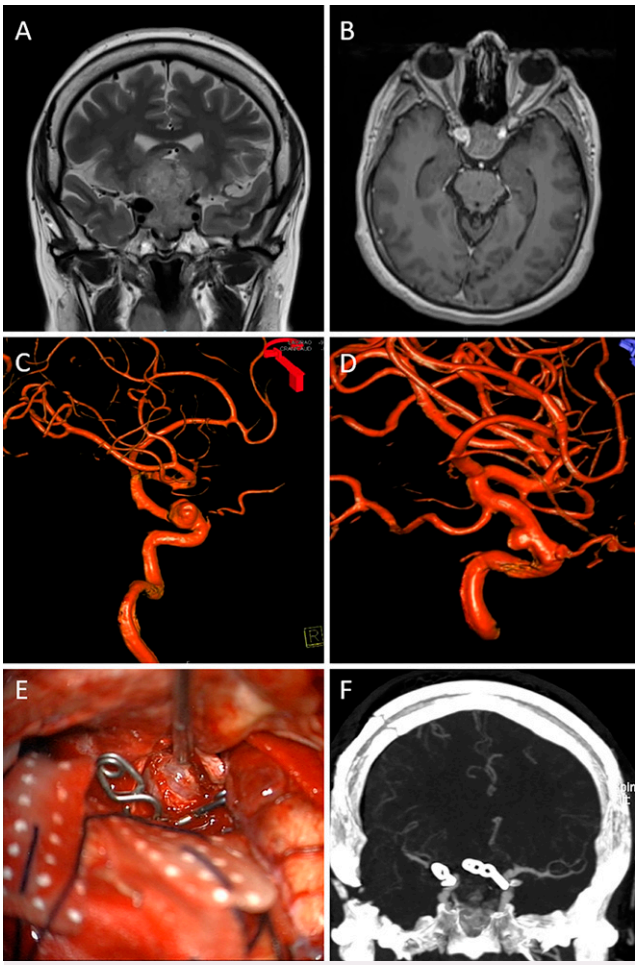

FIG. 1. A: T2-weighted coronal MRI with the presence of a heterogeneous lesion in the sellar and suprasellar regions, it has defined borders, a bilobed appearance, and it dorsally displaces the third ventricle. It extends mainly into the cavernous sinus on the left side without surpassing the lateral tangent of the supra and intracavernous carotids. Increased thickness of the right supracavernous carotid. (Knosp 2, Hardy Wilson IIIC). **B:** Contrast T1 coronal MRI with a lesion in the sellar region that heterogeneously captures the contrast medium with hyperintense signal in the bilateral carotid portion with an increase in size predominantly on the right side. **C:** 3D reconstruction of angiography of the right upper lateral dome ICA. **D:** 3D reconstruction of angiography of the left ICA ventral medial dome. **E:** Transsurgical image of clipping of paraclinoid aneurysms and resection of sellar lesion. **F:** Coronal section tomography angiography with the presence of clips with adequate bilateral aneurysmal exclusion. 3D = three-dimensional.

The patient underwent surgery in August 2021 with simultaneous aneurysm clipping and tumor resection through a standard pterional approach with intradural clinoidectomy (Fig. 1E).

The aneurysms were successfully clipped after the tumoral debulking with preservation of the pseudocapsule. After the clipping, the pseudocapsule was fully resected. Postoperative computed tomography scan demonstrated successful gross total resection and adequate bilateral clipping of the aneurysms (Fig. 1F).

In the immediate postoperative period, the patient continued to have right palpebral ptosis, which was treated with a serried bolus of methylprednisolone, with partial remission of the neurological

deficit. Immunohistology microscopic findings confirmed the diagnosis of a nonfunctional pituitary adenoma.

At 4-month follow-up, the patient's palpebral ptosis ophthalmoplegia improved significantly and she continued to have no other neurological deficit.

Discussion

Observations

Intracranial tumors have previously been associated with asymptomatic incidental aneurysms in the literature.

In previous autopsy and angiographic studies, incidence of intracranial aneurysms was reported as ranging from 1% to 7%; their rupture causes dramatic neurological consequences or patient death.[4]

Simultaneous occurrence of pituitary tumors and cerebral aneurysms is uncommon. Wakai et al.[5] reported synchronic aneurysms in 7.4% of 95 pituitary adenomas over a period of 5 years. In the series published by Pant et al.,[6] 25 cases (5.4%) of coexisting intracranial aneurysm were found in a retrospective study of 467 cases of pituitary adenomas. In another series, coexisting asymptomatic aneurysms were reported in 13.8% of growth hormone (GH)-secreting and in 5.1% of nonfunctioning pituitary adenomas.

In the literature, intracranial aneurysms were often reported in association with acromegaly, theoretically secondary to the prolonged GH hypersecretion. In a review by Acqui et al.,[7] approximately 50% of the pituitary adenomas associated with intracranial aneurysms were GH secreting.[6,8] On the other hand, multiple aneurysmatic disease and a nonfunctioning macroadenoma have rarely been reported.

In theory, the pathogenic mechanisms include the mechanical effect due to direct contact between adenoma and aneurysm with vascular infiltration[9] or wall stress secondary to pull force exercised by the adenoma proximate to the arterial wall. Local circulatory stress, endocrinological effect, mechanical effects, and direct invasion have been proposed as mechanisms as well.[10] Other suggested processes have been reported to be the association with radiation therapy for the treatment of a pituitary adenoma,[11,12] rather than other neuroendocrinological effects that may have been involved in our patient with a nonfunctioning adenoma.

In a review by Pant et al.,[6] aneurysms were found in 25 patients (5.4%), 97% of which were located in the anterior circulation. Locations have been reported to be at the A2 and A1 anterior communicating artery. Although the intracranial tract of the ICA was the most common location,[8] aneurysms of the cavernous or supraclinoid carotid that encroach into the pituitary adenomas as in the present case are very rare. The prevalence of sellar region's aneurysm among others is 1%–2%.[13] Usually they are unique lesions, unlike the case presented. In our literature review, only 3 patients have presented with multiple aneurysms associated with a pituitary tumor.[14,15] To our knowledge, this is the first case to be reported with bilateral paraclinoid aneurysms embedded in the pituitary tumor, and to be treated simultaneously during the same surgical procedure.

Tumor growth causes microanatomical alterations in the cerebral circulation, blood flow modification, and greater hemodynamic stress, which predispose patients to aneurysms formation.[10]

Hypervascular lesions, such as glioblastomas and meningiomas, may be associated with flow-related aneurysms on feeding arteries, but aneurysms within the gross tumor are unusual, this most likely due to abnormal oversecretion of growth factors such as vascular endothelial growth factor. These angiogenic factors are hypothesized to promote increased blood flow and possibly secondary changes to arterial walls, thus facilitating the formation of flow-related aneurysms.[16]

Other variants of tumor-related aneurysms are neoplastic cerebral aneurysms most of which, similar to mycotic aneurysms, are small and located prominently in the distal branches of the middle cerebral artery and anterior communicating artery, often buried in a cortical sulcus. They have been most commonly reported as associated with myxoma (60.4%), choriocarcinoma (26.1%), and other metastatic tumors such as pulmonary and breast cancer (13.5%). The pathogenesis mechanisms most likely include the following due to postembolic vascular damage and subsequent endothelial scarring resulting in alteration of flow dynamics: tumor cell infiltrating cerebral vessels via vasa vasorum, tumor cell penetration through intact or damaged endothelium with subintimal growth, eventual penetration and destruction of the entire arteria wall, and finally proliferation of tumor cells in the wall of aneurysms and active invasion of the internal elastic lamina by viable tumor cells.[17]

MRI and MR angiography are considered the procedures of choice in the preoperative assessment of patients with pituitary tumors. The presence of flow voids on T1-weighted and T2-weighted MRI sequences is 100% specific for aneurysms with a sensitivity of 88%[18,19] it has been reported that only 80% of giant aneurysms show signs of blood flow in the aneurysm sac.[20,21] MRI may not be effective to detail small-sized intracranial aneurysms.

In our case, coronal and axial MRI revealed a flow void in the right paraclinoid ICA, which was later confirmed on angiography.

The presence of an aneurysm coexisting with a pituitary tumor represents a therapeutic challenge for both the patient and neurosurgeon. Thorough anatomical information and extensive comprehension of this association is a requisite for planning the correct surgical approach of these cases and avoid life-threatening hemorrhagic complications.

The surgical management of synchronous intracranial pathology can be achieved through various options, including supraorbital keyhole,[19] fronto-orbital approach, pterional approach, and endonasal transsphenoidal approach, either microscopic or endoscopic. In some cases, preoperative endovascular occlusion of unruptured intracranial aneurysms (UIAs) has been reported. Aneurysms in close proximity to an adenoma can be managed in a single stage with clipping of the aneurysm and resection of the adenoma.[1,21,22] Endovascular treatment of this type of aneurismatic lesion is preferred in a significant number of healthcare centers. The tumor could have been managed through an endoscopic endonasal transsphenoidal approach after the aneurysms had been successfully secured via coil embolization or flow diversion, which would have enabled a much less invasive treatment for our patient.

The International Study of Unruptured Intracranial Aneurysms (ISUIA)-1 and ISUIA-2 proposed that there is a minor risk of rupture of intracranial aneurysms <7 mm in size, although many studies have contradicted this conclusion.[23] The International Subarachnoid Aneurysm Trial documented that 52.5% of their patients presented with aneurysms of <5 mm.[24] The most recent guidelines from the American Heart Association and the American Stroke Association for managing unruptured intracranial aneurysms recommend follow-up for small (<7), but these guidelines do not take into account the synchronous involvement of other intracranial pathologies.

Furthermore, the size of the aneurysms, defined as the maximum luminal diameter in millimeters, can be a determining factor when making the decision on whether or not to treat. It is one of the most

important factors for predicting the risk of rupture. It has been confirmed thar 85% of aneurysms with diameter <7 mm remained stable and did not grow during 10 years of follow-up.[25,26] Aneurysms >5 mm are at increased risk of rupture and a diameter between 5 and 7 mm is generally considered the limit for considering treatment.[26]

In a survey performed by Salih et al.[27] for aneurysms measuring 2–4 mm, the majority of physicians preferred regular follow-up, however, for aneurysms 5 to 7 mm, the majority favor treatment. A cost-effectiveness analysis performed by Veet et al.[28] indicated that the most cost-effective management strategy was immediate surgical treatment for small (<7 mm) unruptured intracranial aneurysms with incremental cost effectiveness ratio of $45,772 reactive to active surveillance in patients <70 years of age, with increased quality-adjusted life-year, but coiling was not modeled in this study due to the high complexity associated with recurrence and retreatment.

When discussing the difference of effectiveness between microsurgical ligation and endovascular coiling, much controversy is still present. While clip ligation of an aneurysm neck is thought to be the most durable treatment, patients may be at a continued risk for recurrent aneurysms and the development of de novo aneurysms, with reported recurrence rates of 0.14%–0.53%.[29] In a meta-analysis published by Ruan et al.,[30] it was found that in terms of death, bleeding, cerebral ischemia, occlusion of aneurysm, and independence in daily activities, the risk ratios of both modalities of treatment were similar. In a study performed by Huang and You,[31] it was reported that surgical clipping of UIAs was associated with a low rate of complications and a high rate of complete aneurysmal obliteration for patients with multiple risk factors, however, the endovascular management showed successful results, with a shorter length of stay and a modest procedure-related morbidity. Further long-term outcome studies are necessary.

Unfortunately, endovascular treatment poses an important economic burden for the patients at our institution by not being a readily available option at all times. For this reason, we proceeded with a simultaneous surgical approach.

Lessons

The presence of multiple paraclinoid aneurysms enclosed within a nonfunctioning pituitary tumor is exceptionally uncommon. Cautious assessment of preoperative imaging is fundamental, especially in those with particular anatomical configuration, such as in our patient.

Various treatment options are available. Although endovascular securing of the aneurysms prior to the tumor resection would be ideal for saccular aneurysms in other locations, in paraclinoid aneurysms surgery must be considered as a therapeutic option, tailoring the treatment to each patient's specifics. Another situation is that this resource is not readily available at all times, thus the surgeon must be prepared to solve pathologies with an elevated level of complexity.

In our case, taking into consideration the location of the aneurysm, surgical resection and clipping was successfully performed prior to near total resection of the tumor without transoperative complications. Our patient was satisfied with the postoperative results.

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Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author Contributions

Conception and design: Mondragon-Soto, Villanueva-Castro, Aragón-Arreola, Gómez-Amador. Acquisition of data: Mondragon-Soto, Villanueva-Castro, Tovar-Romero, Aragón-Arreola, Sangrador-Deitos, Villanueva-Solórzano. Analysis and interpretation of data: Mondragon-Soto, Villanueva-Castro, Sangrador-Deitos. Drafting the article: Mondragon-Soto, Villanueva-Castro, Sangrador-Deitos, Gómez-Amador. Critically revising the article: Mondragon-Soto, Villanueva-Castro, Tovar-Romero, Sangrador-Deitos, Cano-Velazquez, Gómez-Amador. Reviewed submitted version of manuscript: Mondragon-Soto, Villanueva-Castro, Tovar-Romero, Sangrador-Deitos, Cano-Velazquez, Gómez-Amador. Approved the final version of the manuscript on behalf of all authors: Mondragon-Soto. Statistical analysis: Villanueva-Castro. Administrative/technical/material support: Mondragon-Soto, Villanueva-Castro, Aragón-Arreola, Cano-Velazquez, Gómez-Amador. Study supervision: Mondragon-Soto, Villanueva-Castro, Tovar-Romero, Aragón-Arreola, Sangrador-Deitos, Cano-Velazquez, Gómez-Amador.

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